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IGG4-RELATED DISEASE PRESENTING AS CAVERNOUS SINUS SYNDROME: A DIAGNOSTIC CHALLENGE

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Abstract ID: 36

Aim/ Background: To highlight a rare presentation of probable IgG4-related disease (IgG4-RD) involving the cavernous sinus in an adolescent male presenting with painful ophthalmoplegia and initially suspected autoimmune etiology.

Materials & Methods: An 18-year-old male presented with holocranial headache and horizontal diplopia worsening on right gaze. Initial MRI brain showed bulky enhancing right cavernous sinus. CSF analysis was normal. Autoimmune evaluation revealed ANA positivity with anti-Sm antibodies, and he initially received pulse methylprednisolone for presumed lupus-related cavernous sinus involvement. On referral to our centre, detailed neurological, ophthalmological, radiological, and rheumatological evaluations were performed, including repeat CSF studies, autoimmune profile, serum ACE, complement levels, and serum IgG4 levels.

Results: Neurological examination revealed isolated right lateral rectus palsy. Repeat contrast MRI brain and orbit demonstrated asymmetric enlargement and enhancement of the right cavernous sinus suggestive of inflammatory pathology. Repeat autoimmune workup was negative, including ANA (IF), ENA, ANCA, RF, and CRP. Serum ACE and complement levels were normal. Serum IgG4 level was elevated (184 mg/dL) with normal total IgG, favouring probable IgG4-RD. Histopathological confirmation was not feasible because of the high-risk location. The patient received intravenous methylprednisolone (1 g/day for 5 days) with complete resolution of diplopia and marked improvement in headache.

Conclusion: This case highlights that isolated antibody positivity, in the absence of supportive clinical criteria, should not be considered sufficient evidence for systemic lupus erythematosus-related neurological disease. IgG4-related disease should be considered in patients presenting with subacute, afebrile cavernous sinus syndrome with inflammatory imaging findings and normal CSF studies. Marked responsiveness to corticosteroid therapy further supports the diagnosis and emphasizes the importance of early recognition of this potentially treatable condition.

Keywords: IgG4-related disease, cavernous sinus, Lateral rectus palsy

A LONGITUDINAL STUDY OF SITE PREVALENCE ASSOCIATED CLINICAL AND SOCIO-DEMOGRAPHIC FACTORS, TREATMENT AND OUTCOME IN NON-TRAUMATIC INTRA-CRANIAL HEMORRHAGE PATIENTS PRESENTING AT A TERTIARY CARE FACILITY IN NCR, INDIA.

Dr. Aditya Seth, Dr. Megha Luthra, Dr. Tushar Marbate, Dr. Puneet Ohri, Dr. Ashwani Sharma

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Abstract ID: 8

Aim/ Background: To study prevalence and outcomes associated with clinico-socio-demographic factors in treated NTICH (non-traumatic intra-cranial hemorrhage) patients in Institute Of Human Behaviour And Allied Sciences (IHBAS)

Materials & Methods:

- Study Design: Hospital-based cross-sectional study.
- Study Site: Institute of Human Behaviour and Allied Sciences (IHBAS), Delhi.
- Study Period: 33 months (January 2022 to September 2024).
- Study Population: All newly and previously diagnosed NTICH patients presenting to the Neurosurgery Department.
- Selection Criteria: Mandatory written informed consent from patient or caregiver.
- Inclusion: Diagnosis confirmed via clinical history and NCCT Brain imaging.
- Exclusion: Patients referred out or those who left against medical advice (LAMA).
- Sample Size & Technique: 100 cases selected via total enumeration (census method) to reflect real-time patient load.
- Data Collection: Consecutive enrollment based on anatomical classification of hemorrhage on NCCT. Data was gathered using a structured questionnaire covering GCS scores, clinical/biochemical features, socio-demographics, treatment, and outcomes.
- Data Analysis: Statistical analysis performed using the latest version of SPSS.

Results: This study analyzes 100 NTICH cases collected via total enumeration over 33 months. Diagnosis and anatomical classification were confirmed using NCCT Brain imaging. The analysis correlates clinico-socio-demographic variables and GCS scores with patient outcomes.

Ethical Status: Final statistical analysis and data presentation are pending Institutional Ethics Committee (IEC) approval. Upon clearance, the full dataset will be processed using SPSS to determine definitive prevalence rates and clinical associations.

Conclusion: This study highlights the critical need for structured longitudinal data in managing non-traumatic intracranial hemorrhage within a tertiary care setting.

The definitive conclusions and statistical inferences will be established upon receiving ethical clearance.

Keywords: Non-traumatic Intracranial Hemorrhage (NTICH), Prevalence

EFFECT OF GREATER OCCIPITAL NERVE BLOCK ON HEADACHE BURDEN IN PATIENTS WITH MEDICATION OVERUSE HEADACHE: A PROSPECTIVE INTERVENTIONAL STUDY (GON-MOH STUDY)

Dr. Agrata Sharma

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Abstract ID: 29

Aim/ Background: Medication overuse headache (MOH) is a disabling chronic headache disorder resulting from excessive use of acute symptomatic medications in patients with pre-existing headache disorders, most commonly migraine. Management is often challenging because withdrawal may temporarily worsen headache symptoms and reduce treatment adherence. Greater occipital nerve (GON) block has shown benefit in chronic migraine and refractory headache disorders and may act as a bridging therapy during medication withdrawal. This study aimed to evaluate the effect of GON block on headache burden in patients with MOH.

Materials & Methods: This prospective interventional study included 30 adult patients diagnosed with MOH according to ICHD-3 beta criteria. Patients received three sequential monthly greater occipital nerve blocks using 2% lignocaine with dexamethasone under aseptic precautions. Participants were followed for 3 months. Outcomes assessed included headache intensity using Visual Analogue Scale (VAS), headache frequency (days/month), headache duration per attack (hours), abortive medication use, and headache-related disability using HIT-6 score.

Results: Significant improvement was observed across all clinical parameters following GON block. Mean headache intensity reduced from 8.9 ± 0.92 at baseline to 3.12 ± 2.93 at 3 months ($p < 0.001$). Headache frequency decreased from 20.23 ± 9.32 to 4.53 ± 6.83 days/month ($p < 0.001$), while headache duration per attack reduced from 30.9 ± 20.54 to 4.56 ± 6.94 hours ($p < 0.001$). Abortive medication use declined from 23.07 ± 12.91 to 1.65 ± 2.26 per month ($p < 0.001$). HIT-6 scores improved from 72 ± 5.66 to 41.18 ± 8.89 ($p < 0.001$). No serious adverse events were reported.

Conclusion: Greater occipital nerve block appears to be a safe and effective adjunctive therapy in MOH, significantly reducing headache burden, medication overuse, and disability. It may serve as a useful bridging therapy during withdrawal and preventive treatment.

Keywords: Medication overuse headache Greater occipital nerve block Ch

EXPANDING THE ROLE OF APIXABAN IN CEREBRAL VENOUS THROMBOSIS: A SINGLE-CENTRE CASE SERIES

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AIIMS, Bhopal

Abstract ID: 30

Aim/ Background: Cerebral venous thrombosis (CVT) is an uncommon but potentially life-threatening cause of stroke, frequently affecting young adults and often presenting with headache, seizures, focal neurological deficits, or altered sensorium. Vitamin K antagonists remain the conventional long-term anticoagulant therapy following acute management with low-molecular-weight heparin (LMWH); however, they require regular monitoring and are associated with variable anticoagulant effects. Evidence regarding the use of direct oral anticoagulants (DOACs), particularly apixaban, in CVT remains limited.

Materials & Methods: We conducted a single-centre case series of 16 consecutive patients with radiologically confirmed CVT treated with apixaban following initial anticoagulation with LMWH. Demographic characteristics, clinical presentation, risk factors, neuroimaging findings, treatment details, adverse events, and radiological outcomes were analyzed. Patients underwent serial clinical and radiological follow-up to assess safety, thrombus progression, and venous recanalization.

Results: The mean age of patients was 36.9 years, with headache and seizures being the most common presenting manifestations. Several patients had hemorrhagic venous infarcts at presentation. Superior sagittal and transverse sinuses were the most frequently involved venous structures. All patients received apixaban 5 mg twice daily after acute stabilization with LMWH. No patient developed recurrent thrombosis, worsening intracranial hemorrhage, or major systemic bleeding during follow-up. Only one patient experienced worsening menstrual bleeding. Complete radiological recanalization was observed in all 16 patients, with most achieving recanalization within 3–5 months.

Conclusion: Apixaban demonstrated a favorable safety profile and excellent radiological outcomes in this real-world CVT cohort, including patients with hemorrhagic venous infarction. Larger prospective studies are warranted to further validate these findings.

Keywords: Cerebral venous thrombosis; CVT; Apixaban; Direct oral antic

BREAKING THE CLUSTER CYCLE: GREATER OCCIPITAL NERVE BLOCK AS TRANSITIONAL THERAPY IN EPISODIC CLUSTER HEADACHE — A TWO-PATIENT CASE SERIES

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Abstract ID: 31

Aim/ Background: Cluster headache (CH) is a severe trigeminal autonomic cephalalgia characterized by recurrent unilateral headache attacks associated with ipsilateral cranial autonomic symptoms. Although high-flow oxygen and triptans effectively abort acute attacks, patients often continue to experience frequent disabling episodes during active cluster periods. Greater occipital nerve (GON) block has emerged as a useful transitional therapy aimed at reducing attack burden while preventive therapies achieve therapeutic effect. This case series highlights the clinical utility of GON block in two patients with episodic cluster headache.

Materials & Methods: Two male patients with episodic cluster headache presenting during active cluster periods were evaluated clinically. Both patients demonstrated characteristic unilateral headache attacks associated with conjunctival injection, lacrimation, rhinorrhea, and marked restlessness. Acute attacks were managed with high-flow 100% oxygen administered through a non-rebreather mask. As transitional therapy, GON block was administered using local anesthetic combined with corticosteroid (methylprednisolone or dexamethasone). Clinical response was assessed based on reduction in attack frequency and severity following intervention.

Results: Both patients demonstrated rapid relief of acute attacks with high-flow oxygen therapy. Following GON block, there was a marked reduction in attack frequency and intensity during the active cluster period. No procedure-related complications were observed. The intervention was well tolerated and associated with sustained clinical improvement in both cases.

Conclusion: GON block appears to be an effective and safe transitional therapy in episodic cluster headache. By modulating nociceptive transmission within the trigeminocervical complex, the procedure may reduce attack burden during active cluster periods. In both patients, GON block provided significant symptomatic improvement and complemented established abortive therapy with oxygen. These findings support the growing evidence for the role of occipital nerve blockade in cluster headache management. Larger prospective studies are required to establish optimal treatment protocols and long-term efficacy.

Keywords: Cluster headache; Greater occipital nerve block; Episodic cl

THE THICK DURA: SITE, SEROLOGY AND STEROID RESPONSE IN HYPERTROPHIC PACHYMENINGITIS

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Abstract ID: 411

Aim/ Background: Hypertrophic pachymeningitis (HP) is a rare fibro-inflammatory dural disorder. We describe its spectrum at a tertiary centre in a tuberculosis-endemic region.

Materials & Methods: Retrospective series of eleven patients with contrast-MRI-proven pachymeningeal, cavernous sinus or pituitary disease (May 2025 – July 2026). All underwent standardised infective, granulomatous, autoimmune and prothrombotic screening, with biopsy where uncertain

Results: Eleven patients (five female; 32–71 years). Six had IgG4-related HP (serum IgG4 1.58–3.80 g/L; upper normal 0.864), two Tolosa–Hunt syndrome, one MPO/p-ANCA-associated HP, one biopsy-proven idiopathic HP, and one seronegative relapsing polyneuritis cranialis. Anterior disease — cavernous sinus, orbital apex, hypophysitis — dominated, affecting cranial nerves III, IV, VI and V1 (ESR 42–100). The ANCA case presented with bilateral deafness, otorrhoea and scleritis after failing empirical antitubercular therapy elsewhere. The idiopathic case presented posteriorly: eight-year unilateral lower cranial neuropathy (VII–XII, Collet–Sicard), dural venous sinus thrombosis, normal markers, biopsy showing fibrosis with negative IgG4 immunohistochemistry. All received methylprednisolone pulses. Three IgG4 cases relapsed on tapering and required rituximab; the ANCA case required cyclophosphamide; one developed azathioprine-induced pancytopenia and others are on steroid sparing agents.. The idiopathic case responded incompletely.

Conclusion: Site, serology and treatment response travelled together — anterior, marker-positive disease was steroid-responsive but relapse-prone, whereas posterior fossa fibrotic disease was refractory. Serology directed therapy: IgG4 to rituximab, ANCA to cyclophosphamide. Empirical antitubercular therapy delayed diagnosis in one patient, underlining the need for definitive exclusion

Keywords: OFFLINE

ACUTE-ON-CHRONIC HYPERCAPNIC RESPIRATORY FAILURE IN AN ADULT WITH SCN1A-POSITIVE DRAVET SYNDROME: A PHARMACOMETABOLIC VICIOUS CYCLE

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Abstract ID: 135

Aim/ Background: Dravet syndrome (DS) is a severe SCN1A-related developmental epileptic encephalopathy with improving survival into adulthood. Respiratory complications in adult DS remain poorly characterised. We report the first genetically confirmed adult DS case presenting with acute-on-chronic hypercapnic (Type 2) respiratory failure arising from a progressive pharmacometabolic cascade.

Materials & Methods: A 26-year-old male with SCN1A-positive DS (splice-site variant c.965–1G>A), receiving valproate, clobazam, levetiracetam, topiramate), with global developmental delay, morbid obesity, type 2 diabetes mellitus, and obesity hypoventilation syndrome on nocturnal BiPAP, presented with breathlessness, fever, altered sensorium, and SpO₂ 86% on room air. Arterial blood gas confirmed acute-on-chronic hypercapnic respiratory failure. Comprehensive clinical, biochemical, radiological, and genetic evaluation was performed.

Results: Long-term valproate contributed to metabolic dysfunction and obesity, insulin resistance, and dyslipidaemia — establishing OHS. Clobazam suppressed central respiratory drive; topiramate impaired ventilatory compensation. Febrile illness with constipation-related abdominal splinting precipitated acute decompensation. Treatment with BiPAP, antibiotics, glycaemic optimisation, and bowel decompression resulted in complete clinical recovery with return to compensated baseline.

Conclusion: This case identifies a pharmacometabolic-respiratory vicious cycle unique to adult DS expanding the recognised phenotype beyond previously reported paediatric DS hypoxemic respiratory failure. This adult presentation was hypercapnic, driven by chronic polypharmacy-induced metabolic syndrome and OHS. Sodium channel-blocking antiseizure medications are contraindicated in DS, limiting therapeutic options and compounding metabolic risk. Adult DS patients require proactive multidisciplinary surveillance encompassing metabolic, respiratory, and pharmacological dimensions. This case expands the phenotypic spectrum of adult DS and highlights a preventable, under-recognised cause of morbidity and mortality.

Keywords: DS, SCN1A Mutation, Hypercapnic RF, OHA, ASM Polytherapy

POST-TRAUMATIC PSEUDOBULBAR PALSY FOLLOWING TRAUMATIC BRAIN INJURY A RARE CLINICO-RADIOLOGICAL CORRELATION

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Abstract ID: 59

Aim/ Background: Pseudobulbar palsy is medically defined as a clinical syndrome caused by bilateral upper motor neuron lesions of the corticobulbar pathways that connect the cerebral cortex to the brainstem .

Pseudobulbar palsy is an uncommon sequela of traumatic brain injury (TBI), particularly in the absence of a structural brainstem lesion.

Materials & Methods:

A 19-year-old male sustained a road traffic accident resulting in frontal and orbital injuries with traumatic subarachnoid hemorrhage, managed conservatively. 4 days later, he developed difficulty chewing, swallowing, and handling food along with emotional lability. Detailed neurological examination, cognitive assessment, MRI brain, and whole-spine screening were performed.

Results: Cognition was preserved (MMSE 29/30). Neurological examination revealed brisk jaw jerk, bilateral frontal release signs, hypophonia, emotional lability, and bilateral upper motor neuron features suggestive of pseudobulbar palsy. MRI brain demonstrated bilateral symmetrical putaminal signal changes extending into the external capsules without diffusion restriction or hemorrhage. No structural brainstem lesion was identified. MRI whole-spine screening showed no cord compression or intrinsic cord pathology.

Conclusion: The absence of a brainstem lesion with bilateral basal ganglia and external capsule involvement suggests disruption of corticobulbar pathways as the likely substrate for pseudobulbar dysfunction. The delayed onset after TBI supports a post-traumatic etiology.

Post-traumatic pseudobulbar palsy is a rare but important complication of TBI. Bilateral putaminal and external capsule abnormalities may contribute to corticobulbar tract dysfunction, emphasizing the need for careful clinical-radiological correlation in patients presenting with delayed bulbar symptoms after head injury.

Keywords: Traumatic brain injury,pseudo bulbar palsy

DEVELOPMENT AND PRELIMINARY VALIDATION OF HIP-PRO: AN INTELLIGENT WEARABLE HIP PROTECTION SYSTEM FOR PROACTIVE FALL INJURY PREVENTION IN OLDER ADULTS

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Abstract ID: 83

Aim/ Background: Fall-related injuries are a leading cause of morbidity, disability, and mortality among older adults, with hip fractures resulting in substantial functional decline and loss of independence. Conventional hip protectors offer passive protection, while most wearable devices focus on fall detection after impact. HIP-PRO was developed as an intelligent wearable system that continuously monitors body motion, predicts impending falls, and deploys pre-emptive hip protection before ground impact to reduce injury severity.

Materials & Methods: HIP-PRO integrates a waist-mounted inertial measurement unit (3-axis accelerometer and gyroscope), embedded firmware, machine learning-based motion analysis, a real-time risk classification engine, and a reusable micro-pump-driven airbag. The device continuously analyses movement during activities of daily living to distinguish normal motion from high-risk patterns. It also incorporates a rechargeable power system and SOS alert functionality. Preliminary validation included laboratory assessment of sensing accuracy, response time, airbag deployment, and usability under simulated fall-risk conditions.

Results: The prototype demonstrated reliable real-time motion monitoring, timely airbag deployment, and stable operation during simulated mobility scenarios. Consistent sensing performance, dependable airbag inflation, and comfortable day-long wearability were observed. Initial pilot feedback from healthcare professionals and end users indicated high usability and increased confidence during mobility. The technology has received an Indian patent (Patent No. 506998) and multiple innovation grants supporting further clinical validation.

Conclusion: HIP-PRO combines wearable sensing, intelligent motion analysis, and pre-impact hip protection in a lightweight device designed to mitigate injury before impact. This approach has the potential to reduce hip fractures, preserve mobility, and support independent living among older adults. Future studies will focus on large-scale clinical validation, regulatory approval, and real-world implementation across healthcare settings.

Keywords: Falls Hip Protection Hip Fracture Wearable Sensors

SURVEYS TO KNOW THE OPTIMUM USE OF LACOSAMIDE & ITS EFFICACY, SAFETY AND TOLERABILITY IN VARIOUS SEIZURE TYPES (OPULENT – I & II SURVEYS)

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Abstract ID: 95

Aim/ Background: Despite its wide availability in India since 2009, specialists' perspectives on lacosamide's efficacy-safety and use across various epilepsy-related conditions including focal-onset seizures (FOS), generalized tonic-clonic seizures (GTCS), status epilepticus (SE) and drug-resistant epilepsy (DRE) are scant. Hence, this survey was envisaged.

Materials & Methods: A pan-India questionnaire-based, cross-sectional e-survey among 181 consenting neurologists and neurosurgeons.

Results: Specialists preferred lacosamide as: First line add on (56%) followed by second add-on (31%) and also as first-line monotherapy (27%) in FOS. Beyond FOS, as add-on, 83.88% respondents preferred lacosamide over other antiseizure medications in GTCS, while 96.66 % considering it in DRE.

Lacosamide was most frequently added on to levetiracetam (89%) and sodium valproate (38%). 88% prescribed intravenous lacosamide, typically (79%) for 3–5 days. 66% also used lacosamide in children aged 4-17.

79% and 75% rated its efficacy as rated good-to-excellent in DRE and SE respectively. 92.21% respondents rated lacosamide to have good-excellent tolerability. ~80% stated side effects to occur in only up to 10% patients, majorly including dizziness (48.33%) and sleepiness/unusual drowsiness (18.33%) and mostly (87.77%) mild.

Attainment of seizure freedom (43.33%), safety/tolerability issues (42.22%) and planning/being pregnant (35.5%) were top reasons for discontinuing lacosamide.

71.66% respondents expressed preference for liquid dosage form of lacosamide in children.

Conclusion: Lacosamide is widely perceived as a choice of add-on ASM to manage FOS, GTCS, DRE as also SE, in not only adults but also children. Its flexible use (oral/IV) and favourable efficacy-safety outcomes in real-world practice support its integration into routine clinical management of these conditions.

Keywords: Lacosamide, FOS, GTCS, DRE, SE

ALLIANCE-I SURVEY – ASSESSING LONG TERM SAFETY AND EFFICACY OF LEVETIRACETAM AND CLOBAZAM IN MANAGING EPILEPSY

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Abstract ID: 96

Aim/ Background: To understand Indian specialists' perspectives on pattern of usage and efficacy-safety profile of levetiracetam and clobazam combination in epilepsy management.

Materials & Methods: Cross-sectional, pan-India e-survey among 390 consenting adult/paediatric neurologists and neurosurgeons.

Results: 44.1% respondents prescribed clobazam to 26–50% of their epilepsy patients. 55.13% and 24.36% added it in 10-25% and 25-50% of patients on levetiracetam (61.28% as first-line drug) respectively. The combination was preferred as first-line monotherapy/first add-on by 61.28% & second-line monotherapy/first add-on by 18.72%. 96.3% rated levetiracetam-clobazam combination as clinically useful; its effectiveness was rated effective/very/highly effective by 39.49%/27.69%/7.69% respectively. 92.3% agreed to its broad-spectrum utility, across a wide range of patients profiles, including - Generalized-onset (44.61%), focal-onset (43.33%), pediatric drug-resistant epilepsy (40.77%), Lennox-Gastaut syndrome etc. (35.38%). Addition was mainly for persistent seizures despite optimum levetiracetam treatment (75.89%), for rapid control (38.97%) and to mitigate levetiracetam behavioural effects (22.56%). Usual doses were 5–10 mg/day in adults (65.38%) and 0.3–0.5 mg/kg in children (68.21%). Typical duration of use was >12 months (35.9%) followed by 3-6 months (29.23%) and 6-12 months (27.44%). Major safety concerns included sedation (37.17%), dependence/withdrawal (18.46%), cognition (15.38%) and tolerance (14.87%). Other benefits of the combination cited by the respondents included anxiety relief (42.82%), circumventing need to escalate dose of individual antiepileptics (30%) and treatment of insomnia (24.87%).

Conclusion: Levetiracetam–clobazam combination is widely used and broad-spectrum, effective and generally safe regimen, often used up to a year. It is particularly useful in patients with anxiety, insomnia and needing dose escalation of individual antiepileptics.

Keywords: Levetiracetam, clobazam, combination, epilepsy management

ALLIANCE-II SURVEY – ASSESSING LONG TERM SAFETY AND EFFICACY OF LEVETIRACETAM IN MANAGING EPILEPSY: NATIONWIDE E-SURVEY

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Abstract ID: 98

Aim/ Background: To understand Indian specialists' perspectives on efficacy-safety and usage of various forms of levetiracetam in epilepsy.

Materials & Methods: Cross-sectional, pan-India e-survey among 390 consenting adult/paediatric neurologists and neurosurgeons.

Results: 63.34% respondents stated they prescribed levetiracetam as first-line monotherapy in >50% new cases, most frequently in newly-diagnosed adults with generalized-onset seizures (56.92%). 67.43%, 51.03% and 29.74% respectively cited its broad-spectrum efficacy, minimal drug-drug interactions and good tolerability in children, elderly and pregnant women as top reasons to prefer levetiracetam over other antiepileptics. 52.05% rated levetiracetam to be very-highly effective in newly-diagnosed patients. 66.92% stated usual maintenance dose as 500-1000 mg/day. 45.64% rated its tolerability as very good-excellent. 90% respondents stated $\leq 25\%$ of their patients requiring dose modification due to side effects. Behavioral/psychiatric side effects constituted the most frequent reason for discontinuation/switching (by 69.74%). 78.21% saw women of reproductive age/pregnant/lactating as the patient group wherein levetiracetam yielded its most favourable risk-benefit ratio. Respondents cited lower teratogenic potential (75.64%), no significant drug-drug interactions with oral contraceptives (48.2%), no-minimal risk of hormonal fluctuations (47.43%) and of hirsutism (27.69%) vs alternatives as levetiracetam's key benefits in women with epilepsy.

93.84% stated availability of its oral liquid formulation as a key factor impacting levetiracetam prescription in children. 59.23%, 53.58% and 31.79% respectively, stated better compliance, lesser frequency of administration and stable/predictable efficacy-safety profile as reasons to prefer levetiracetam extended-release tablets. 91.79% respondents reported they prescribed levetiracetam to prevent seizures in sub-arachnoid haemorrhage.

Conclusion: Indian specialists perceive levetiracetam as a versatile, broad-spectrum, efficacious and safe antiepileptic (especially in females).

Keywords: Levetiracetam, dosage forms, use, efficacy-safety

BEYOND CLASSICAL CIDP: ANTI-NEUROFASCIN-186 AUTOIMMUNE NODOPATHY PRESENTING AS RELAPSING MOTOR-PREDOMINANT NEUROPATHY

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Abstract ID: 153

Aim/ Background: Autoimmune nodopathies associated with antibodies against nodal proteins represent a distinct subgroup of inflammatory neuropathies with clinical and therapeutic differences from classical chronic inflammatory demyelinating polyneuropathy (CIDP). Anti-Neurofascin-186 (NF186) antibody-associated neuropathy is uncommon and remains incompletely characterized. We report a young male with relapsing motor-predominant neuropathy with isolated NF186 antibody positivity and variable response to immunotherapy

Materials & Methods: Clinical records, neurological examination findings, laboratory investigations, and treatment details of a 23-year-old male with relapsing motor neuropathy were reviewed. Autoimmune evaluation including nodal antibody panel was performed. Treatment responses to corticosteroids, plasma exchange, immunosuppressive agents, rituximab, and intravenous immunoglobulin (IVIg) were analyzed.

Results: The patient initially presented in 2022 with acute progressive quadriparesis and was diagnosed as Guillain-Barré syndrome with partial improvement following IVIg. Subsequent relapses led to a revised diagnosis of motor-predominant CIDP. Despite treatment with corticosteroids, azathioprine, mycophenolate mofetil, plasma exchange, and rituximab, he continued to have fluctuating weakness. Neurological examination revealed pure motor involvement with preserved sensory functions and reduced or absent deep tendon reflexes. Serum nodal antibody testing demonstrated isolated anti-Neurofascin-186 positivity, while Neurofascin-155, Contactin-1, and Caspr1 antibodies were negative. Repeated maintenance IVIg provided the most consistent clinical benefit, although symptoms recurred before subsequent doses.

Conclusion: Anti-NF186 autoimmune nodopathy is rare and may present with a relapsing motor phenotype mimicking CIDP. Recognition of nodal antibodies is important because these disorders differ from classical CIDP in pathophysiology and therapeutic response. Our case highlights the heterogeneity of treatment outcomes and the importance of antibody profiling in patients with atypical or treatment-refractory inflammatory neuropathies.

Isolated anti-Neurofascin-186 positivity should be considered in relapsing motor neuropathies with suboptimal response to conventional therapy. Early identification of autoimmune nodopathy may facilitate individualized treatment strategies and improve patient outcomes.

Keywords: autoimmune demyelination NF186 polyneuropathy

AKINETIC-RIGID PARKINSONISM FOLLOWING NEUROTOXIC SNAKEBITE: A RARE CASE REPORT

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Abstract ID: 66

Aim/ Background: Background: Snakebite is a major public health problem in India, causing nearly 50,000 deaths annually. While neurotoxic envenomation commonly presents with acute neuromuscular paralysis, delayed movement disorders are exceedingly rare. We report a case of delayed akinetic-rigid parkinsonism following recovery from snakebite.

Materials & Methods: case report prepared after due consent.

Results: Case Report: A 40-year-old construction worker developed ptosis, neck weakness, quadriparesis, respiratory failure, and altered sensorium approximately 90 minutes after a snakebite. He required mechanical ventilation and anti-snake venom therapy, following which he recovered completely and was discharged after five days. Approximately seven days after the bite, he developed blurred vision, progressive bradykinesia, and rigidity. Examination revealed mask-like facies, aphonia, axial and appendicular rigidity, slow horizontal and vertical saccades, broken pursuits, brisk deep tendon reflexes, and flexor plantar responses. Contrast-enhanced MRI brain demonstrated symmetrical T2/FLAIR hyperintensities involving the bilateral caudate nuclei and putamina with additional frontoparietal cortical hyperintensities, without diffusion restriction or contrast enhancement. MR spectroscopy was normal. EEG showed generalized 4-Hz delta slowing, while cerebrospinal fluid and routine laboratory investigations were unremarkable. The patient was treated with levodopa/carbidopa, trihexyphenidyl, intravenous methylprednisolone, and later amantadine, with only partial improvement, remaining non-ambulatory at two months.

Conclusion: Conclusion: Delayed parkinsonism is a rare neurological complication of snakebite and may represent immune-mediated or toxin-induced basal ganglia injury. Bilateral basal ganglia hyperintensities on MRI provide an important diagnostic clue. Clinicians should recognize this uncommon presentation in patients developing delayed extrapyramidal symptoms after apparent recovery from neurotoxic snake envenomation.

Keywords: snake bite, parkinsonism, basal ganglia

BRAIN EATING STROKE: A CASE SERIES OF FREE LIVING AMOEBA MASQUERADING AS STROKE

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Abstract ID: 141

Aim/ Background: Free-living amoebae, *Naegleria*, *Acanthamoeba*, and *Balamuthia* are known as opportunistic and deadly protozoa in humans and animals. They are widely found in soil and water. They cause primary amoebic meningoencephalitis (PAM) and granulomatous meningitis, which usually result in death.

While the common presentation of CNS infiltration of FLA in humans is similar to other infective meningoencephalitis, rare case reports of them presenting as stroke have been found.

Materials & Methods:

Case 1: A 42 year old male presented with acute bilateral cerebellar infarcts with right parieto-occipital infarction.

Patient was discharged after acute stroke management but reported back with fever, neck rigidity and altered sensorium. CSF picture suggestive of meningitis, did not respond to routine therapy. CSF hanging drop preparation showed FLA which was treated and patient improved.

Case2 :A 70 year old female presented with malignant left MCA infarct but leptomeningeal enhancements led on to further investigation. Her CSF picture was suggestive of meningitis and hanging drop preparation positive for FLA. She was started on five drug regimen and later expired.

Case 3: 67 year old male presented with IVH. On admission, he developed features of meningitis and CSF was supportive. Hanging drop preparation showed presence of FLA. Patient received 14 days of in hospital treatment and was discharged.

Results: Fatma GER AKARSU et al. reported a case of *Naegleria* meningitis presenting with ischemic infarct in Turkey.

Conclusion:

Atypical stroke work up, especially in tropical countries, when no other more common causes of stroke are found should involve a workup for meningitis, and if necessary a search for motile trophozoites.

Keywords: Stroke, Free living Amoeba

MENINGOENCEPHALITIS

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Abstract ID: 78

Aim/ Background: This is a case of a male patient admitted with fever headache and vomiting

Materials & Methods: He was treated as a case of meningitis with iv antibiotics and supportive measures since he didn't show improvement further investigations were sent

Results: This showed evidence of trophozoites in CSF with mri showing multiple abscesses. This was then confirmed to be a case of acanthamoeba meningoencephalitis

Conclusion: Amoebic meningoencephalitis outbreak has been recently seen in kerala mostly after exposure to water bodies. Patient improved with iv antibiotics, fluconazole and cotrimoxazole for a duration of 28 days

Keywords: Amoebic meningoencephalitis

SUCCESSFUL MECHANICAL THROMBECTOMY BEYOND 24 HOURS IN BASILAR ARTERY OCCLUSION: A CASE OF REMARKABLE NEUROLOGICAL RECOVERY

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Abstract ID: 63

Aim/ Background: Basilar artery occlusion (BAO) is a neurological emergency associated with high mortality and severe disability. Although mechanical thrombectomy is recommended within 24 hours in selected patients, evidence for intervention beyond this window remains limited. We report a case of successful thrombectomy performed more than 24 hours after symptom onset with favorable neurological recovery.

Materials & Methods: A 37-year-old male presented with vomiting followed by altered sensorium for 2 days. On admission, GCS was E1V1M1, requiring intubation and mechanical ventilation. He was hypotensive (80/40 mmHg) requiring noradrenaline support. Neurological examination revealed bilateral non-reactive pupils. MRI brain showed multiple acute infarcts involving the bilateral ventromedial thalami, cerebral peduncles, pons, left brachium pontis, vermis, bilateral occipital lobes, and left cerebellar hemisphere. Digital subtraction angiography demonstrated basilar artery top occlusion, and mechanical thrombectomy was performed successfully.

Results: Following successful recanalization, the patient showed gradual neurological and hemodynamic improvement. Vasopressor support was discontinued, ventilatory support was successfully weaned, and neurological status improved progressively. At present, GCS has improved to E3V2M6.

Conclusion: This case highlights that selected patients with BAO may derive meaningful benefit from mechanical thrombectomy even beyond 24 hours of symptom onset. Advanced imaging-based patient selection, rather than strict time criteria alone, may help identify candidates for late-window endovascular therapy and improve outcomes in this otherwise devastating condition.

Keywords: BAO- Basilar Artery Occlusion

DANCING TO AN AUTOIMMUNE TUNE: CHOREA AS THE INITIAL MANIFESTATION OF ANTIPHOSPHOLIPID SYNDROME IN YOUNG MALE

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Abstract ID: 104

Aim/ Background: Anti-phospholipid syndrome (APS) commonly occurs in females with vascular thrombosis and obstetric complications. Chorea as initial manifestation of the disease is rare and seen in 0.9 to 2% of cases.

Here we discuss a rare case of chorea presenting as initial manifestation of Primary APS in a young male.

Materials & Methods: An 18-year-old nil comorbid male born out of non-consanguineous marriage presented with complaints of behaviour disturbances for 2 months and involuntary movements of upper-limb and lower-limb for 20 days and on examination found to have choreiform movements involving all four limbs (right > left) with perioral and preorbital involuntary movements, spooning of hand and milkmaid grip and normal Cranial nerve, motor, sensory and cerebellar examination.

Extensive evaluation for secondary causes of chorea was unrevealing, including infectious, inflammatory, metabolic, autoimmune encephalitis, and Wilson disease workup. Autoimmune testing demonstrated positive lupus anticoagulant (DRVVT ratio 1.97) and anticardiolipin IgG (23 PIU/mL), while ANA, ANCA, anti- β 2 glycoprotein, and other investigations were negative. MRI brain showed acute lacunar infarct in right centrum semiovale and prominent perivascular spaces in bilateral peritrigonal white matter. (MRI and Movement disorder video available).

Results: The patient fulfilled the diagnostic criteria for primary APS presenting with chorea. Treatment with Tetrabenazine for symptomatic relief and Nicoumalone for long-term anticoagulation resulted in marked clinical improvement during follow-up, with significant reduction in involuntary movements.

Conclusion: This case illustrates that APS can initially manifest as an isolated movement disorder and the need for testing for autoimmune disorders in initial workup for new onset chorea to diagnose and adequately treat to prevent fulminant disease.

Keywords: Antiphospholipid syndrome , Chorea , Young Male

ONE ANTIBODY, TWO STORIES: A TWO-CASE SERIES OF CASPR2 AUTOIMMUNITY WITH DISTINCT CLINICAL ASSOCIATIONS

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Abstract ID: 122

Aim/ Background: CASPR2 autoimmunity is a rare immune-mediated neurological disorder with diverse clinical manifestations. While thymoma is the best-recognized association, infectious and environmental triggers remain poorly understood. Although the association between tuberculosis and CASPR2 autoimmunity is not well established, tuberculosis may plausibly contribute to autoimmunity through immune dysregulation.

Materials & Methods: We describe the clinical presentation, investigations, treatment, and follow-up of two patients with CASPR2 autoimmunity managed at a tertiary care centre.

Results: A 53-year-old man diabetic, presented with one month of recurrent stereotyped episodes characterized by transient dysarthria, post-event confusion, behavioural changes, severe insomnia, giddiness, and labile blood pressure. Cerebrospinal fluid (CSF) was sterile with positive CASPR2 antibodies. During evaluation for occult malignancy, bronchoalveolar lavage and CT-guided lung biopsy established pulmonary tuberculosis and malignancy was excluded. He was treated with rituximab alongside antitubercular therapy, resulting in sustained neurological recovery. A 43-year-old woman presented with one month of severe insomnia, visual hallucinations, myokymia, and cognitive dysfunction. CSF was sterile, while electromyography demonstrated peripheral nerve hyperexcitability. Serum CASPR2 antibodies were positive, malignancy screening was negative, and serum mercury levels were markedly elevated in the setting of prolonged Ayurvedic medication use. She showed marked clinical improvement following intravenous immunoglobulin and rituximab.

Conclusion: These cases broaden the spectrum of clinical associations observed in CASPR2 autoimmunity. Although causality cannot be established, tuberculosis may represent a potential autoimmune trigger in susceptible individuals through immune dysregulation. In endemic regions, clinicians should actively evaluate for tuberculosis and environmental triggers like heavy metal exposure. Early neuronal surface antibody testing and timely immunotherapy may improve neurological outcomes.

Keywords: CASPR2 autoimmunity, Tuberculosis, Mercury exposure

FAHR'S DISEASE PRESENTING AS CORTICOBASAL SYNDROME: A CASE REPORT

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Abstract ID: 136

Aim/ Background: Primary familial brain calcification (Fahr's disease) is a rare neurodegenerative disorder classically causing parkinsonism, cognitive decline, and psychiatric symptoms. Presentation mimicking corticobasal syndrome (CBS) is exceedingly uncommon. We describe a patient with radiologically confirmed Fahr's disease manifesting as CBS.

Materials & Methods: A 59-year-old female, with known diabetes mellitus (10 years) and hypertension (8 years), presented with a 4-year history of progressive slowness of activities of daily living, right hand grip weakness, and handwriting deterioration, followed by 2 years of cognitive decline (recent memory loss) and 2 months of gait imbalance with recurrent falls. Examination revealed hypophonic speech, mask facies, right-sided bradykinesia, right upper-limb cogwheel rigidity, slow saccades with broken pursuits, reduced arm swing gait, and probable right upper-limb ideomotor apraxia — an asymmetric akinetic-rigid syndrome with apraxia and cognitive impairment consistent with CBS. Outside MRI brain reported bilateral basal ganglia, thalamic, and dentate nuclei calcification. Serum PTH was mildly elevated (73), with otherwise unremarkable calcium-phosphate metabolism workup.

Results: A diagnosis of Fahr's disease presenting as corticobasal syndrome was made. The patient was managed with carbidopa-levodopa (up-titrated), memantine for cognitive symptoms, and supportive measures for constipation and sleep.

Conclusion: Fahr's disease should be considered in the differential diagnosis of CBS, particularly when asymmetric parkinsonism, apraxia, and cognitive decline coexist with characteristic bilateral basal ganglia, thalamic, and dentate calcification on imaging. Early recognition guides appropriate metabolic evaluation and symptomatic management.

Keywords: Fahr's disease, Corticobasal syndrome

ARRESTED SUBACUTE SCLEROSING PANENCEPHALITIS WITH FUNCTIONAL SURVIVAL INTO ADULTHOOD: A CASE REPORT

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Abstract ID: 144

Aim/ Background: Subacute sclerosing panencephalitis (SSPE) is a rare, progressive, and usually fatal complication of measles infection. Most patients die within 1–3 years after neurological symptoms begin. Long-term functional survival is uncommon. We report a patient with SSPE who showed prolonged clinical stabilization and remained functionally alive eight years after disease onset.

Materials & Methods: Retrospective review of clinical records, serial cerebrospinal fluid (CSF) measles antibody indices, serial EEG, serial neuroimaging, genetic testing and treatment response of a young adult with SSPE.

Results: A 19-year-old left-handed male who had not received measles vaccination developed progressive visual loss, myoclonus, cognitive decline, gait ataxia with recurrent falls, and one generalized tonic–clonic seizure at 11 years of age. Cerebrospinal fluid analysis demonstrated a markedly elevated measles IgG titre with a positive CSF/serum antibody index, confirming intrathecal antibody synthesis. The initial EEG showed characteristic periodic generalized complexes, which later evolved into diffuse background slowing with theta–delta activity. Serial MRI scans from 2017 to 2025 revealed progressive bilateral parieto-occipital, temporal, and periventricular white matter changes, followed by generalized cerebral atrophy, corpus callosum thinning, and ventriculomegaly, while subcortical U-fibers remained relatively preserved. Targeted genetic testing for CLN3 did not identify a pathogenic variant. The patient received long-term treatment with oral ribavirin, isoprinosine, levetiracetam, clobazam, and valproate. Intrathecal interferon was not administered because of financial constraints. Despite continued radiological progression, his neurological status stabilized. Eight years after symptom onset, he remains alive, ambulates with support, recognizes family members, and retains meaningful functional abilities.

Conclusion: This case demonstrates that prolonged clinical stabilization and functional survival into adulthood are possible in selected patients with SSPE, even when MRI shows ongoing disease progression. Early diagnosis and sustained combination antiviral and immunomodulatory therapy may help slow clinical decline. The case also highlights the critical importance of measles vaccination in preventing this devastating but largely avoidable disease.

Keywords: SSPE

EIGHT-AND-A-HALF SYNDROME: A RARE BRAINSTEM STROKE SYNDROME – A CASE REPORT

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Abstract ID: 131

Aim/ Background: Eight-and-a-half syndrome is a rare brainstem syndrome resulting from a unilateral lesion involving the medial longitudinal fasciculus, paramedian pontine reticular formation or abducens nucleus, and facial nerve fascicle. We report a rare case of left eight-and-a-half syndrome secondary to acute pontine infarction, emphasizing the importance of bedside neuroanatomical localization

Materials & Methods: A patient presenting with acute binocular diplopia and left facial weakness underwent detailed neurological examination and neuroimaging. Ocular motility, cranial nerve findings, and MRI brain were evaluated to establish the diagnosis.

Results: Examination revealed left horizontal gaze palsy, left internuclear ophthalmoplegia with abducting nystagmus of the right eye, and left lower motor neuron facial palsy, consistent with left eight-and-a-half syndrome. MRI brain demonstrated an acute infarction involving the left dorsal pontine tegmentum affecting the medial longitudinal fasciculus, paramedian pontine reticular formation/abducens nucleus, and facial nerve fascicle. Appropriate acute stroke management and secondary prevention were initiated

Conclusion: Eight-and-a-half syndrome is an uncommon but clinically distinctive localization syndrome. Recognition of its characteristic clinical triad enables accurate localization to the dorsal pontine tegmentum, facilitates prompt diagnosis of brainstem stroke, and allows early institution of appropriate management, thereby improving patient outcomes.

Keywords: Eight-and-a-half syndrome; Pontine infarction; Internuclear

BILATERAL MEDIAL MEDULLARY STROKE-THE HEART SIGN

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Abstract ID: 107

Aim/ Background: To present a rare case of bilateral medial medullary infarction (BMMI) with its characteristic "Heart Sign" on diffusion-weighted MRI.

To highlight the clinical presentation, neuroanatomical correlation, vascular etiology, and radiological features of this uncommon posterior circulation stroke.

Materials & Methods: Single-patient case report.

Patient

A 55-year-old man presented with acute onset weakness of all four limbs, dysarthria, dysphagia, and gait imbalance.

Results: Bilateral medial medullary infarction is a rare but important cause of acute quadriparesis and bulbar dysfunction. The Heart Sign on diffusion-weighted MRI is a characteristic radiological marker that facilitates early diagnosis.

Conclusion: Bilateral medial medullary infarction (BMMI) is an extremely rare subtype of posterior circulation ischemic stroke, accounting for less than 1% of all cerebral infarctions. It most commonly results from occlusion of the anterior spinal artery or the paramedian branches of the vertebral artery, leading to bilateral ischemia of the medial medulla.

The medial medulla contains several critical neuroanatomical structures, including the corticospinal tracts (pyramids), medial lemniscus, hypoglossal nucleus and fascicles, and medial longitudinal fasciculus. Consequently, patients typically present with rapidly progressive quadriparesis or tetraplegia, dysarthria, dysphagia, tongue weakness, impairment of proprioception and vibration sensation, and occasionally respiratory failure due to extensive brainstem involvement.

Keywords: MEDIAL MEDULLARY STROKE, HEART SIGN

STROKE MIMICKING AS PARAKINESIA BRACHITIS OSCITANS WITH SUPERIMPOSED CHOREIFORM MOVEMENTS- “CLOCK THAT TRIGGERS LIMB ELEVATION”

Dr. Chandra Mohan Bhardwaj

RAJASTHAN

Abstract ID: 126

Aim/ Background: To Describe the rare phenomenon-The rare phenomenon of abnormal involuntary movement of paralyzed upper limb in association with yawning following stroke termed as Parakinesia Brachitis Oscitans(PBO).

Materials & Methods: Case Report

Results: We report a case of 66 year old female who presented with left upper and lower limb weakness , with involuntary dance like movements of left upper and left lower limb (Choreiform), with slurring of speech , with parakinesias (another action) in form of twisting, elevating left upper limb while walking, yawning. MRI brain showed acute infarct in Right Putamen, right posterior limb of internal capsule, Patient was started on Dual antiplatelets, and Statins along with physiotherapy

Conclusion:

- Parakinesia brachitis oscitans is a rare unique phenomenon, which occurs following stroke , triggered by yawn
- Clinicians should be aware of this transient phenomenon which should not be mistaken as a chronic movement disorder or focal epileptic seizures.

Keywords: Parakinesia Brachitis Oscitans, Choreiform, Yawn,

A RARE BLEND OF 6TH AND 7TH CRANIAL NERVE PALSY – EIGHT AND HALF SYNDROME SECONDARY TO INFARCT

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Kota, Rajasthan

Abstract ID: 127

Aim/ Background: TO DELINEATE HOW EIGHT AND HALF SYNDROME IS HELPFUL IN CLINICAL LOCALISATION IN STROKE PATIENTS OF ANY AETIOLOGY

Materials & Methods: CASE REPORT

Results: .We report a case of 84-year-old gentleman, retired stenographer, who presented to emergency department, with vomiting 3-4 episodes, with imbalance on walking towards left side, with binocular diplopia, Cranial nerve examination revealed- Right eye adduction defect with left abducting eye nystagmus, with Right LMN facial palsy, MRI brain with stroke Protocol one and half showed acute infarct in Tegmentum of Pons.

Conclusion: Brainstem strokes are difficult to localise but extremely fascinating. Eight-and-a-half syndrome is a rare neurological condition characterised by loss of ipsilateral horizontal conjugate eye movement, internuclear ophthalmoplegia and lower motor neuron type facial palsy. It is due to a lesion affecting median longitudinal fasciculus (MLF), paramedian pontine reticular formation (PPRF) and facial nerve fascicle on the same side at the level of pons. The PPRF is located at the level of pons. It is the centre for horizontal conjugate gaze. At the same time, MLF facilitates the yoked eye movement. The common causes include stroke and demyelinating conditions but can also be seen in cases of space occupying lesions of brain

The term 'eight-and-a-half' syndrome was coined by Eggenberger. When there is a lesion of ipsilateral PPRF, MLF and facial nerve fascicles resulting in one-and-a-half syndrome with lower motor neuron type facial nerve palsy, this constellation of signs is termed eight-and-a-half syndrome. Eight and Half syndrome is rare and can be missed if not recognised during clinical evaluation.

Keywords: Eight and Half Syndrome, INO, Facial Palsy

NODES AND BEYOND

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Abstract ID: 26

Aim/ Background: Immune-mediated neuropathies have expanded beyond the classical Guillain–Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) spectrum to include autoimmune nodopathies, characterized by antibodies targeting nodal and paranodal proteins. These disorders are increasingly recognized as distinct entities with unique clinical, electrophysiological, and therapeutic profiles. However, they are frequently underrecognized due to overlap with GBS, CIDP, motor neuron disease (MND), and painful neuropathy syndromes.

Materials & Methods: We retrospectively analyzed nine patients presenting with atypical immune-mediated neuropathies, treatment resistance, or treatment-related fluctuations. Clinical presentation, serial nerve conduction studies (NCS), CSF findings, MRI features, treatment response, and antibody profiles were reviewed. Antibody testing included neurofascin-140 (NF140), neurofascin-155 (NF155), neurofascin-186 (NF186), CASPR2, sulfatide IgM, and neuronal antibody panels using ELISA and cell-based assays.

Results: The cohort demonstrated a broad clinical spectrum including acute GBS-like presentations, relapsing nodopathy, CIDP-like neuropathy, multifocal motor neuropathy/MND mimic, mononeuritis multiplex, painful small-fiber neuropathy, and immune brachial plexitis.

Conclusion: Autoimmune nodopathies represent a heterogeneous but mechanistically distinct group of immune neuropathies characterized by nodal/paranodal dysfunction rather than classical demyelination. Clinical clues such as tremor, severe neuropathic pain, autonomic dysfunction, treatment fluctuation, conduction block without temporal dispersion, and IVIG resistance should prompt consideration of nodopathy.

Keywords: Autoimmune nodopathy; Neurofascin antibodies; CIDP; Guillain

AI-ASSISTED MULTIDOMAIN SCORING OF MULTIPLE SCLEROSIS DISABILITY AND DISEASE ACTIVITY

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Abstract ID: 27

Aim/ Background: Conventional disability assessment in multiple sclerosis (MS), particularly the Expanded Disability Status Scale (EDSS), remains heavily weighted toward ambulation and may under-represent cognition, visual dysfunction, fatigue, biological activity, and subclinical disease progression. This limitation is especially relevant in settings such as India, where delayed diagnosis, optic-spinal presentations, high mimic burden, limited biomarker access, and socio-economic barriers influence clinical interpretation.

Materials & Methods: To propose an artificial intelligence-assisted multidomain scoring framework for MS that integrates clinical, cognitive, imaging, electrophysiological, and biofluid parameters to improve ease, consistency, and depth of disability and disease activity assessment.

Results: The model is designed around five domains: cognition, clinical disability, biofluids, electrophysiology, and imaging. Artificial intelligence can assist by standardizing scale administration, reducing manual scoring burden, integrating heterogeneous data, flagging progression independent of relapse activity, generating longitudinal visual summaries, and supporting risk stratification. The tool may be especially useful for follow-up visits, registry-based data capture, tele-neurology, and resource-stratified clinical decision support.

Conclusion: An AI-enabled multidomain MS scoring model may complement EDSS by capturing visible and invisible disability, biological activity, and imaging progression. Prospective validation in diverse Indian and global cohorts is required before adding in clinical practice.

Keywords: Multiple sclerosis; artificial intelligence; EDSS

EMERGING GENOTYPIC-PHENOTYPIC PATTERNS IN SPINOCEREBELLAR ATAXIA TYPE 8: A SYSTEMATIC REVIEW OF GLOBALLY PUBLISHED CASES

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Abstract ID: 25

Aim/ Background: Spinocerebellar ataxia type 8 is an autosomal dominant neurodegenerative disorder characterised by slowly progressive ataxia with onset in the third to fifth decade .Despite of numerous data , there is a lack of data representing genotypic-phenotypic patterns, leaving gaps in understanding of disease.

Materials & Methods: A systematic review without meta analysis of published case reports and case series of SCA8 was performed .Extracted data from articles has been compiled .Risk of bias has been assessed using Joanna Briggs Institute Critical Appraisal Tools.

Results: A total of 58 cases from 37 articles were reviewed. Mean onset of age of symptoms is 34.99 ± 19.05 years. Clinical presentation was heterogeneous, with majority clinical features are gait disturbances (89.66%), dysarthria (58.62%), motor symptoms in limbs other than gait disturbances(51.72%), cognitive problems(36.21%), psychiatric problems (25.86%), dysphagia(24.14%), and others. On neurological examination, predominantly revealed cerebellar signs including gait ataxia(86.21%), dysadochokinesia (20.69%) and dysmetria (25.86%). The correlation between age of onset and number of repeats is inconsistent. The majority of patients demonstrate cerebellar atrophy (67.24%) on neuro imaging. SYNE1 variants were identified in 7 patients, whereas SCA8 repeat expansions were reported in 51 patients and SYNE variants have an early age of onset of symptoms.

Conclusion: Repeat size does not reliably predict disease onset. For diagnosis of SCA8 a thorough clinical, neurological, radiological evaluation, and genetic testing has to be done.

Keywords: Spinocerebellar Ataxia Type 8, Ataxia, Movement Disorders

A RARE CLINICAL CONFLUENCE ; SUCCESSFUL PREGNANCY IN A PATIENT WITH NMOSD

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Abstract ID: 61

Aim/ Background: To highlight the importance of early immunotherapy in a pregnant patient diagnosed with NMOSD during pregnancy.

Materials & Methods: A 26 years old pregnant female with a gestational age of 29 weeks presented with acute onset paraparesis. MRI spine suggested LETM from C1 to D4 Segments. She was tested positive for anti-AQP 4 antibodies. She was diagnosed with NMOSD and managed with IV Methylprednisolone followed by IVIG x 5 days along with other supportive treatment.

Results: Patient showed gradual recovery. At 37 weeks, she went on spontaneous labour and had a normal vaginal delivery and delivered a baby weighing 2.2 kg. She showed marked neurological improvement with a power of 4/5 in both lower limbs at the time of delivery.

Conclusion: Pregnancy associated with NMOSD is a rare condition which presents significant diagnostic and therapeutic challenges. This case highlights the importance of early recognition of NMOSD in a pregnant patient presenting with acute onset paraparesis. High dose IV Methylprednisolone remains the first line therapy for acute attacks. In our case, IVIG was also administered because of severe neurological deficits and favourable maternal safety profile. The patient showed significant neurological recovery following early immunotherapy and rehabilitation. Unlike previously reported cases of NMOSD in pregnancy, which were complicated by recurrent relapses, emergency delivery, caesarean section or permanent neurological sequelae, our patient remained clinically stable during follow up and had a normal vaginal delivery.

Keywords: NMOSD, PREGNANCY, PARAPARESIS, ANTI-AQP4, IVIG

CENTRAL VARIANT OF PRES: A CASE SERIES

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Abstract ID: 408

Aim/ Background: To describe the clinical presentations and radiological characteristics of patients diagnosed with the central variant of PRES.

Materials & Methods: We prospectively identified four patients who fulfilled the clinico radiological criteria for central variant PRES. Detailed clinical features, laboratory parameters, and MRI findings were analyzed.

Results: The first patient had accelerated hypertension with encephalopathy and a pan-cerebellar syndrome. The second patient, also hypertensive, developed subacute visual blurring, headache, dysarthria, gait ataxia, and bulbar symptoms. The third patient presented with fever, diplopia, dysarthria, gait ataxia, encephalopathy and hypertension. The fourth patient had acute encephalopathy with dysarthria and cerebellar dysfunction progressing to an akinetic-mute state. MRI in all cases demonstrated predominant involvement of central structures – brainstem, thalami, deep white matter, or basal ganglia – with relative cortical sparing, consistent with central PRES.

Conclusion: The central variant of PRES is a rare subtype in which the brainstem, basal ganglia, thalami, or periventricular white matter are primarily affected, with relative sparing of the cortical regions. Central PRES should be considered in patients presenting with unexplained brainstem or deep gray matter lesions, especially in the setting of hypertensive crisis or CKD. Early identification and prompt management of BP and precipitating factors are crucial parts of management.

Keywords: PRES, Posterior Reversible Encephalopathy Syndrome, Central variant

WHEN MOTOR OUTPUT BECOMES THE BOTTLENECK OF INHIBITION IN PARKINSON'S DISEASE

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Abstract ID: 42

Aim/ Background: Inhibitory control deficits in Parkinson's disease (PD) have long been conventionally localised to fronto-striatal dysfunction. The framework of this study implicitly assumes that performance on conflict tasks reflects cognitive processing limits. We tested this competing hypothesis that motor execution constraints, at corticospinal and spinal levels, constitute a limiting stage in inhibitory control expression.

Materials & Methods: 46 PD subjects underwent bilateral-response of Flanker Task assessment alongside structured upper limb neurological examination (deep tendon reflexes, tone, and MRC strength). Reaction time (RT), accuracy, and congruency cost were analysed using non-parametric and regression approaches.

Results: Subjects demonstrated globally impaired performance (RT: 659 ± 154 ms; accuracy: 40.6%), indicating operation near a cognitive-motor performance ceiling. Notably, the canonical flanker congruency effect was absent at group level, not due to preserved inhibition, but due to response saturation under an RT ceiling constraint, collapsing variance between conditions.

Crucially, effector-specific motor signatures emerged:

Left-sided MRC strength showed consistent moderate associations with congruent and mean RT ($\rho \approx 0.30$ – 0.32), indicating that response execution capacity systematically modulates task latency.

Right triceps DTR negatively predicted congruency cost ($\rho = -0.301$), implicating spinal reflex excitability in conflict resolution dynamics.

In contrast, composite motor measures failed to explain variance in multivariate models ($R^2 < 0.12$), indicating that motor contributions to inhibitory control are localized, not global.

Conclusion: These findings challenge the dominant cortical-centric model of inhibitory control in PD. Instead, they support a motor-limited framework, where inhibitory performance reflects the interaction between response selection and downstream execution constraints. The data suggest that corticospinal and spinal excitability are integral components of conflict resolution, not merely output pathways.

Methodologically, the demonstration of performance ceiling-induced collapse of the flanker effect highlights a systematic underestimation of inhibitory deficits in PD literature.

This study positions motor output not as a passive endpoint, but as an active determinant of cognitive performance, redefining the motor-cognitive interface in Parkinson's disease.

Keywords: Inhibitory Control, Motor–Cognitive Interaction

THE EFFECTS OF REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION AT DORSOLATERAL PREFRONTAL CORTEX IN REDUCING THE DISABILITY OF PATIENTS WITH CHRONIC MIGRAINE

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Abstract ID: 20

Aim/ Background: Chronic Migraine causes significant disability to patient. Repetitive Trans cranial Magnetic Stimulation(rTMS) at dorsolateral prefrontal cortex can be an effective treatment modality. The present study aims to study the efficacy of high frequency rTMS over left dorsolateral prefrontal cortex for reducing the disability of migraine.

Materials & Methods: Patients fulfilling International classification of headache disorders(ICHD 3) criteria were retrospectively enrolled between July 2025 to December 2025. Severity of pain was determined by visual analog scale(VAS). Migraine Disability Assessment (MIDAS) were calculated. All patients were given 12 session of 600 pulses of rTMS at left dorolateral prefrontal region(F3) at a frequency of 10 Hz at intensity of 90% of resting motor threshold

Results: Out of 15 patients included in the study, majority (75%) were females. Mean age were 34.4 ± 9.6 years. Mean duration of illness were 3.7 ± 2.1 years. The mean MIDAS score reduced significantly from 57.6 ± 14.8 at baseline to 36.4 ± 15 . after rTMS($p=0.01$). During follow up at 4 weeks after last session, MIDAS score were significantly low (43.8 ± 16.4 , $p=0.03$) compared to baseline.

Conclusion: The study showed that rTMS has a distinctive role in reducing the disability in chronic Migraine. The improvement was sustained over the next four weeks period sugesting that the response was less likely to be due non-specific factors or a placebo effect.

Keywords: Chronic Migraine, rTMS, disability

A Middle-Aged Man with Weight Loss, Weakness, and Autonomic Dysfunction: Expect the Unexpected

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Abstract ID: 405

Aim/ Background: Lambert–Eaton myasthenic syndrome (LEMS) is a rare presynaptic neuromuscular junction disorder that is frequently associated with an underlying malignancy, particularly small-cell lung carcinoma. We present a case highlighting the characteristic clinical and electrophysiological features of LEMS and emphasize the importance of early recognition and evaluation for an occult malignancy.

Materials & Methods: A middle-aged man presented with a 9-month history of gradually progressive, symmetric proximal weakness involving all four limbs, initially affecting the lower limbs followed by the upper limbs. The weakness demonstrated diurnal variation with relative improvement later in the day. Over the preceding 3 months, he developed fatigable bulbar symptoms in the form of difficulty chewing, resulting in reduced intake of solid food. During the last 2 months, he also reported autonomic symptoms including dry mouth and bladder dysfunction, accompanied by significant weight loss. There were no sensory symptoms, distal weakness, muscle wasting, or fasciculations.

Results: Nerve conduction studies demonstrated a markedly reduced baseline compound muscle action potential (CMAP) amplitude in the median nerve (0.36 mV), which increased to 3.2 mV following brief exercise, consistent with a significant post-exercise increment. Repetitive nerve stimulation showed characteristic findings of a presynaptic neuromuscular junction disorder. FDG-PET imaging revealed a metabolically active spiculated nodule in the right upper lobe of the lung with associated mediastinal and retroperitoneal lymphadenopathy, raising strong suspicion for an underlying malignancy.

Conclusion: This case highlights the importance of considering LEMS in patients presenting with proximal weakness, fatigable bulbar symptoms, autonomic dysfunction, and unexplained weight loss. Characteristic electrophysiological findings are pivotal for diagnosis and should prompt a thorough search for an underlying malignancy. Early recognition facilitates timely oncological evaluation and appropriate management.

Keywords: Lambert–Eaton myasthenic syndrome (LEMS); Paraneoplastic neurological syndrome; Small-cell lung carcinoma; Repetitive nerve stimulation.

Demyelinating Neuropathy Beyond CIDP: Clinical Clues to POEMS Syndrome

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Abstract ID: 407

Aim/ Background: To describe the clinical spectrum of POEMS syndrome–associated neuropathy and highlight diagnostic challenges, particularly in patients initially fulfilling criteria for chronic inflammatory demyelinating polyneuropathy (CIDP).

Materials & Methods: We report a case series of three patients presenting with progressive sensorimotor neuropathy involving both upper and lower limbs. Patients 1 and 3 had a length-dependent, distal symmetric polyneuropathy, while patient 2 had predominantly distal symptoms. All patients experienced severe small-fibre neuropathic symptoms. Erectile dysfunction was reported by patients 1 and 2. Systemic features included progressive skin hyperpigmentation, fatigue, and unintentional weight loss in all patients. On examination, hepatosplenomegaly was present in all cases, lymphadenopathy in 2, pedal edema in 2, and ascites in 1 patient.

Results: Nerve conduction studies met the 2021 EFNS/PNS diagnostic criteria for CIDP in all patients, with terminal latency indices greater than 0.4 in each case. Serum immunofixation electrophoresis revealed a monoclonal gammopathy in two patients, with IgG lambda, and in one patient, IgA lambda. Endocrinopathy was documented in all patients. Imaging with low-dose CT and/or PET scans demonstrated sclerotic bone lesions in all cases. Serum vascular endothelial growth factor (VEGF) levels were within normal limits in the two patients in whom they were measured. Despite initial electrophysiological features suggestive of CIDP, all patients ultimately fulfilled established diagnostic criteria for POEMS syndrome.

Conclusion: POEMS syndrome is a rare paraneoplastic multisystem disorder associated with plasma cell dyscrasia and frequently masquerades as CIDP. Clinicians should consider POEMS syndrome in patients with CIDP who exhibit prominent small-fibre symptoms, systemic features, or poor response to standard immunotherapy.

Keywords: OFFLINE

ROBOT-ASSISTED VS STANDARD REHABILITATION IN UKRAINIAN VETERANS WITH UPPER LIMB AMPUTATION: PSYCHOSOCIAL AND COGNITIVE OUTCOMES

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Abstract ID: 7

Aim/ Background: Upper limb amputation in military personnel leads to significant psychological and cognitive challenges. Robotic rehabilitation (PABLO®) may improve outcomes, but comparative data versus standard therapy are limited.

Materials & Methods: A prospective study included 54 Ukrainian veterans; 28 received PABLO® and 26 standard therapy, 3 sessions/week for 8 weeks. Outcomes (PCL-5, GAD-7, PHQ-9, MoCA) were measured at days 0, 14, 30, and 120. Repeated measures ANOVA assessed within- and between-group differences; η^2 quantified effect size.

Results: PCL-5: Robot group median (IQR) 45 (42–48), 42 (39–45), 39 (36–42), 37 (34–40); control 44 (41–47), 43 (41–45), 42 (40–44), 41 (39–43). ANOVA $F(3,162)=6.5$, $p<0.01$, $\eta^2=0.11$.

GAD-7: Robot 13 (12–14), 12 (11–13), 11 (10–12), 10 (9–11); control 13 (12–14), 13 (12–14), 12 (11–13), 11 (10–12). ANOVA $F(3,162)=5.2$, $p<0.01$, $\eta^2=0.09$.

PHQ-9: Robot 11 (10–12), 10 (9–11), 9 (8–10), 8 (7–9); control 11 (10–12), 11 (10–12), 10 (9–11), 9 (8–10). ANOVA $F(3,162)=4.6$, $p<0.01$, $\eta^2=0.08$.

MoCA: Robot 25 (24–26), 26 (25–26), 26 (25–27), 27 (26–28); control 25 (24–26), 25 (24–26), 25 (24–26), 26 (25–27). ANOVA $F(3,162)=3.1$, $p=0.03$, $\eta^2=0.05$.

Conclusion: PABLO®-assisted rehabilitation led to modest but statistically significant improvements in PTSD, anxiety, depression, and cognitive function compared to standard therapy. Effect sizes (η^2 0.05–0.11) indicate small-to-moderate clinical relevance. Robotic therapy is feasible and may provide additional psychosocial benefit in Ukrainian veterans with upper limb amputation.

We gratefully acknowledge the support of the National Research Foundation of Ukraine (NRFU) for our project No. 2025.07/0021, “Enhancing the Effectiveness of Limb Function Recovery in Military Personnel with Amputations and Traumatic Injuries.”

Keywords: amputation; robotic rehabilitation; PTSD

PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY MASQUERADING AS OPERCULAR SYNDROME: A RARE CLINICAL PRESENTATION

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Abstract ID: 80

Aim/ Background: Progressive Multifocal Leukoencephalopathy Masquerading as Opercular Syndrome: A Rare Clinical Presentation

Materials & Methods: A 61-year-old man with progressive bulbar dysfunction underwent detailed neurological examination, serial MRI brain imaging, and laboratory investigations, including HIV serology, viral load, CD4 count, and CSF JC virus PCR. Clinical, radiological, and laboratory findings were reviewed following informed consent.

Results: The patient demonstrated clinical features of opercular syndrome with progressive dysarthria, dysphagia, inability to protrude the tongue, and preserved yawning. Repeat MRI revealed bilateral frontal perisylvian cortical U-fiber and bilateral cerebellar white matter involvement, suggestive of progressive multifocal leukoencephalopathy. HIV serology was positive, with a viral load of 31.2 million copies/mL and a CD4 count of 127 cells/ μ L, confirming underlying immunosuppression. JC virus PCR in CSF was sent for confirmation.

Conclusion: Opercular syndrome is most commonly associated with bilateral opercular infarction and is rarely caused by progressive multifocal leukoencephalopathy (PML). This case highlights PML as an important stroke mimic in patients with progressive bulbar dysfunction and atypical neuroimaging. Bilateral cortical U-fibre involvement with cerebellar white matter lesions, along with clinical progression despite the absence of diffusion restriction, prompted evaluation for underlying immunosuppression, leading to the diagnosis of previously undetected HIV infection. Early recognition of this uncommon presentation facilitated timely initiation of antiretroviral therapy and appropriate supportive management.

Keywords: opercular , PMLE, HIV, neuroinfection, Bulbar

WHEN LOW PRESSURE BECOMES HIGH PRESSURE -PARADOXICAL SECONDARY INTRACRANIAL HYPERTENSION IN SPONTANEOUS INTRACRANIAL HYPOTENSION

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Abstract ID: 81

Aim/ Background: WHEN LOW PRESSURE BECOMES HIGH PRESSURE -PARADOXICAL SECONDARY INTRACRANIAL HYPERTENSION IN SPONTANEOUS INTRACRANIAL HYPOTENSION

Materials & Methods: A 34-year-old previously healthy man with progressive headache with changing character, underwent detailed neurological examination, fundus evaluation, lumbar puncture, MRI brain with MR venography, contrast MR cisternography/myelography, and neurosurgical intervention. Clinical, radiological, and procedural findings were analyzed.

Results: The patient presented with a history of a fall followed one week later by low pressure headache that worsened on standing and improved on lying down for 1.5 months. Subsequently, the headache evolved into a severe non-postural headache with symptoms of raised Intracranial pressure mainly associated with recurrent vomiting, progressive blurring of vision, neck and shoulder heaviness, giddiness, gait instability, and recurrent falls. On admission, fundus examination revealed bilateral Grade IV papilledema. Lumbar puncture showed an elevated CSF opening pressure of 36 cm H₂O, with transient improvement in vision following anti-edema measures. Persistent headache prompted repeat MRI brain, as an MRI performed one month earlier had been reported as normal. Repeat imaging demonstrated bilateral frontoparietal subdural hematomas, drooping of the splenium of the corpus callosum, and mild cerebellar tonsillar descent. Contrast MR cisternography/myelography localized a CSF leak through the left L2–L3 neural foramen with an associated epidural collection. The patient underwent an epidural blood patch followed by bilateral burr-hole evacuation of the subdural hematomas, resulting in significant improvement in headache and visual symptoms.

Conclusion: Spontaneous intracranial hypotension typically presents with orthostatic headache; however, delayed diagnosis may result in bilateral subdural hematomas, brain sagging, and paradoxical secondary intracranial hypertension with papilledema. This case highlights the importance of recognizing the transition from low-pressure headache to raised intracranial pressure symptoms. Early repeat neuroimaging and MR cisternography enabled localization of the spinal CSF leak and guided definitive treatment with an epidural blood patch and surgical evacuation, leading to favorable clinical recovery.

Keywords: spontaneous intracranial hypotension, intracranial hypertens

CIRCULATING EBV DNA AS AN ANCILLARY BIOMARKER IN IMMUNOCOMPETENT PRIMARY CNS LYMPHOMA: EVIDENCE FROM A PROSPECTIVE CASE–CONTROL STUDY

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PGIMER, Chandigarh

Abstract ID: 158

Aim/ Background: Primary Central Nervous System Lymphoma (PCNSL) is an aggressive extranodal non-Hodgkin lymphoma posing significant diagnostic challenges due to its clinical and radiological overlap with other CNS disorders. Stereotactic biopsy, the diagnostic gold standard, is invasive and yields no results following corticosteroid use. While Epstein–Barr virus (EBV) is well-established in immunocompromised PCNSL, its diagnostic relevance in immunocompetent patients remains poorly defined. This study evaluated whole-blood EBV DNA as a minimally invasive ancillary biomarker in this population.

Materials & Methods: This prospective case-control study enrolled 40 consecutive immunocompetent, histologically confirmed PCNSL patients and 120 controls comprising three subgroups: CNS inflammatory disorders (n=40), other CNS malignancies (n=40), and healthy controls (n=40). Peripheral blood EBV DNA was quantified using validated quantitative PCR prior to corticosteroid or chemotherapy administration. Diagnostic performance was assessed via ROC curve analysis.

Results: EBV DNA was detected in 45% of PCNSL patients, significantly exceeding rates in CNS inflammatory (25%), other CNS malignancy (22.5%), and healthy (12.5%) controls. Against the combined control group, PCNSL demonstrated a 3.27-fold enrichment of EBV DNA positivity (OR 3.27; 95% CI: 1.50–7.11; p=0.003). Median viral load in PCNSL (20,940 copies/mL) was significantly higher than CNS inflammatory controls (424.5 copies/mL; p=0.007), though no significant difference was observed versus other CNS malignancies or EBV-positive healthy individuals. ROC analysis yielded moderate diagnostic performance (sensitivity 66.7%; specificity 62.5%).

Conclusion: Whole-blood EBV DNA is significantly enriched in immunocompetent PCNSL, likely reflecting underlying immunological dysregulation and impaired viral containment. Although insufficient as a standalone diagnostic tool, it holds promise as a complementary liquid biopsy biomarker, particularly when integrated with CSF cytokines, MYD88 mutation analysis, and tumor profiling.

Keywords: EBV, PCNSL, Viral load, biomarker

A RARE DISEASE WITH A RARER MANIFESTATION- THE NEUROBRUCCELLOSIS THERAPEUTIC CONUNDRUM

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Abstract ID: 108

Aim/ Background: Neurobrucellosis is a rare but severe complication of systemic brucellosis, accounting for 3–10% of cases. Clinical presentations range from meningitis to stroke, mimicking neuro-tuberculosis in endemic countries, leading to delayed and often misdiagnosis.

Materials & Methods: A 58-year-old man presented with intermittent fever, polyarthralgia, and headache for 3 weeks. His systemic and neurological examination were normal. A detailed evaluation for pyrexia of unknown origin was conducted.

Results: CSF analysis revealed lymphocytic pleocytosis, elevated protein, and decreased glucose, consistent with an infectious etiology. Blood cultures isolated *Brucella*, and serum agglutination was positive. MRI spine showed thoracic discitis. Antibiotic therapy with ceftriaxone, rifampicin, and doxycycline was started. Four weeks later, despite optimal medical management, he developed recurrent transient neurological deficits followed by persistent focal deficits of dysarthria and right hemiparesis. MRI brain with angiography revealed acute lentiform infarcts and stenosis of the left middle cerebral artery M1 segment. Dedicated vessel wall imaging showed concentric enhancement, indicating vasculitis. High-dose corticosteroids were initiated while continuing antibiotics. There was no further deterioration, and he stabilized over the following weeks. Steroids were tapered and continued for 6 months along with ceftriaxone. Doxycycline and rifampicin were administered for one year. Persistent neurological sequelae included minimal dysarthria without limb weakness at the end of one year.

Conclusion: Vasculitis in neurobrucellosis is very rare, with no consensus on treatment protocol. Starting steroids is a cliffhanger with the risk of worsening an active systemic infection and the presumed benefit of neurological improvement. Our experience highlights that steroids, when judiciously used in selective complications like CNS vasculitis, along with antibiotics, can improve the outcome.

Keywords: Neurobrucellosis, CNS vasculitis, ischemic stroke, steroids

PALATAL MYOCLONUS: A RARE CLINICAL ENTITY

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Abstract ID: 109

Aim/ Background: Hypertrophic olivary degeneration (HOD), a rare form of transsynaptic degeneration, is secondary to dentato-rubro-olivary pathway injuries in some cases. We describe a unique case of an HOD patient who presented with palatal myoclonus post trauma secondary to Guillian mollaret triangle injury.

Materials & Methods: 30 year old male presented with history of severe traumatic brain injury (Marshall's grade 2) in March 2022, following which he developed , difficulty in walking with slurring of speech in the subsequent months which persisted and was non progressive. Patient required two person support to stand and walk and on walking he was noted to sway to either side. Patient had clumsiness of upper limbs while eating and on attempting to reach for objects.

Results: Neurological examination demonstrated dysarthria, horizontal nystagmus, bilateral cerebellar ataxia, and 2–3 Hz rhythmic contractions of the soft palate and upper larynx suggestive of palatal myoclonus (video). I/v/o palatal myoclonus and ataxia- inferior olivary nucleus hyperthrophy was considered. Magnetic resonance imaging (MRI) of the brain done which showed bilateral inferior Olivary nucleus hypertrophy. Post traumatic IONH secondary to Guillian Mollaret triangle injury was considered . Other causes where palatal myoclonus was seen include SCA 20, Alexander's disease, progressive ataxia palatal tumour syndrome (PAPT).

Conclusion: This case report suggests that patient with a history of severe traumatic brain injury especially palatal myoclonus, should be alert to the possibility of delayed bilateral HOD.

Keywords: Hypertrophic olivary degeneration, palatal myoclonus

FROM HEADACHE TO DIAGNOSIS: SOLVING A CLINICAL MYSTERY

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Abstract ID: 73

Aim/ Background: Posterior reversible encephalopathy syndrome (PRES) is a neurological emergency with diverse etiologies. Although hypertension is a common precipitant, an underlying large-vessel vasculitis is an exceptionally rare cause. We report an atypical presentation with misleading clinical and radiological features that posed a significant diagnostic challenge and required multimodal imaging to establish the final diagnosis..

Materials & Methods: A 48-year-old woman presented with acute severe headache, bilateral painless visual blurring, abdominal pain, vomiting, and generalized tonic-clonic seizures. MRI brain demonstrated bilateral cortical-subcortical T2/FLAIR hyperintensities with cerebellar microhemorrhages, leptomeningeal enhancement, and thin subdural effusion, raising differentials including infectious meningitis, inflammatory disorders, and atypical PRES. Extensive cerebrospinal fluid, infectious, autoimmune, and inflammatory investigations were performed. Whole-body PET-CT and cerebral/renal digital subtraction angiography (DSA) were subsequently undertaken.

Results: Routine investigations excluded tuberculosis, autoimmune disorders, MOGAD, NMOSD, and IgG4-related disease. PET-CT demonstrated diffuse metabolically active large-vessel vasculitis, while DSA revealed bilateral subclavian, right renal, and superior mesenteric artery occlusions, confirming Type V Takayasu arteritis. Right renal artery occlusion resulted in renovascular hypertension, explaining the neurological syndrome. The patient improved clinically following treatment with corticosteroids, tocilizumab, antihypertensives, and antiepileptic therapy.

Conclusion: This case highlights the importance of considering occult systemic vasculitis in patients with atypical neuroimaging, accelerated hypertension, and inconclusive routine investigations. Advanced vascular imaging played a pivotal role in diagnosis. Early recognition and targeted immunosuppressive therapy are essential to prevent irreversible vascular and neurological complications.

Keywords: PRES; takayasu Arteritis; large vessel vasculitis; DSA;

WHEN RECURRENT STROKE IS NOT THE FINAL DIAGNOSIS: A DIAGNOSTIC CHALLENGE

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Abstract ID: 74

Aim/ Background: Recurrent ischemic strokes despite appropriate secondary prevention should prompt evaluation for uncommon etiologies, including inflammatory vasculopathies. CNS vasculitis often presents with nonspecific clinical and radiological findings, making diagnosis challenging. Early recognition and timely immunosuppressive therapy are essential to prevent irreversible neurological injury.

Materials & Methods: A 67-year-old man with a past history of multiple ischemic strokes presented with fever, severe headache, and difficulty performing daily activities. He was normotensive with no focal neurological deficits. MRI brain revealed new acute and evolving bilateral cerebral infarcts with diffuse gyral enhancement involving the left parieto-occipito-temporal region and multiple cortical and subcortical microhemorrhages. CSF analysis showed elevated protein with lymphocytic pleocytosis, while extensive infectious workup was negative. Digital subtraction angiography showed no evidence of vasculitis. Laboratory evaluation demonstrated elevated inflammatory markers, positive PR3/C-ANCA antibodies (quantitative-50 times ULN), and negative ANA. Based on the clinical, radiological, CSF, and serological findings, C-ANCA-associated CNS vasculitis was diagnosed. The patient received pulse intravenous methylprednisolone followed by rituximab.

Results: The patient showed marked clinical improvement following immunosuppressive therapy, with resolution of headache and stabilization of neurological status. No further cerebrovascular events occurred during hospitalization, and he was discharged in a stable condition on appropriate follow-up.

Conclusion: C-ANCA-associated CNS vasculitis should be considered in patients with recurrent ischemic strokes and atypical neuroimaging despite normal blood pressure. A multidisciplinary approach and early immunosuppressive therapy can facilitate diagnosis and significantly improve patient outcomes.

Keywords: Recurrent stroke; headache, c-anca, small vessel vasculitis

MULTIMODAL MACHINE LEARNING-BASED CLASSIFICATION OF MILD AND MAJOR NEUROCOGNITIVE DISORDER USING CLINICAL, EEG, AND NEUROIMAGING FEATURES

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Abstract ID: 71

Aim/ Background: Aim:

To characterize mild and major neurocognitive disorder using clinical, EEG, and neuroimaging features and to develop a machine learning framework for classification and prediction of progression.

Background:

Mild neurocognitive disorder and major neurocognitive disorder are clinically important stages within the spectrum of cognitive decline, and early differentiation between them is essential for diagnosis and management. Conventional clinical assessment alone may not adequately capture the heterogeneity of these conditions. Multimodal integration of clinical variables, electrophysiological findings, and neuroimaging features may improve diagnostic characterization and prediction of progression.

Materials & Methods: This prospective follow-up study included individuals with mild neurocognitive disorder, major neurocognitive disorder, and cognitively normal controls. Clinical evaluation, cognitive assessment, MRI brain, EEG, and optional FDG PET-CT were used to generate a multimodal dataset. Supervised machine learning models were applied for binary classification of major versus mild neurocognitive disorder and ternary classification including healthy controls.

Results: Cognitive scores showed a progressive decline from healthy controls to mild neurocognitive disorder and major neurocognitive disorder. MRI findings demonstrated generalized cortical atrophy predominantly in neurodegenerative groups and ischemic changes in vascular groups, while EEG generalized slowing was more frequent in major neurocognitive disorder and in individuals who later converted from mild to major neurocognitive disorder. Machine learning models showed strong classification performance, supporting the value of multimodal prediction

Conclusion: Multimodal assessment combining clinical, EEG, and neuroimaging data may improve objective classification of neurocognitive disorders and support early identification of individuals at risk of progression.

Keywords: Mild neurocognitive disorder; major neurocognitive disorder;

PROGRESSIVE NON COMPRESSIVE MYELOPATHY IN A YOUNG WOMEN- A DIAGNOSTIC CHALLENGE

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Abstract ID: 142

Aim/ Background: Filar arteriovenous fistula (AVF) is an exceptionally rare spinal vascular malformation resulting from a direct communication between the artery and vein of the filum terminale. Its clinical presentation is often insidious, with progressive congestive myelopathy and nonspecific MRI findings, leading to delayed diagnosis and irreversible neurological deficits. This report aims to highlight the importance of recognising spinal vascular malformations in patients presenting with progressive non-compressive myelopathy and to emphasise the role of spinal digital subtraction angiography (DSA) in diagnosis and treatment planning.

Materials & Methods: A 23-year-old woman presented with a two-year history of progressive spastic paraparesis, sensory impairment, and bowel and bladder dysfunction. MRI of the spine demonstrated longitudinal cord oedema extending from D6 to the conus medullaris with multiple serpiginous perimedullary flow voids, suggesting a spinal vascular malformation. Definitive diagnosis was established by spinal DSA, which identified a filar AVF at the L3 level. The patient underwent successful transarterial glue embolisation followed by comprehensive multidisciplinary neurorehabilitation.

Results: Post-embolisation angiography demonstrated complete obliteration of the fistula without procedural complications. Clinically, flexor spasms resolved within two days, and the patient regained the ability to walk with a walker within two weeks of rehabilitation.

Conclusion: This case illustrates that filar AVF, although rare and more commonly reported in males, should be considered in young patients with progressive non-compressive myelopathy. MRI findings of long-segment cord oedema with perimedullary flow voids should prompt spinal DSA, the diagnostic gold standard. Early recognition and definitive treatment are crucial to halt venous congestive myelopathy and improve neurological recovery, whereas delayed diagnosis may result in permanent disability. This case underscores the need for increased clinical awareness of this rare but treatable cause of progressive myelopathy.

Keywords: Filar arteriovenous fistula, artery and vein of the filum te

FROM INFARCTION TO INFECTION: DELAYED CEREBRAL ABSCESS AS A SEQUELA OF ISCHEMIC STROKE - CASE REPORT

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Abstract ID: 92

Aim/ Background: Intracranial abscess formation is an uncommon complication following ischemic stroke. Secondary infections may occur due to tissue necrosis, impaired local immunity, or invasive procedures. Delayed presentation can obscure diagnosis and increase morbidity.

Materials & Methods: We report a patient who developed a brain abscess as a delayed complication of an ischemic stroke. The patient initially presented with acute focal neurological deficits, and neuroimaging confirmed cerebral infarction. Conservative management with standard stroke care resulted in partial neurological recovery. Several weeks later, the patient developed fever, worsening headache, and neurological deterioration.

Results: Repeat imaging showed a ring-enhancing lesion with surrounding edema at the previous infarct site, consistent with a cerebral abscess. Broad-spectrum intravenous antibiotics were initiated, followed by neurosurgical drainage. Targeted antimicrobial therapy led to gradual clinical and radiological improvement during follow-up.

Conclusion: Brain abscess is a rare but life-threatening delayed complication of ischemic stroke, likely resulting from blood–brain barrier disruption that facilitates hematogenous spread of infection. Because patients may present with nonspecific symptoms such as fever, headache, altered mental status, or new neurological deficits, diagnosis can be delayed. Clinicians should maintain a high index of suspicion in post-stroke patients with unexplained clinical deterioration. Early contrast-enhanced neuroimaging is essential for diagnosis, while prompt antimicrobial therapy and neurosurgical intervention, when indicated, are critical for improving outcomes. Greater awareness of this uncommon complication may enable earlier recognition and reduce associated morbidity and mortality.

Keywords: Cerebral abscess, Post- stroke complication, Hematogenous

CULTURAL PERCEPTIONS AND CLINICAL DETERMINANTS OF TREATMENT CHOICE IN EPILEPSY: A CROSS-SECTIONAL OBSERVATIONAL STUDY

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Abstract ID: 102

Aim/ Background: Managing epilepsy is complex in few communities due to a stern and false cultural belief on non-allopathic/ traditional medication. We aim to study the factors that influence patients'/caretakers' decision to opt for non-allopathic medication for seizure control.

Materials & Methods: A cross- sectional observational study was conducted among adults with epilepsy who attended the neurology outpatient department between January 2024 to July 2025. Participants were interviewed telephonically using a structured questionnaire assessing demographic characteristics, educational background, clinical factors of epilepsy, and personal beliefs related to the condition. Descriptive and inferential statistical analyses were performed.

Results: 266 patients with epilepsy were enrolled in this study. The mean age of the participants was 27.34 ± 18.35 years, with an equal male female distribution. Around 21.4% of patients with epilepsy had used non-allopathic treatment either at onset of illness (38.6%) or after use of allopathic treatment (61.4%). 17.3% believed epilepsy to be associated with supernatural causes, majority stemming from familial belief (60.86%). On bivariate analysis, treatment choice was significantly associated with rural background, longer duration of epilepsy, presence of adverse effects from ASM, uncontrolled attacks, and cultural perception with superstitious beliefs (all $p < 0.05$).

Conclusion: Treatment choice in epilepsy was influenced by both clinical factors and cultural perceptions, highlighting the need of a holistic approach in management. Addressing beliefs, setting realistic expectations, and managing side effects proactively may help improve adherence to allopathic treatment and reduce the reliance on unproven alternative methods.

Keywords: Cultural beliefs, non-allopathic treatment, superstitions

DEVELOPMENT OF STRIDE: AN INTELLIGENT WEARABLE VIBROTACTILE CUEING SYSTEM FOR PREDICTION OF FREEZING OF GAIT AND MOBILITY ASSISTANCE IN PARKINSON'S DISEASE

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Abstract ID: 159

Aim/ Background: FREEZING OF GAIT (FoG) is a disabling motor symptom affecting up to 80% of individuals with advanced PARKINSON'S DISEASE (PD), frequently leading to falls, reduced mobility, loss of independence, and impaired quality of life. Existing treatment approaches primarily rely on pharmacological management and intermittent clinical assessment, which often fail to address real-world mobility challenges. This study presents STRIDE, an intelligent wearable system designed to predict FoG episodes and deliver real-time vibrotactile cueing to support gait continuation.

Materials & Methods: STRIDE integrates an INERTIAL MEASUREMENT UNIT (IMU), embedded processing, wireless communication, and AI/ML-based gait analysis algorithms within a lightweight ankle-worn platform. Continuous gait data are acquired and processed to identify movement patterns associated with impending FoG episodes. Upon prediction of a FoG event, the system provides real-time vibrotactile cues to facilitate movement initiation and gait continuation. The device was developed for long-term ambulatory use in both clinical and home-based environments.

Results: The developed prototype demonstrated reliable acquisition of gait signals, real-time processing capability, and successful delivery of synchronized vibrotactile cues during ambulatory testing. Preliminary system validation confirmed the feasibility of continuous mobility monitoring and closed-loop cueing within a compact wearable architecture suitable for real-world deployment.

Conclusion: STRIDE represents a promising DIGITAL NEURO-REHABILITATION platform that combines continuous gait monitoring, FoG prediction, and real-time intervention in a single wearable system. The proposed approach has the potential to improve mobility, reduce fall risk, and support independent living in individuals with Parkinson's Disease while enabling scalable deployment across clinical and community settings.

Keywords: Parkinson, Cueing, Freezing of Gait, Falls

A DUAL QSAR FRAMEWORK FOR BACE1/BACE2 SELECTIVITY SCREENING IN ALZHEIMER'S DRUG DISCOVERY.

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Abstract ID: 106

Aim/ Background: This study developed a dual-target quantitative structure-activity relationship (QSAR) framework to identify potent BACE1 inhibitors that selectively spare BACE2.

Materials & Methods: Bioactivity data for BACE1 and BACE2 were collected from the ChEMBL database. BACE1 bioactivity data were obtained from ChEMBL target ChEMBL4822, and BACE2 bioactivity data were obtained from the confirmed human, single-protein ChEMBL target ChEMBL2525. For both targets, only records reporting IC₅₀ as the standard activity type, an exact relation (standard_relation = "="),

Results: Screening a library of 8,301 candidate compounds identified 3,601 (43.4%) potent BACE1 inhibitors with a pIC₅₀ greater than 7. Applying the selectivity filter (a delta pIC₅₀ of 2 or greater) successfully isolated 142 selective compounds, representing 1.7% of the total library, which are predicted to selectively inhibit BACE1 while sparing protective BACE2 activity. The top-ranked hit by selectivity exhibited a delta pIC₅₀ of 3.67, with a predicted BACE1 pIC₅₀ of 8.75 and a BACE2 pIC₅₀ of 5.08. The candidate with the highest absolute BACE1 potency reached a predicted pIC₅₀ of 11.10, maintaining a delta pIC₅₀ of 3.45. Notably, top-ranked compounds exceeding a pIC₅₀ of 10 surpass the maximum training data bioactivity ceiling (where the BACE2 maximum was 9.72) and are explicitly flagged as computational extrapolations falling outside the model's formal applicability domain

Conclusion: This dual-model framework successfully addresses the single-target gaps in existing pipelines by isolating compounds that selectively inhibit BACE1 while sparing BACE2. The BACE1 model showed stable cross-validation metrics ($R^2 = 0.69 \pm 0.01$), whereas the smaller BACE2 dataset ($n = 1,214$) led to higher variability ($R^2 = 0.59 \pm 0.07$), reflecting how understudied BACE2 remains in literature. The 1.7% hit yield is a modest, realistic selection. Preserving BACE2 activity is biologically vital, as literature confirms it actively degrades nascent amyloid-beta; co-inhibiting it could inadvertently dismantle a crucial natural protective mechanism.

Keywords: β -Secretase; QSAR model; β -amyloid precursor pr

THE ENIGMA OF AUTOIMMUNE ENCEPHALITIS: A CASE WITH HIDDEN SECONDARY PATHOGEN

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Abstract ID: 404

Aim/ Background: NMDA receptor encephalitis is a rare autoimmune disorder that presents with a combination of psychiatric, neurological, and autonomic symptoms, making early diagnosis challenging. Advances in understanding its pathophysiology have highlighted the critical role of tumor-mediated autoimmunity in disease development.

Materials & Methods: A 22 year-old female with recurrent episodes of altered behaviour and recently diagnosed as NMDAR encephalitis (CSF NMDAR Ab+) was referred in view of minimal response after being managed with pulse IV methylprednisolone and two doses of rituximab. Her CECT chest abdomen and USG pelvis done earlier were unremarkable.

Results: A detailed evaluation to look for no response to immunotherapy was done and MRI pelvis showed a well-defined cystic lesion with a thin peripheral layer of fat contents in left ovary likely mature cystic teratoma despite a normal CECT abdomen pelvis. Therefore, she underwent laparoscopic left salpingo-oophorectomy and was also kept on maintenance IV MPS, IV Ig and monthly cyclophosphamide and she responded very well in follow up.

Conclusion: Anti-NMDA receptor encephalitis is notably associated with ovarian teratomas, particularly in young women. The presence of NMDA receptor-expressing neural tissue within teratomas may trigger an autoimmune response, leading to encephalitis. Aggressive management including early detection and surgical removal of the teratoma, combined with immunotherapy, are crucial for favorable outcomes. Delayed diagnosis can result in significant morbidity and mortality. Therefore, aggressive management along with understanding of poor response are essential to improve patient prognosis.

Conclusion: NMDA receptor encephalitis with ovarian teratoma is a treatable but potentially life-threatening condition requiring early diagnosis and prompt intervention. Combined immunotherapy and surgical removal of the teratoma significantly improve outcomes.

Keywords: NMDAR Encephalitis, Teratoma

CLINICAL PROFILE, AUTONOMIC FEATURES AND QUALITY OF LIFE IN PATIENTS WITH POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME: A CASE SERIES

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Abstract ID: 417

Aim/ Background: Postural Orthostatic Tachycardia Syndrome (POTS) is an under-recognised disorder of autonomic dysfunction that predominantly affects young individuals and often results in delayed diagnosis. We aimed to describe the clinical profile, autonomic characteristics and quality-of-life impact in a case series of patients with POTS.

Materials & Methods: We retrospectively analysed 10 consecutive patients fulfilling diagnostic criteria for POTS based on clinical evaluation and head-up tilt testing. Demographic characteristics, presenting symptoms, orthostatic vital signs, laboratory investigations, autonomic evaluation, treatment and quality-of-life measures were reviewed

Results: Ten patients (9 females, 1 male; mean age 27.6 ± 11.8 years) were included. The median symptom duration before diagnosis was 2 years (range 1 month–17 years). Palpitations, light-headedness, presyncope/syncope, and fatigue were universal, while chest pain and dyspnea occurred in 60%, headache/neck pain and tremulousness in 50%, and sleep disturbance in 60%. Four patients (40%) had preceding COVID-19 infection. Connective tissue features were present in 30%, and Raynaud's phenomenon / photosensitive rash in 10%. All patients demonstrated a heart rate increase ≥ 30 bpm on standing/HUTT without orthostatic hypotension, with positive HUTT in every case. Cardiac evaluation was normal in all patients. Autoimmune testing and nerve conduction studies were largely unremarkable. Mean Malmö POTS Symptom Score was 72 ± 9 , with significant SF-36 impairment. Symptomatic improvement occurred in 80% following lifestyle modification and beta-blocker therapy.

Conclusion: POTS predominantly affects young females and is associated with considerable functional disability and delayed diagnosis. Careful assessment of orthostatic symptoms with bedside orthostatic vital signs and head-up tilt testing facilitates early diagnosis. Greater awareness among neurologists may reduce misdiagnosis and improve patient outcomes.

Keywords: Postural Orthostatic Tachycardia Syndrome; POTS; Dysautonomia; Head-up Tilt Test; Orthostatic Intolerance

CYTOKINE SIGNATURE OF IDIOPATHIC INTRACRANIAL HYPERTENSION: INSIGHTS INTO IMMUNE-MEDIATED PATHOPHYSIOLOGY

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Abstract ID: 173

Aim/ Background: Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure without an identifiable intracranial lesion, and its pathophysiology remains poorly defined. Emerging evidence suggests immune-mediated mechanisms may contribute to disease onset and progression. This study aimed to characterize serum and CSF cytokine profiles in IIH patients and correlate them with disease severity and outcomes.

Materials & Methods: A prospective case-control study was conducted including 100 adult patients with IIH and 100 age- and sex-matched controls at a tertiary care center in India (IEC-03/2019-1141; CTRI/2020/03/023920). Serum and CSF samples were analyzed for IL-2, IL-4, IL-6, IL-10, IL-17A, TNF- α , and IFN- γ using BD™ Cytometric Bead Array technology. Cytokine profiles were evaluated against disease severity, treatment response, and clinical outcomes.

Results: Distinct inflammatory alterations were observed in both serum and CSF of patients with IIH compared to controls, suggesting a disease-associated immune signature. Variations in specific pro- and anti-inflammatory cytokine levels correlated with disease severity and treatment response.

Conclusion: These findings support a potential role for immune dysregulation in IIH pathogenesis. Cytokine profiling in serum and CSF may serve as a promising, minimally invasive approach for identifying biomarkers of disease activity and prognosis, warranting further validation in larger cohorts.

Keywords: Idiopathic intracranial hypertension; Cytokines; Cerebrospinal

EVALUATING REHABILITATION PROGRESS IN AUTISM SPECTRUM DISORDER: A LONG-TERM CASE STUDY

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Abstract ID: 44

Aim/ Background: This paper examines the effectiveness of rehabilitation therapies in improving the condition of a child with autism spectrum disorder. It presents the case of a nine-year-old boy who was diagnosed with autism at the age of five. The study highlights the importance of early, multidisciplinary intervention in addressing the emotional, motor, speech, and social difficulties commonly associated with autism.

Materials & Methods: The paper is based on an observed case study of a nine-year-old boy over a period of two years. A multidisciplinary rehabilitation approach was used, which included speech therapy, occupational therapy, social skill training, group therapy, play therapy, applied behaviour analysis, and cognitive behavioural analysis. These interventions were applied to support development across multiple areas of functioning.

Results: The findings suggest that the therapeutic interventions were effective in improving several developmental domains over the two-year period. Notable progress was observed in emotional regulation, motor functioning, speech and language development, and social interaction. The case supports the view that early and consistent multidisciplinary rehabilitation can produce meaningful improvements in children with autism spectrum disorder.

Conclusion: The study concludes that early, sustained, conventional rehabilitation is effective in improving emotional and selected social difficulties in a young child with ASD over a relatively short developmental period (about two years).

It supports the view that autism is not static and that with appropriate, multidisciplinary intervention, meaningful functional gains are possible in real-world clinical settings, in line with larger long-term outcome studies.

Keywords: speech therapy, occupational therapy, social skill training,

EXPLORING THE SOLUBLE IMMUNE CHECKPOINT AXIS FOR THE DIAGNOSIS OF AUTOIMMUNE ENCEPHALITIS

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PGIMER, Chandigarh

Abstract ID: 146

Aim/ Background: Autoimmune encephalitis (AE) is an inflammatory condition of the brain parenchyma caused by a misdirected immune response, implicating as autoantibodies against neuronal receptors resulting in their damage. The PD-1/PD-L1 signaling pathway, including its soluble counterparts (sPD-1/sPD-L1) are closely associated with immune tolerance in various autoimmune conditions, but its role has not been investigated in AE.

Materials & Methods: Prospectively, 25 patients of possible AE and 25 age/sex matched control were recruited. Clinical and laboratory parameters were noted. sPD-1 and sPD-L1 were measured in serum and CSF of enrolled cases through ELISA

Results: 60 patients with clinically suspected AEs were screened, 25 were labelled as possible AEs (15 seropositive and 10 seronegative). Serum levels of sPD-1 [302.0 pg/ml (IQR: 206.2) vs 176.50 pg/ml (IQR: 102.44); $p=.006$] and sPD-L1 [63.80 pg/ml (IQR: 71.20) vs 29.00 pg/ml (IQR: 12.50); $p=.002$] were significantly elevated in AE patients compared to control while CSF levels didn't show significant difference. Moreover, serum levels of sPD-1 [302.0 pg/ml (IQR: 206.2) vs 10.72 (IQR: 33.23); $p<0.0001$] and sPD-L1 [63.80 pg/ml (IQR: 71.20) vs 15.50 pg/ml (26.21); $p=0.0001$] were significantly raised compared to CSF. The antibody-positive and antibody-negative AE patients didn't show significant differences in serum PD-1/PD-L1 level

Conclusion: Patients with AE exhibited an elevated sPD-1 and sPD-L1 in the serum, suggesting a potential involvement of immune checkpoint molecules in the pathogenesis of AE. Similarly, elevated levels of sPD-1/sPD-L1 were also seen in seronegative AE patient cohort. This data suggests that immune checkpoint molecules may serve as a potential diagnostic biomarker, but further validation on larger cohort is needed.

Keywords: Autoimmune encephalitis, PD-1, PD-L1, checkpoint molecules

THE SIGNIFICANCE OF WHOLE CORD INVOLVEMENT: ETIOLOGY, SEROLOGICAL CORRELATION, AND DIAGNOSTIC OVERLAP IN LONGITUDINALLY EXTENSIVE TRANSVERSE MYELITIS.

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Abstract ID: 181

Aim/ Background: To determine the etiological spectrum of LETM and evaluate the diagnostic value of true whole cord involvement (C1-conus) relative to patient antibody status.

Materials & Methods: This hospital-based observational cohort study enrolled 71 adult patients with MRI-confirmed LETM at a tertiary care center in Western Rajasthan, India. Statistical association were evaluated using the Chi-square test.

Results: A significant correlation was found between antibody status and the spinal cord involvement pattern (Chi Square= 7.083, $p = 0.029$). Notably, true whole cord involvement was observed exclusively in MOG-IgG positive (27.3%) and seronegative (7.9%) patients.

Conclusion: True whole-cord involvement is a rare but distinctive imaging pattern in LETM. MRI lesion extent alone is insufficient for etiological classification, emphasizing the need for integrated assessment using neuroimaging, serology, and evaluation for infectious causes.

Keywords: LETM-longitudinal extensive transverse myelitis

CEREBRAL VENOUS THROMBOSIS COMPLICATED BY HSV-1 ENCEPHALITIS AND REBOUND SPONTANEOUS INTRACRANIAL HYPOTENSION WITH SUBDURAL HEMORRHAGE : A FATAL DIAGNOSTIC CHALLENGE

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Abstract ID: 140

Aim/ Background: To report a rare and fatal case of cerebral venous thrombosis (CVT) complicated by concurrent HSV-1 encephalitis, followed by rebound spontaneous intracranial hypotension (SIH) with subdural hemorrhage, highlighting the diagnostic overlap between high- and low-pressure intracranial disorders and the challenges in their sequential management.

Materials & Methods: A retrospective single-case analysis based on clinical records, neuroimaging (MRI brain with MR venogram, repeat MRI/CT brain, CT myelogram), and cerebrospinal fluid (CSF) analysis of a patient managed in the Department of Neurology between June and August 2025.

Results: A 45-year-old chronic alcoholic and tobacco user presented with an 11-day history of headache, fever, and blurring of vision, with bilateral papilledema, left lateral rectus palsy, and neck rigidity. MRI/MR venogram showed right sigmoid, transverse sinus, and internal jugular vein thrombosis with pachymeningeal enhancement; CSF PCR was positive for HSV-1. He improved with anticoagulation, acyclovir, and acetazolamide. Five weeks later, he developed postural headache; repeat imaging showed bilateral subdural hemorrhage with sulcal effacement, and CT myelogram confirmed a CSF leak at C5 and C7-D1, consistent with SIH. Despite epidural blood patching, burr-hole evacuation, and decompressive surgery for worsening edema, he had intraoperative and subsequent ICU cardiac arrests and died

Conclusion: Rebound SIH with subdural hemorrhage is a rare but potentially fatal complication following anticoagulation for CV & DISCUSSION - This case underscores the need for vigilant postural-headache screening and early neuroimaging after CVT anticoagulation, and for prompt recognition of SIH-associated subdural hemorrhage before it progresses to refractory cerebral edema and herniation.

Keywords: Cerebral venous thrombosis; HSV-1 encephalitis; Spontaneous

THE RIND THAT UNMASKED THE MASQUERADE: INFECTION TO MALIGNANCY

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Abstract ID: 176

Aim/ Background:

To highlight a rare diagnostic challenge where meningoencephalitis clinically and radiologically mimics carcinomatous (leptomeningeal) meningitis and to compare the clinical, radiological and CSF findings between them.

Materials & Methods: Single patient case report and retrospective review of clinical records.

Results: A 36 year old male, known case of diabetes mellitus, psychiatric illness and seizure disorder since 2016, on regular medication, presented with headache and altered sensorium for 3 days, along with 2–3 episodes of GTCS and vomiting.

On examination, GCS was E2V2M3. Neck stiffness was present. Left eye ptosis with a dilated, non-reacting pupil was noted.

****CSF analysis:**** 30 cells, monocytes 40%, lymphocytes suggestive of a viral/inflammatory aetiology. Autoimmune panel was negative. BioFire was negative. Paraneoplastic panel was negative. Malignant cells were absent.

MRI brain :T2/FLAIR hyperintensities around the brainstem with subtle leptomeningeal enhancement – BLOOMING RIND SIGN

On further evaluation, USG abdomen revealed enlarged lymph nodes. Hence, CECT chest and abdomen was performed, which showed diffuse stomach wall thickening with local lymph nodes.

****Endoscopy:**** Malignant gastric ulcer was identified and sent for histopathological examination, which confirmed ****signet ring cell carcinoma**

Conclusion: This case illustrates a presentation of meningoencephalitis masquerading as an underlying malignant aetiology, helping in early diagnosis and management. Despite advances in neuroimaging and targeted therapies, prognosis remains poor, with limited survival even with advanced treatment.

Keywords: carcinomatous meningitis

PRION DISEASE PRESENTING AS SUPERREFRACTORY STATUS EPILEPTICUS

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Abstract ID: 105

Aim/ Background: Prion diseases are a group of uniformly fatal neurodegenerative diseases caused by the transformation of an endogenous protein, PrP (prion-related protein), into a misfolded protein called the prion. Incidence of human prion diseases is about 1–1.5 per million per year. The disease typically affects middle-aged to elderly individuals with a median survival of 4–6 months.

2017 European Consortium criteria include rapidly progressive cognitive impairment with two of the following: myoclonus, visual or cerebellar disturbance, pyramidal or extrapyramidal signs, akinetic mutism. This presents a challenge for physicians in diagnosing this rare condition.

CJD presenting as super refractory Status epilepticus ,with a very short history is a very uncommon presentation and can mimic autoimmune encephalitis .

Materials & Methods: not applicable

Results: not applicable

Conclusion: We present a case of a 70-year-old lady with one month history of progressive neurological symptoms including vertigo, rapid cognitive decline ,hemiparesis,and seizures. She developed super refractory status epilepticus within 3 weeks of her symptom onset.Initial brain MRI showed small acute and subacute multifocal gyral DWI hyperintensities mimicking infarcts, but repeat MRI 2 weeks later revealed characteristic hyperintensities in the frontal and paracentral cortices, caudate, putamen, thalami.EEG showed showed periodic sharp wave complexes occurring one per second supporting the clinical suspicion of CJD. In view of short history, refractory SE, and MRI findings initially treated as seronegative autoimmune encephalitis.Routine CSF analysis done twice was normal ,negative for infections and antibodies for autoimmune encephalitis. Finally CSF 14-3-3 was positive confirming the diagnosis of Sporadic CJD

Keywords: Superrefractory status epilepticus, rapid cognitive decline,

NOT EVERY MENINGITIS IS TUBERCULAR

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Abstract ID: 49

Aim/ Background: Chronic meningitis in India is frequently presumed to be tubercular, particularly in the presence of lymphocytic cerebrospinal fluid pleocytosis, low CSF glucose, arachnoiditis, and leptomeningeal enhancement. This case highlights primary CNS B-cell lymphoma with lymphomatous meningitis masquerading as tubercular meningitis, resulting in diagnostic delay and therapeutic misdirection.

Materials & Methods: This is a single-patient case report of a 37-year-old right-handed male with subacute meningoencephalitic presentation, progressive cranial neuropathy, paraparesis, and diffuse craniospinal leptomeningeal disease. Serial clinical examinations, repeated CSF analyses, MRI brain and whole spine, PET imaging, CSF cytology, flow cytometry, and bone marrow evaluation were reviewed.

Results: The patient initially presented with vomiting, altered sensorium, confusion, and progressive bilateral lower limb weakness. CSF showed marked inflammatory meningitis with very high protein, pleocytosis, and subsequent hypoglycorrhachia; MRI demonstrated progressive pachy- and leptomeningeal enhancement with basal cisternal exudates and diffuse spinal arachnoiditis. He was treated empirically with broad antimicrobials, antitubercular therapy, steroids, and later antifungals because tubercular, fungal, and neoplastic meningitis were all considered. Definitive diagnosis was established only after repeat CSF cytology and flow cytometry demonstrated monoclonal CD19/CD20-positive lambda-restricted B cells, consistent with primary CNS B-cell lymphoma with lymphomatous meningitis and diffuse leptomeningeal dissemination.

Conclusion: Not every chronic meningitis with arachnoiditis and low CSF glucose is tubercular. In atypical or treatment-refractory cases, early CSF cytology, flow cytometry, and directed malignancy workup are crucial to avoid delay in diagnosing lymphomatous meningitis.

Keywords: Chronic meningitis, arachnoiditis, primary CNS B-cell lymphoma

UNMASKING NODO-PARANODOPATHY IN TREATMENT-RESISTANT INFLAMMATORY NEUROPATHIES: A SIX-PATIENT CASE SERIES FROM A TERTIARY CARE NEUROLOGY CENTRE IN RAJASTHAN, INDIA

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Abstract ID: 50

Aim/ Background: Nodo-paranodopathies are immune-mediated neuropathies caused by antibodies targeting nodal and paranodal proteins of myelinated peripheral nerve fibres, characteristically refractory to conventional immunotherapy including intravenous immunoglobulin (IVIg) and corticosteroids. We report six patients with treatment-resistant inflammatory neuropathy in whom targeted antibody evaluation unmasked the underlying aetiology and guided definitive therapy.

Materials & Methods: We retrospectively reviewed six patients with treatment-resistant inflammatory neuropathy (Guillain–Barré syndrome or chronic inflammatory demyelinating polyradiculoneuropathy phenotype) evaluated at a tertiary care neurology centre in Rajasthan, India. Demographics, clinical features, nerve conduction studies (NCS), CSF analysis, gadolinium-enhanced MRI of the lumbosacral plexus, autoimmune neuropathy antibody panel, treatment, and functional outcome (MRC sum score at 3 and 6 months) were recorded.

Results: The cohort comprised three males and three females, median age 40 years (range 36–50). All six patients had previously received at least one first-line immunotherapy without adequate response. Antibody testing identified anti-NF140 in one patient, anti-NF155 alone in one, combined anti-NF140+Nf186 (pan-neurofascin) in three, and dual anti-NF155 plus anti-GM1 co-positivity in one. CSF protein ranged from 55 to 503 mg/dL; all five anti-neurofascin-positive patients showed albumin-cytological dissociation. MRI lumbosacral plexus showed root or plexus enhancement in three patients. Pan-neurofascin-positive patients had the most severe presentations, including cranial nerve palsies, bulbar dysfunction, autonomic involvement, and, in one case, complete electrical inexcitability. All six patients received rituximab (1g × 2 doses, 15 days apart) as definitive therapy. Mean MRC sum score improved from 27.3 at 3 months to 41.3 at 6 months. One pan-neurofascin-positive patient (Case 5) with the longest interval from onset to rituximab showed the worst outcome (6-month MRC: 14/60).

Conclusion: Nodo-paranodopathy in the Indian clinical setting presents with phenotypic heterogeneity mimicking GBS and CIDP. Early antibody testing and prompt rituximab initiation — before irreversible axonal injury accumulates — are critical. Prolonged diagnostic delay correlates with poor functional recovery. Dual anti-NF155 and anti-GM1 co-positivity may contribute to a mixed demyelinating-axonal electrophysiological pattern.

Keywords: Neurofascin antibody; nodo-paranodopathy; rituximab; treatme

TOXOPLASMOSIS-INDUCED YOUNG-ONSET PARKINSONISM MIMICKING BASAL GANGLIA ENCEPHALITIS AND NBIA: A DIAGNOSTIC CHALLENGE

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Abstract ID: 51

Aim/ Background: Young-onset parkinsonism presents with diverse etiologies including autoimmune, infectious, and neurodegenerative causes. Toxoplasmosis involving the basal ganglia is an uncommon but potentially reversible cause of secondary parkinsonism. We report a case of a young male with progressive parkinsonism and dystonia, initially suspected to have autoimmune or neurodegenerative etiology, later diagnosed as toxoplasmosis-induced basal ganglia dysfunction.

Materials & Methods: A 33-year-old right-handed male presented with insidious, gradually progressive tremulousness of both lower limbs, followed by upper limb tremor, rigidity, bradykinesia, hypophonia, and eyelid-opening apraxia. Symptoms were associated with truncal dystonia, emotional incontinence, and cognitive slowing. There was no sensory, bladder, bowel, cerebellar, or autonomic involvement. Past history included nephrotic syndrome, pulmonary tuberculosis, and opioid addiction. Examination revealed resting and postural tremor, bradykinesia, rigidity, truncal dystonia, extensor plantar, pseudobulbar affect, and chorioretinal scarring. MRI brain showed bilateral putaminal hyperintensities. ANA, ENA, autoimmune encephalitis, and NBIA genetic panel were negative. Serum IgG for *Toxoplasma gondii* was significantly elevated, confirming post-infectious etiology.

Results: The patient was treated with cotrimoxazole (sulfamethoxazole-trimethoprim) and supportive therapy. He demonstrated marked improvement in rigidity, tremor, and daily functioning within weeks of treatment initiation.

Conclusion: Toxoplasmosis involving the basal ganglia can present with secondary parkinsonism, mimicking autoimmune or neurodegenerative disorders such as NBIA. Recognition of this treatable etiology is essential, as timely antimicrobial therapy may result in significant recovery.

Keywords: Toxoplasmosis, young onset parkinson, NBIA, Basal ganglia en

HEART IN THE BRAIN: BILATERAL MEDIAL MEDULLARY INFARCTION PRESENTING WITH AN ELSBERG PATTERN OF WEAKNESS

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Abstract ID: 121

Aim/ Background: Bilateral medial medullary infarction (BMMI) is an exceptionally rare subtype of posterior circulation stroke, accounting for a very small proportion of ischemic strokes. It is characterized by the characteristic “heart appearance” sign on diffusion-weighted MRI and often presents with rapidly progressive quadriparesis, bulbar weakness, and respiratory compromise. The clinical presentation may mimic cervical cord disorders, leading to delayed diagnosis. We report a rare case of BMMI presenting with an Elsberg pattern of weakness and highlight the importance of early clinicoradiological recognition.

Materials & Methods: A 60 year-old hypertensive male presented with sudden-onset leftt upper limb weakness followed by left lower limb weakness and slurred speech within the first 24 hours. Over the next 48 hours, he developed progressive weakness involving the right lower and upper limbs along with bulbar palsy, resulting in rapidly evolving quadriparesis. Neurological examination revealed dysarthria, bulbar weakness, Medical Research Council grade 3/5 power in all four limbs, and bilateral extensor plantar responses. MRI brain with diffusion-weighted imaging demonstrated symmetrical diffusion restriction in the bilateral medial medulla, producing the classic “heart appearance” sign consistent with acute bilateral medial medullary infarction. A review of the published literature was also performed.

Results: The patient’s clinical presentation closely resembled upper cervical cord pathology because of the progressive ascending pattern of weakness. MRI findings established the diagnosis of bilateral medial medullary infarction. Literature review identified only 38 reported cases, with a mean age of 62.2 years and a male predominance (74.2%). Motor weakness was the most frequent presenting feature, vertebral artery atherosclerosis was the commonest etiology, and outcomes were generally poor, with mortality approaching 24% and significant long-term dependency among survivors.

Conclusion: Bilateral medial medullary infarction is a rare but devastating stroke syndrome that may initially mimic cervical spinal cord disease because of its atypical presentation. Recognition of the characteristic “heart sign” on diffusion-weighted MRI, together with careful neurological evaluation, is essential for early diagnosis. Since initial imaging may occasionally be normal, maintaining a high index of suspicion is crucial to facilitate timely diagnosis and appropriate management, potentially improving clinical outcomes.

Keywords: Heart sign; Brainstem stroke; Elsberg pattern;

CISTERNAL OCULOMOTOR NERVE SCHWANNOMA: A RARE CAUSE OF VERTICAL DIPLOPIA

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Abstract ID: 414

Aim/ Background: Oculomotor nerve schwannoma (ONS) is an exceptionally rare benign cranial nerve tumor, accounting for only a very small fraction of intracranial schwannomas. Fewer than 100 cases have been reported, and cisternal lesions are particularly uncommon. We report a rare small cisternal ONS presenting with chronic progressive diplopia and ocular misalignment.

Materials & Methods: A 40-year-old man presented with 2.5 years of headache, binocular vertical diplopia and progressive abnormal upward deviation of the right eye. Neurological and ocular-motor examination was performed along with contrast-enhanced MRI brain, CT angiography, cerebrospinal fluid analysis and evaluation for neuromuscular junction disorders.

Results: Examination revealed right hypertropia with restriction of elevation of the left eye in primary position, abduction and adduction. Pupils were equal and reactive, with no ptosis. Ice-pack test, repetitive nerve stimulation and neostigmine testing were normal. CSF was non-inflammatory. Contrast-enhanced MRI demonstrated a 5.7 × 5.4 mm enhancing lesion along the cisternal segment of the left oculomotor nerve, suggestive of schwannoma. CT angiography showed no aneurysm, vascular malformation or hemodynamically significant stenosis. The patient was referred for stereotactic radiosurgical evaluation.

Conclusion: ONS should be considered in the differential diagnosis of chronic progressive ocular-motor dysfunction, even when the lesion is very small and the clinical deficit is atypical. High-resolution contrast-enhanced MRI is crucial for identifying these lesions and excluding vascular mimics. Given the substantial risk of postoperative third-nerve dysfunction reported with microsurgical resection, stereotactic radiosurgery represents an important treatment option for selected small lesions.

Keywords:

A RARE CAUSE OF MULTIAXIAL CNS INVOLVEMENT – SUPERFICIAL SIDEROSIS

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Abstract ID: 157

Aim/ Background: Superficial siderosis (SS) is a rare disease caused by deposition of iron-containing compounds in the subpial surface of the central nervous system (CNS). It has two subtypes: ‘classical’ infratentorial SS (iSS) and cortical SS (cSS). It is characterised by sensorineural deafness, cerebellar ataxia, dementia and signs of pyramidal tract dysfunction.

Materials & Methods: We are reporting a case of SS with diffuse CNS involvement.

Results: A 50-year-old gentleman had unsteadiness and frequent falls since eight months; dysarthria, headache, progressive hearing loss, and forgetfulness since one month with a past history of moderate head injury 12 years back. He had sensorineural hearing impairment, spastic dysarthria, asymmetric limb spasticity, exaggerated tendon reflexes, past pointing, and gait ataxia.

MRI brain revealed diffuse superficial curvilinear blooming on gradient echo sequence and MRI spinal cord showed diffuse meningeal based T2 hypointensity including conus medullaris suggestive of diffuse superficial siderosis. CT myelogram, however, did not reveal any source of focal bleeding. CSF examination was acellular with raised CSF protein (79 mg/dl) and CSF ferritin level (127 mcg/l).

He was treated with iron chelators and physical therapy. He had improvement in spasticity and ataxia after six months of follow up.

Conclusion: Classical SS is caused by a chronic low-volume leakage of blood into the subarachnoid space through a dural defect. Earlier it was diagnosed only at autopsy or intraoperatively but now neuroimaging and CSF analysis (CSF red blood cells, ferritin, bilirubin) are useful diagnostic tools. High index of suspicion and proper diagnostic evaluation is warranted in case of such multiaxial CNS diseases.

Keywords: Superficial siderosis, hemosiderin, subarachnoid hemorrhage,

REVERSIBLE PAN-CEREBELLAR ATAXIA DUE TO SUBCLINICAL HYPOTHYROIDISM: A RARE CASE REPORT

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Abstract ID: 76

Aim/ Background: Cerebellar ataxia is an uncommon neurological manifestation of hypothyroidism and is rarely reported in subclinical hypothyroidism

Materials & Methods: A 68-year-old man presented with progressive gait unsteadiness and limb incoordination for one year. Clinical evaluation, neuroimaging, cerebrospinal fluid analysis, autoimmune screening, and paraneoplastic antibody testing were performed.

Results: Examination revealed pan-cerebellar ataxia. MRI brain showed only chronic microvascular changes without cerebellar pathology. Thyroid testing demonstrated elevated TSH (10.62 mIU/mL) with normal T3 and T4 levels and markedly elevated anti-thyroid peroxidase antibodies (681 IU/mL). Levothyroxine therapy resulted in complete clinical recovery.

Conclusion: Severe cerebellar ataxia can occur despite mild thyroid dysfunction (3,5). Autoimmune thyroiditis may contribute to cerebellar dysfunction (6-8). Conclusion: Subclinical hypothyroidism should be considered in unexplained adult-onset cerebellar ataxia because prompt thyroid hormone replacement can lead to complete neurological recovery

Keywords: Anti TPO antibody, cerebellar ataxia, reversible ataxia

CASE REPORT OF CEREBRAL VENOUS THROMBOSIS INVOLVING THE POSTERIOR FOSSA LESION IN A RVD PATIENT

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Abstract ID: 6

Aim/ Background: Cerebral venous thrombosis (CVT) is an uncommon cause of stroke, with an incidence of approximately 3–4 per million annually. It most commonly involves the superior sagittal and transverse sinuses. Isolated posterior fossa parenchymal involvement is rare and is associated with poorer outcomes

Materials & Methods: A 54-year-old woman presented with headache and binocular diplopia, without fever, vomiting, giddiness, or loss of consciousness. Her medical history included rheumatic valvular heart disease, Graves' disease, disseminated tuberculosis, and hypertension. On examination, she had impaired upward gaze, gaze-evoked nystagmus, diplopia, and difficulty with tandem gait.

Laboratory investigations showed leukocytosis and dyslipidemia, with normal renal and metabolic parameters. MRI brain with MR venography demonstrated thrombosis of the superior vermician, precentral, and median mesencephalic veins, with a subacute hemorrhagic infarct involving the superior vermis and right superior colliculus. CSF analysis revealed mildly elevated protein and was negative for infectious and malignant causes.

Results: CVT has a highly variable clinical presentation and can mimic other neurological disorders. Multiple risk factors, including systemic inflammatory and hypercoagulable states, may coexist. Posterior fossa involvement, though rare, is associated with higher morbidity and mortality.

Conclusion: This case highlights the importance of MRI with MR venography in evaluating atypical headache presentations. Early recognition of CVT, particularly with posterior fossa involvement, is crucial as timely treatment can significantly improve outcomes.

Keywords: CVT POSTERIOR FOSSA RVD

A 40-SECOND SMARTPHONE TEST: AUTOMATED MACHINE LEARNING CLASSIFICATION OF PARKINSON'S DISEASE USING SMARTPHONE-CAPTURED FINGER TAPPING VIDEOS IN UNCONTROLLED ENVIRONMENTS

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Abstract ID: 62

Aim/ Background: To evaluate the accuracy of a machine learning model using smartphone-captured finger tapping videos to distinguish patients with Parkinson's disease (PD) from healthy controls (HC) in both clinical and home environments.

Clinical assessment of finger tapping, a key component of the MDS-UPDRS, is subjective. Existing video-based studies are often limited to controlled clinical settings, brief recording durations, and manual frame selection, reducing real-world applicability. An automated, accessible tool for objective assessment is needed.

Materials & Methods: We recorded 40-second finger tapping videos via smartphone from 30 PD patients and 30 HC (60 participants, 120 hands). Videos were preprocessed by removing the first/last 5 seconds, resizing frames, and automatically rejecting low-quality frames. MediaPipe extracted thumb tip and index fingertip landmarks. Thirteen temporal features (amplitude, velocity, frequency, halts/hesitations) were derived from the distance time-series. A Random Forest classifier was trained and tested on unseen data (10 PD, 7 HC videos). Feature correlation was analyzed.

Results: The Random Forest model achieved 88.23% accuracy, 83.33% precision, 90.0% recall, and an F1-score of 0.90 on the test set. Correlation analysis revealed strong coupling between velocity and amplitude features (e.g., F_{vel_var} – F_{amp_var} $r=0.89$) and high internal consistency among frequency metrics, while also highlighting feature redundancy.

Conclusion: Our automated pipeline, utilizing longer-duration smartphone videos from varied environments and robust preprocessing, accurately classifies PD. This approach supports accessible, objective telemonitoring. Future work will focus on severity prediction and extracting explainable kinematic biomarkers.

Keywords: PD, AI/ML, Video Analysis, Finger Tapping

ANTI-SRP-ASSOCIATED IMMUNE-MEDIATED NECROTIZING MYOPATHY IN A YOUNG FEMALE: A RARE CASE REPORT

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Hyderabad, Telangana

Abstract ID: 184

Aim/ Background: Anti-signal recognition particle (anti-SRP)-associated immune-mediated necrotizing myopathy (IMNM) is a rare subtype of idiopathic inflammatory myopathy characterized by rapidly progressive proximal muscle weakness, markedly elevated serum creatine kinase (CK) levels, and poor functional outcomes if not diagnosed and treated early.

Materials & Methods: A 20-year-old female presented with gradually progressive symmetrical proximal muscle weakness predominantly affecting the upper limbs, along with neck and trunk muscle weakness. Clinical examination revealed significant proximal muscle weakness and visible muscle atrophy of both upper limbs. Sensory, cerebellar, and cranial nerve examinations were unremarkable, suggesting a primary myopathic process.

Results: Laboratory investigations demonstrated markedly elevated serum creatine phosphokinase (CPK) of 5000 U/L and elevated liver enzymes (AST 155 U/L, ALT 146 U/L). Muscle MRI showed T2-weighted and STIR hyperintensities involving the shoulder and pelvic girdle muscles, consistent with active inflammatory myopathy. Myositis antibody profile revealed strongly positive anti-SRP antibodies (3+) with concurrent anti-Ro positivity, establishing the diagnosis of anti-SRP-associated immune-mediated necrotizing myopathy.

Conclusion: This case emphasizes the importance of considering anti-SRP-associated immune-mediated necrotizing myopathy in young patients presenting with proximal muscle weakness, muscle atrophy, and markedly elevated CPK levels. Early recognition supported by muscle MRI and myositis-specific antibody testing facilitates timely initiation of aggressive immunosuppressive therapy, which is essential for improving muscle strength, functional recovery, and long-term outcomes.

Keywords: ANTI SRP IMMUNE MEDIATED MYOPATHY

NEUROMELIOIDOSIS

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Abstract ID: 185

Aim/ Background: Neuromelioidosis is a rare CNS infection caused by *Burkholderia pseudomallei* and is characterized by high morbidity and mortality. Our report presents the diagnostic and therapeutic approach of the case of neuromelioidosis .

Materials & Methods: A 32-year-old man with a medical history of no comorbidities, no significant family history, non smoker, non alcoholic presented with complaints of severe headache and fever followed by dysarthria, swallowing difficulty and decreased level of consciousness on day 5 .

Results: After extensive investigations including MRI brain with contrast suggestive of flair hyperintensities in ventral pons, periventricular region of fourth ventricle extending to medulla and middle cerebellar peduncle ,cervical and dorsal cord with heterogeneous patchy contrast enhancement with ring like enhancement in cervical cord, CSF analysis suggestive of 405 cells with lymphocyte predominance , tb pcr negative, oligiclonal bands negative, biofire negative . Serum nmo mog antibodies negative, serum ace levels in normal limits . Treated with antibiotics and immunosuppresants. Microbiological cultures suggestive of *Burkholderia pseudomallei* ,diagnosis of neuromelioidosis was established. Antibiotics upgraded , the patient showed no clinical improvement and remained in a severely compromised neurological state under mandatory mechanical ventilationand patient succumbed to death.

Conclusion: Neuromelioidosis can pose a diagnostic challenge requiring an extensive diagnostic evaluation because of its uncommon clinical and radiological presentations.

Keywords: NEUROMELIOIDOSIS - infection by *burkholderia pseudomallei*.

DYKE-DAVIDOFF-MASSON SYNDROME VARIANT: A RARE CASE

REPORT ON ADULT PRESENTATION

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Abstract ID: 32

Aim/ Background: Dyke-Davidoff-Masson syndrome (DDMS) is a rare neurological condition characterized by cerebral hemiatrophy resulting from brain injury during fetal development or early childhood. This report aims to describe a rare clinical variant of DDMS in a 29-year-old female, highlighting the diagnostic importance of neuroimaging in differentiating this syndrome from other causes of cerebral atrophy

Materials & Methods: A 29-year-old female presented with recurrent generalized tonic-clonic seizures (2–5 episodes per week), left-sided hemiparesis, facial asymmetry, and persistent drooling. Clinical evaluation included a Mini-Mental State Examination (MMSE), neurological examination, and laboratory workup. Diagnostic confirmation was sought through Non-Contrast Computed Tomography (NCCT) of the brain and paranasal sinuses, alongside Electroencephalogram (EEG) monitoring

Results: Neurological examination revealed spastic hemiplegia (2/5\$ upper limb, 3/5\$ lower limb power) and cognitive impairment (MMSE: 15/30\$). NCCT revealed hemiatrophy and gliotic changes of the right cerebral hemisphere, dilatation of the ipsilateral lateral ventricle, right frontal sinus hyperpneumatization, and compensatory calvarial thickening. EEG findings showed focal and generalized wave slowing, confirming a seizure disorder

Conclusion: The patient's features align with the acquired subtype of DDMS, likely stemming from a traumatic event at age six. Management focused on a holistic approach using anticonvulsants (levetiracetam, clobazam), glycopyrrolate for sialorrhea, and baclofen for spasticity, supplemented by long-term physiotherapy. Awareness of DDMS is critical for clinicians to ensure early diagnosis and multidisciplinary management, particularly in cases with refractory seizures where surgical interventions like hemispherectomy may be indicated.

Keywords: Dyke-Davidoff-Masson Syndrome, Refractory Seizures, Hemiatro

HEREDITARY SPASTIC PARAPLEGIA (HSP): CLINICAL PROFILE AND REHABILITATION OUTCOMES

Dr. N.C.Sudarshan,

Rama Hospital hapur, Delhi

Abstract ID: 33

Aim/ Background: Hereditary Spastic Paraplegia (HSP) is a genetically diverse group of neurodegenerative disorders primarily characterized by progressive lower limb spasticity and weakness. While it is classified into "pure" and "complicated" forms based on the presence of additional neurological symptoms, the primary challenge remains the management of motor dysfunction. This report outlines a clinical case of HSP, detailing the presentation, diagnostic workup, and the efficacy of a comprehensive multidisciplinary rehabilitation strategy.

Materials & Methods: A clinical assessment was conducted on a patient presenting with classic signs of gait disturbance and progressive stiffness in both lower extremities. Evaluation included a detailed family history, neurological examination focusing on spasticity (Modified Ashworth Scale), muscle power, and deep tendon reflexes. Imaging and laboratory investigations were utilized to exclude secondary causes of spasticity. The management plan integrated pharmacological intervention with intensive physical and occupational therapy.

Results: Examination revealed hallmark features of HSP, including bilateral lower limb spasticity, hyperreflexia, and extensor plantar responses. Genetic and imaging studies supported the diagnosis of HSP by excluding structural spinal cord lesions. Following a structured rehabilitation protocol—incorporating muscle-strengthening exercises, stretching, and gait training—the patient demonstrated measurable improvements in mobility, a reduction in the frequency of painful spasms, and enhanced performance in daily living activities.

Conclusion: HSP significantly impacts patient autonomy due to its progressive nature. This case underscores that while there is currently no curative treatment, a holistic management approach is vital. The combination of early diagnosis and a robust multidisciplinary rehabilitation program can effectively mitigate symptoms, slow functional decline, and substantially improve the quality of life. Continued longitudinal monitoring remains essential to adapt therapeutic interventions to the patient's evolving needs.

Keywords: Neurodegenerative Disorders, Spastic Paraplegia, Genetic

COGNITIVE IMPAIRMENT IN PATIENTS WITH HYPERTENSION AND ITS CORRELATION WITH CLINICAL ASSESSMENT AND NEUROIMAGING

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Abstract ID: 34

Aim/ Background: Hypertension is a significant global health challenge and a leading risk factor for vascular cognitive impairment and dementia. Despite its prevalence, cognitive decline in hypertensive patients often remains underdiagnosed in early stages. This study aimed to evaluate the prevalence of cognitive impairment among hypertensive patients in a rural population of Western Uttar Pradesh and to correlate these deficits with clinical parameters and neuroimaging findings.

Materials & Methods: A hospital-based cross-sectional study was conducted involving 100 hypertensive patients. Cognitive function was assessed using the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA) scales. Detailed clinical histories, including duration of hypertension and treatment adherence, were recorded. Neuroimaging (MRI or CT brain) was performed to identify structural changes such as leukoaraiosis, cerebral atrophy, or silent infarcts. Statistical analysis was used to determine the correlation between blood pressure control, duration of illness, and cognitive scores.

Results: The study identified a high prevalence of cognitive impairment among hypertensive patients, particularly affecting executive function and memory. Longer duration of hypertension and poor blood pressure control were significantly correlated with lower MoCA scores ($p < 0.05$). Neuroimaging revealed that a significant percentage of patients exhibited white matter hyperintensities (leukoaraiosis) and cortical atrophy, which strongly associated with the severity of cognitive decline. Age and lower literacy levels were also identified as contributing factors to increased cognitive vulnerability.

Conclusion: Chronic hypertension is a major driver of subclinical brain injury and subsequent cognitive decline. The findings underscore the necessity of a multidisciplinary approach involving regular cognitive screening as part of routine hypertension management. Early identification of cognitive deficits and aggressive blood pressure regulation are critical to preventing progression to dementia. Public health initiatives should prioritize cognitive health education and integrated screening protocols to improve long-term functional outcomes in hypertensive populations.

Keywords: Cognitive Impairment, Hypertension, MoCA, Neuroimaging

THE CLINICAL SPECTRUM AND OUTCOME OF PATIENTS WITH FUNCTIONAL MOVEMENT DISORDERS

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Abstract ID: 400

Aim/ Background: Functional movement disorders (FMD) are characterised by abnormal involuntary movements associated with significant psychosocial burden and functional impairment. Early diagnosis and multidisciplinary management improve outcomes. **Aims and Objectives:** To study the clinical spectrum and three-month outcome of patients with functional movement disorders presenting to a tertiary care centre

Materials & Methods: This study included 80 patients fulfilling DSM-5 criteria for FMD presenting to Dayanand Medical College and Hospital, Ludhiana, between April 2024 and December 2025. Clinical characteristics, psychosocial factors, and treatment outcomes were assessed using the Psychogenic Movement Disorder Rating Scale (PMDRS) and Hospital Anxiety and Depression Scale (HADS) at baseline and three-month follow-up. Patients received multidisciplinary treatment comprising Cognitive Behaviour Therapy (CBT), pharmacotherapy, and physiotherapy.

Results: The mean age was 38 years, with female predominance (80%). Tremor (45%) was the commonest phenomenology, followed by dystonia (26.3%) . Multidisciplinary treatment was used . 40% of patients improved. PMDRS and HADS scores improved significantly following treatment ($p=0.001$ each). Shorter symptom duration ($p=0.004$) and use of CBT ($p=0.019$) were associated with favourable outcomes. Follow-up and delta PMDRS and HADS scores were significantly associated with outcome, indicating that changes over time are better predictors than baseline values. PMDRS severity differed significantly across phenotypes, whereas psychosocial improvement was independent of phenotype.

Conclusion: Tremor was the predominant FMD phenotype. Functional motor symptoms may occur as mixed phenotypes and are not confined to fixed neuroanatomical pathways. CBT improves symptoms by modulating attention and motor control mechanisms. Physiotherapy aids motor retraining, while pharmacotherapy alone is often insufficient. PMDRS is a valuable tool for monitoring treatment response and clinical outcomes in FMD. Early diagnosis and multidisciplinary management, particularly Cognitive Behaviour Therapy, are associated with improved clinical outcomes.

Keywords: Functional movement disorders, Psychogenic Movement Disorder Rating Scale (PMDRS) and Hospital Anxiety and Depression Scale (HADS), Cognitive Behaviour Therapy (CBT)

CO-OCCURRENCE OF PARANEOPLASTIC STRIATAL ENCEPHALITIS WITH BRAIN METASTASIS IN SMALL CELL LUNG CANCER

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Abstract ID: 401

Aim/ Background: Paraneoplastic striatal encephalitis (PSE) is a rare immune-mediated neurological syndrome, most commonly associated with small cell lung cancer (SCLC). It typically presents with subacute chorea and bilateral striatal abnormalities on MRI, making early diagnosis challenging, particularly in antibody-negative cases.

Materials & Methods: A 57-year-old nonsmoking woman presented with one month of gait instability, behavioural changes, and emotional lability. Brain MRI demonstrated bilateral caudate and putaminal T2/FLAIR hyperintensities with normal CSF findings. She showed partial clinical and radiological improvement following corticosteroid therapy. Persistent generalised chorea and a 20-kg weight loss prompted further evaluation. Repeat MRI revealed partial resolution of the striatal lesions with a new right temporal ring-enhancing lesion suggestive of metastasis. PET-CT demonstrated a left upper lobe lung mass with cervical nodal and brain metastases. Histopathology confirmed small cell neuroendocrine carcinoma. Comprehensive paraneoplastic antibody testing was negative. The patient received chemotherapy and palliative cranial radiotherapy.

Conclusion:

Discussion: The coexistence of antibody-negative PSE and brain metastasis in SCLC is exceptionally rare. Characteristic MRI findings, steroid responsiveness, and subacute chorea supported a paraneoplastic aetiology despite negative antibody testing, emphasising the importance of considering occult malignancy in similar presentations.

Conclusion: PSE should be suspected in patients with unexplained subacute chorea and bilateral striatal MRI abnormalities, even when antibody panels are negative. Early malignancy screening is crucial for timely diagnosis and appropriate management.

Keywords:

WHEN BREATHLESSNESS BECOMES NEUROLOGICAL

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Abstract ID: 183

Aim/ Background: Guillain–Barré Syndrome (GBS) may initially present with nonspecific neurological symptoms such as ataxia, making early diagnosis difficult—particularly when accompanied by systemic illness such as sepsis.

Materials & Methods: A 64-year-old male presented with acute gait imbalance and truncal ataxia on day 1, followed by rapidly progressive ascending bilateral lower-limb weakness, eventually involving upper limbs. Neurological examination showed flaccid quadriparesis, areflexia, and intact cranial nerves, with no sensory level or cortical signs. Initial labs demonstrated marked leukocytosis (TLC $17\text{--}18 \times 10^9/\text{L}$ with neutrophilia) and metabolic acidosis (pH 7.10; lactate 13.4 mmol/L), consistent with sepsis. Mild renal dysfunction and electrolyte abnormalities were also present. CT brain was normal, and the working diagnosis was GBS (AIDP variant) complicated by sepsis.

Results: The patient was shifted to the ICU for respiratory and autonomic monitoring. Broad-spectrum antibiotics were initiated for sepsis, along with fluid resuscitation and correction of metabolic derangements. IVIG therapy was started for GBS, and serial neurological and respiratory assessments were performed.

Conclusion:

This case underscores the importance of recognizing early ataxia as a GBS manifestation, especially when masked by coexisting sepsis, and highlights the need for rapid immunotherapy and vigilant critical care monitoring.

Keywords: Guillain–Barré Syndrome, AIDP, Ataxia, Ascending paralysis,

NEUROFIBROMATOSIS TYPE 1 AS A CAUSE OF ISCHEMIC STROKE IN THE YOUNG: A CASE OF UNDERRECOGNIZED VASCULOPATHY

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Abstract ID: 156

Aim/ Background: Neurofibromatosis type 1 (NF1) is an autosomal dominant disorder caused by mutations in the NF1 gene, characterized by diverse cutaneous, neurological, and systemic manifestations. Although NF1-associated vasculopathy is well recognized, it remains an underdiagnosed cause of ischemic stroke in children and young adults. We present a case of pediatric ischemic stroke secondary to NF1-associated carotid vasculopathy to highlight the importance of early recognition.

Materials & Methods: A 7-year-old girl presented with acute-onset speech difficulty followed by focal seizures involving the right upper limb and face. Clinical examination revealed multiple café-au-lait macules, axillary freckling, Lisch nodules, and limb anomalies fulfilling the diagnostic criteria for NF1. Neuroimaging demonstrated a left cortical infarct, CT angiography revealed significant stenosis of the left internal carotid artery from the petrous segment to its bifurcation, and genetic testing confirmed an NF1 mutation.

Results: The patient was diagnosed with acute ischemic stroke secondary to NF1-associated vasculopathy. Imaging findings correlated with the neurological deficits and demonstrated significant carotid artery stenosis, establishing the vascular etiology of stroke in the setting of genetically confirmed NF1

Conclusion: NF1-associated vasculopathy includes arterial stenosis, occlusion, aneurysm formation, and Moyamoya-like changes resulting from abnormal vascular remodeling due to neurofibromin deficiency. These abnormalities may remain clinically silent until a cerebrovascular event occurs. NF1 should be actively considered in young stroke patients with characteristic phenotypic features, as early diagnosis facilitates appropriate evaluation, risk stratification, and long-term management.

Keywords: Neurofibromatosis Type 1, Ischemic Stroke, Pediatric Stroke,

A COMPARATIVE STUDY OF THE PREVALENCE OF ANXIETY AND DEPRESSION IN PATIENTS WITH EPILEPSY AND PSYCHOGENIC NON- EPILEPTIC SEIZURES IN A TERTIARY CARE CENTRE.

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Abstract ID: 58

Aim/ Background: Psychogenic Non-Epileptic Seizures (PNES) and Epilepsy share overlapping clinical features, yet carry distinct neuropsychiatric profiles. Despite a growing global literature, comparative data from South Indian tertiary care populations remain limited. This study aimed to characterise and compare anxiety, depression, and cognitive function between confirmed Epilepsy and PNES patients.

Materials & Methods: Cross-sectional comparative observational study. One hundred patients (PNES n=50; Epilepsy n=50) were consecutively enrolled from the Department of Neurology, Government Medical College, Thiruvananthapuram. Diagnosis was confirmed by ILAE 2017 criteria (Epilepsy) and Video-EEG demonstrating habitual events without ictal correlate (PNES). Anxiety was assessed using GAD-7, depression using PHQ-9, and cognition using MoCA and MMSE. Statistical analysis was performed using IBM SPSS v27; chi-square, Fisher's exact test, and independent samples t-test were applied (significance: $p < 0.05$).

Results: The PNES group showed markedly higher anxiety (mean GAD-7: 16.70 vs 4.40; $p < 0.01$), with 90% scoring in the severe category versus 0% in Epilepsy. Depression scores were similarly elevated (mean PHQ-9: 20.08 vs 5.02; $p < 0.01$); 94% of PNES patients had moderately severe or severe depression versus 6% in Epilepsy. Mean cognitive scores did not differ significantly; however, mild cognitive impairment on MMSE was exclusive to PNES (12% vs 0%), while moderate impairment was exclusive to Epilepsy (4% vs 0%; $p = 0.03$).

Conclusion: PNES is associated with substantially higher anxiety and depression burden compared to Epilepsy, with qualitatively distinct cognitive impairment patterns. These findings underscore the critical need for routine neuropsychiatric screening and early referral for Cognitive Behavioural Therapy in all patients presenting with possible PNES.

Keywords: PNES, Epilepsy, Anxiety, Depression, Cognition.

IDIOPATHIC VENTRAL THORACIC CORD HERNIATION PRESENTING AS BROWN-SÉQUARD SYNDROME

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Government Medical College, Thiruvananthapuram, Kerala

Abstract ID: 79

Aim/ Background: Idiopathic spinal cord herniation is a rare, often misdiagnosed cause of progressive myelopathy mimicking Brown-Séquard syndrome. We report a case highlighting this under-recognized entity and its key imaging clues.

Materials & Methods: A 44-year-old right-handed homemaker presented with a 2.5-year history of progressive dissociated sensory loss (pain and temperature) in the right lower limb, followed 1.5 years later by left lower limb weakness, spasticity, and paresthesias up to T6, with transient bowel-bladder urgency. Neurological examination and contrast MRI of the cervical-dorsal spine were performed.

Results: Examination revealed left-sided pyramidal weakness with hyperreflexia and extensor plantar, contrasted by right-sided loss of pain and temperature sensation up to T6, with preserved touch bilaterally — a classic dissociated sensorimotor pattern. Vibration and joint position sense were impaired on the left. Routine bloodwork was unremarkable except mild microcytic anemia. MRI revealed focal anterior and rightward angulation and rotation of the dorsal cord at the D2-D3 level, with obliteration of the adjacent CSF cleft, cord thinning, and intramedullary T2 hyperintensity, without enhancement or diffusion restriction — diagnostic of ventral cord herniation with compressive myelomalacia.

Conclusion: The contralateral dissociation of spinothalamic and corticospinal-posterior column modalities localized the lesion to a ventral hemicord segment, explained by mechanical distortion at the herniation site. Idiopathic ventral cord herniation should be considered in chronic Brown-Séquard-type myelopathy, particularly when initial imaging appears unremarkable, since diagnosis hinges on subtle cues such as cord angulation, CSF cleft obliteration, and dorsal subarachnoid widening. Early recognition is crucial, as surgical reduction can halt or reverse decline.

Keywords: Ventral Cord Herniation, Brown Séquard

EARLY POST STROKE SEIZURES FOLLOWING ACUTE ISCHEMIC STROKE: A CROSS SECTIONAL STUDY FROM A TERTIARY CARE CENTRE IN KERALA

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Government T.D Medical College, Alappuzha, Kerala

Abstract ID: 54

Aim/ Background:

- Stroke is the most common cause of epilepsy in the adult population.
- Post stroke seizures are classified as early(ES) and late(LS)
- ES were classified as spontaneous seizures occurring within 1 week of the stroke event.

AIM:

- Clinical profile of patients with early post stroke seizures.
- Assess the risk factors for early post stroke seizures.

Materials & Methods:

- Inclusion criteria
- All consecutive patients admitted under neurology department during the study period with acute ischemic stroke.
- Exclusion criteria
- All patients with a previous stroke, TIA, CVT, prior history of seizures, or any other epileptogenic comorbidity.
- All patients who met the inclusion criteria were grouped into as those with early post stroke seizures (within one week of stroke event) and those without.
- NIHSS score was used to assess the severity of stroke. Stroke subtype was classified based on the TOAST classification.

Results:

- Early seizures occurred in 20 (10.0%) of acute ischemic stroke patients in our cohort.
- Patients with ES were younger, had a more severe stroke at presentation, had a cortical location of the infarct, and underwent lesser recanalization procedures.
- Large artery disease and cardioembolic subtypes were more common in patients with ES.

Conclusion:

- Early seizures occurred in 20 (10.0%) of acute ischemic stroke patients in our cohort.
- Those individuals at high risk for ES, can be given prophylactic antiseizure drugs which will decrease their infarct volume and morbidity.

Keywords: Early post stroke seizures , Acute ischemic stroke

A STUDY ON PATTERNS OF COGNITIVE IMPAIRMENT IN PATIENTS WITH PURE CEREBELLAR SYNDROME

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Abstract ID: 55

Aim/ Background: Although the cerebellum has classically been associated with motor coordination, accumulating evidence highlights its role in cognition and affect. Cerebellar Cognitive Affective Syndrome (CCAS) is frequently underrecognized, particularly in patients presenting with isolated cerebellar signs. Characterizing the pattern and severity of cognitive impairment in pure cerebellar syndromes is essential for improving diagnosis and management.

1. Objectives: To assess cognitive impairment across different domains in patients with pure cerebellar syndrome using the CCAS/Schmahmann scale
2. To assess the correlation between the extent of cerebellar involvement according to ICARS scale and the degree of cognitive impairment.

Materials & Methods: This descriptive study included 30 adults (>18 years) with clinically and radiologically confirmed pure cerebellar syndrome attending a tertiary care center. Patients with major cognitive disorders, Parkinsonism, psychiatric illness, or non-cerebellar MRI lesions explaining cognitive decline were excluded. Cognitive function was assessed using the CCAS scale, and cerebellar motor impairment was quantified using ICARS. Demographic, clinical, and etiological data were recorded. All patients were reassessed after three months. Statistical analysis was performed using paired t-tests and Pearson correlation.

Results: The mean age was 56.2 ± 10.48 years, with male predominance (70%). Vascular etiology was most common (56.7%), followed by neurodegenerative causes (23.3%), drug-induced ataxia (10%), and space-occupying lesions (10%). At baseline, 96.7% met criteria for definite CCAS. Executive dysfunction and verbal fluency deficits were the most affected domains. A strong negative correlation was observed between CCAS and ICARS scores at baseline and follow-.

Conclusion: Cognitive impairment is highly prevalent in pure cerebellar syndromes and closely correlates with motor severity. Aetiology significantly influences cognitive trajectory, underscoring the importance of routine cognitive screening and targeted rehabilitation in cerebellar disorders.

Keywords: Cognitive impairment, Pure cerebellar syndrome

UPREGULATED DIFFERENTIAL GENE EXPRESSION OF NLRP3 INFLAMMASOME PATHWAY IN THE CEREBROSPINAL FLUID (CSF) AND BLOOD OF IMMUNE-MEDIATED OPTIC NEURITIS PATIENTS

Dr. Oshin, Dr. Karthik V. M.

PGIMER, Chandigarh

Abstract ID: 172

Aim/ Background: Optic neuritis (ON) is an acute inflammatory demyelinating disorder of optic nerve. Reliable biomarkers for subtype differentiation and early diagnosis are currently lacking. We studied the NLRP3 inflammasome pathway in the CSF and blood of ON patients to identify potential biomarkers.

Materials & Methods: There were samples available from 42 acute ON patients (MSON, NMOSD, and MOGAD, 14 each subtype) and 25 non-neurological controls. CSF, blood samples were analysed for target gene expression (NLRP3, IL-18, IL-1 β) and quantified via RT-qPCR. Housekeeping gene (GAPDH) was analysed to derive the differential gene expression.

Results: The all ON cases showed marked upregulation of NLRP3 (38.20-fold CSF / 221.45-fold Blood) and IL-18 (107.31-fold CSF / 138.23-fold Blood) compared to controls, while IL-1 β remained near baseline (<1.0-fold). Distinct subgroup signatures emerged across compartments: NMOSD exhibited peak NLRP3 upregulation in both CSF (129.46-fold) and Blood (384.77-fold) alongside massive peripheral IL-18 (916.62-fold). MOGAD displayed intrathecal IL-18 hyperactivation in CSF (200.63-fold) contrasting with lower Blood IL-18 (21.18-fold) and minimal CSF NLRP3 (4.81-fold). MS-ON maintained symmetrical co-activation of NLRP3 (89.57-fold CSF / 103.56-fold Blood) and IL-18 (139.69-fold CSF / 136.04-fold Blood).

Conclusion: The significant, distinct hyperactivation of the NLRP3/ IL-18 axis highlights the role of innate immunity in ON pathogenesis. These preliminary results offer insights into the role of NLRP3 and downstream molecules in the inflammatory pathogenesis of this entity. These signatures may help identify patients likely to have a worse prognosis, requiring more intensive immunotherapy.

Keywords: : Interleukin-18 (IL-18), Neuroinflammation, Multiple Sclero

UPREGULATED DIFFERENTIAL GENE EXPRESSION OF NLRP3 INFLAMMASOME PATHWAY IN THE CEREBROSPINAL FLUID (CSF) OF IMMUNE-MEDIATED OPTIC NEURITIS PATIENTS

Dr. Oshin

PGIMER, Chandigarh

Abstract ID: 175

Aim/ Background: Optic neuritis (ON) is an acute inflammatory demyelinating disorder of optic nerve. Reliable biomarkers for subtype differentiation and early diagnosis are currently lacking. We studied the NLRP3 inflammasome pathway in the CSF of ON patients to identify potential biomarkers.

Materials & Methods: There were samples available from 42 acute ON patients (MSON, NMOSD, and MOGAD, 14 each subtype) and 25 non-neurological controls. CSF samples were analysed for target gene expression (NLRP3, IL-18, IL-1 β) and quantified via RT-qPCR. Housekeeping gene (GAPDH) was analysed to derive the differential gene expression.

Results: ON cases showed significant upregulation of NLRP3 (38.2-fold) and IL-18 (107.31-fold) compared to controls, however IL-1 β did not show any significant change (0.57-fold). In addition, we observed distinct signatures in the subgroups: MS-ON exhibited upregulated expression of NLRP3 (89.57-fold) and IL-18 (139.69-fold). NMOSD showed the highest NLRP3 upregulation (129.46-fold) alongside elevated IL-18 (44.92-fold). MOGAD displayed IL-18 upregulation (200.6-fold) without any significant elevation of NLRP3 (4.81-fold).
Sr. No. CASES CSF NLRP3

(fold change) IL-18

(fold change) IL-1 BETA

(fold change)

1	All ON Cases	38.2	107.31	0.57
2	NMOSD	129.46	44.92	1.52
3	MOGAD	4.81	200.63	0.23
4	MS-ON	89.57	139.69	0.54

Conclusion: The significant, distinct hyperactivation of the NLRP3/ IL-18 axis highlights the role of innate immunity in ON pathogenesis. These preliminary results offer insights into the role of NLRP3 and downstream molecules in the inflammatory pathogenesis of this entity. These signatures help identify patients likely to have a worse prognosis, requiring more intensive immunotherapy.

Keywords: IL-18, Multiple Sclerosis (MS), NMOSD, MOGAD

NEUROSYPHILIS PRESENTING AS LONGITUDINALLY EXTENSIVE TRANSVERSE MYELITIS: A DIAGNOSTIC MIMIC

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Abstract ID: 16

Aim/ Background: Neurosyphilis, which is a rare but treatable condition, can cause non-compressive myelopathy and look like other diseases that affect the myelin, such as longitudinally extensive transverse myelitis (LETM). Recognizing it early is important to avoid late diagnosis and serious long-term damage

Materials & Methods: We describe a 29-year-old man who had weakness in both legs for 20 days and trouble urinating for 10 days. His exam showed UMN weakness with sensory level of T10 level. A spinal MRI showed a long area of increased signal in the spinal cord from C4 to D10. CSF showing lymphocytic wbc & higher protein levels

Results: The patient was initially thought to have an inflammatory myelopathy and given iv and oral steroids. Even though the MRI shows improvement but his symptoms didn't improve. Because of this mismatch, more tests were done. His blood tests for syphilis came positive, but his spinal fluid test was negative. Based on his symptoms, spinal fluid findings, and positive blood tests, doctors diagnosed him with neurosyphilis that looked like meningomyelitis. He was then treated with iv ceftriaxone and his condition improved

Conclusion: Neurosyphilis can be hard to tell apart from LETM and other demyelinating diseases. Although CSF VDRL is a good test, it might not always find syphilis. So, a negative test doesn't rule out neurosyphilis. The diagnosis should consider the patient's symptoms, blood tests, and spinal fluid results. It's important to check for syphilis in cases of unusual or steroid-resistant myelopathy, because early treatment can greatly improve the outcome

Keywords: Neurosyphilis, LETM, Noncompressivemyelopathy

FROM EMBERS TO SMOKE

SERIAL EVOLUTION OF ADULT INFLAMMATORY FOCAL CEREBRAL ARTERIOPATHY TO MOYAMOYA SYNDROME

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Abstract ID: 402

Aim/ Background: Adult inflammatory focal cerebral arteriopathy (FCA) is an uncommon intracranial vasculopathy with an unpredictable course. Although immunotherapy targets vascular inflammation, progression to moyamoya syndrome is exceptionally rare. We describe the complete clinicoradiological evolution of inflammatory FCA to unilateral moyamoya syndrome documented by serial multimodal imaging and successful cerebral revascularization.

Materials & Methods: A 43-year-old man presented with recurrent stereotyped transient episodes of left upper-limb weakness, numbness, and facial deviation lasting 25–30 seconds with preserved awareness, suggesting focal aware seizures versus transient ischemic attacks. Evaluation included high-resolution vessel-wall MRI, cerebrospinal fluid analysis with infectious and autoimmune work-up, serial MR angiography, digital subtraction angiography, and postoperative imaging.

Results: Initial vessel-wall MRI demonstrated concentric enhancement of the right intracranial internal carotid artery, consistent with inflammatory FCA. Despite negative etiological evaluation, corticosteroids, azathioprine, antiplatelet therapy, and antiseizure medication reduced neurological events. Serial imaging, however, revealed persistent vessel-wall enhancement, progressive supraclinoid ICA–MCA stenosis, evolving watershed infarcts, and robust pial collateralization on DSA, confirming unilateral moyamoya syndrome. Right STA–MCA direct with indirect bypass achieved successful cerebral revascularization, and the patient remains clinically stable without recurrent transient neurological events.

Conclusion: This case illustrates the complete biological continuum of adult inflammatory FCA, demonstrating that clinical improvement may conceal ongoing vascular progression. Serial vessel-wall MRI and DSA are indispensable for timely therapeutic escalation and neurosurgical intervention. When symptoms fall silent, the artery may still be writing its own story.

Keywords: OFFLINE

GAS GEYSER SYNDROME: FATAL CAUSE OF HYPOXIA IN WINTERS

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Abstract ID: 152

Aim/ Background: Gas geyser syndrome is an uncommon yet potentially fatal condition resulting from carbon monoxide (CO) poisoning caused by improper installation or use of LPG gas-powered water heaters.

Materials & Methods: A 14-year-old female was brought to the Emergency Department (ED) by her parents with a chief complaint of unconsciousness and seizures. According to the history provided, the patient had gone to take a bath approximately 45 minutes prior. Her parents became concerned when she did not respond to knocking. upon breaking open the door, they found her lying unconscious on the bathroom floor. The bathroom was described as small (approx. 6x4 feet) with no exhaust fan and the single window closed. An LPG gas geyser was installed inside the bathroom and was found running.

Results: Based on the history of exposure in an enclosed space with a gas geyser, the clinical presentation of altered sensorium and seizures, and the elevated Carboxyhaemoglobin (COHb) levels, a diagnosis of Severe Carbon Monoxide Poisoning (Gas Geyser Syndrome) with hypoxic-ischemic encephalopathy was made.

Conclusion: This case underscores the importance of awareness, prompt diagnosis, and preventive measures to mitigate the risk of CO poisoning.

Keywords: gas geyser, CO poisoning, Hypoxia

ETHAMBUTOL-INDUCED OPTIC NEUROPATHY: A DUAL-HIT MECHANISM LINKING MITOCHONDRIAL INJURY AND HLA-DEPENDENT IMMUNE SUSCEPTIBILITY

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PGIMER, Chandigarh

Abstract ID: 139

Aim/ Background: Ethambutol-induced optic neuropathy (EtON) is a serious adverse effect of ethambutol therapy that can lead to irreversible vision loss. Considerable interindividual variability in susceptibility suggests an underlying genetic predisposition. Human leukocyte antigen (HLA) alleles are established mediators of several drug-induced adverse reactions, but their role in EtON remains poorly understood. This study evaluated the association of HLA-DQB1*05:01 and HLA-DRB1*12:01 with susceptibility to EtON

Materials & Methods: A case–control study included 100 patients with EtON and 190 ethnically matched ethambutol-exposed controls without optic neuropathy. Clinical and demographic data, including age, sex, body mass index, diabetes mellitus, and hypertension, were recorded. High-resolution HLA genotyping was performed using polymerase chain reaction with sequence-specific primers (PCR-SSP). Allele frequencies were compared, and multivariable logistic regression was used to estimate adjusted odds ratios (ORs) and 95% confidence intervals (CIs).

Results: Both HLA-DQB1*05:01 and HLA-DRB1*12:01 were significantly more frequent in EtON cases than controls. HLA-DQB1*05:01 demonstrated a strong association with EtON (OR 4.55, 95% CI 2.59–7.98; $p < 0.001$), while HLA-DRB1*12:01 was also significantly associated (OR 2.23, 95% CI 1.31–3.79; $p = 0.004$). These associations remained significant after adjustment for demographic and clinical variables (adjusted OR 3.77, 95% CI 1.80–7.93; and adjusted OR 1.83, 95% CI 0.91–3.70, respectively).

Conclusion: HLA-DQB1*05:01 and HLA-DRB1*12:01 are independently associated with susceptibility to EtON, supporting an immune-genetic contribution to disease pathogenesis. These findings suggest a potential dual-hit model, in which ethambutol-induced mitochondrial toxicity interacts with HLA-mediated host susceptibility to trigger optic nerve injury. Pre-treatment HLA screening may enable personalized risk stratification and reduce vision-threatening toxicity.

Keywords: EtON; HLA Class II alleles; pharmacogenomics

POST-INFECTIOUS ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY (AMSAN) FOLLOWING SUSPECTED PLASMODIUM VIVAX MALARIA WITH MENINGOENCEPHALITIS

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Dr Nidhi Tyagi

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Abstract ID: 188

Aim/ Background: Background

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that may occur after infectious illnesses. The acute motor and sensory axonal neuropathy (AMSAN) variant is uncommon and can cause rapidly progressive weakness with substantial functional morbidity. Recognition may be challenging when neurological manifestations occur during or after severe systemic infection and encephalopathy.

Materials & Methods: A 66-year-old man with type 2 diabetes mellitus presented with 7–10 days of high-grade fever with chills and rigor, followed by 6–9 days of altered sensorium and abnormal body movements. An outside peripheral smear was reported positive for *Plasmodium vivax*; subsequent thin and thick smears at our centre were negative. On admission, he was febrile (102°F), tachycardic and drowsy with a Glasgow Coma Scale of E3V2M5; neck rigidity was present. Cerebrospinal fluid showed 27 cells/cmm with 90% mononuclear cells, protein 132 mg/dL and normal glucose. The clinical and CSF picture was considered suggestive of meningoencephalitis, and tubercular meningitis was also considered; anti-tubercular therapy with dexamethasone and pyridoxine was initiated while further investigations were pursued. He received intravenous artesunate, empirical antibacterial therapy, anticonvulsant treatment and supportive intensive care. His sensorium progressively improved, after which new upper-limb weakness became apparent. Neurological examination showed power 3/5 in the upper limbs and 4/5 in the lower limbs. Nerve conduction studies demonstrated reduced CMAP amplitudes and abnormalities in motor conduction, with the study interpreted as a motor and sensory axonal polyneuropathy. In the setting of a preceding malarial illness and mildly elevated CSF protein, Neurology considered a post-infectious AMSAN variant of GBS.

Results: IVIG was advised, subject to affordability, with continued physiotherapy and symptomatic management.

Conclusion: This case highlights the diagnostic complexity of acute neurological deterioration following a febrile illness. A post-infectious axonal GBS phenotype should be considered when new flaccid weakness develops after improvement in systemic and encephalopathic features. Serial neurological examination and electrodiagnostic testing are particularly important for identifying evolving AMSAN, especially when the initial presentation is dominated by fever and altered sensorium.

Keywords: Guillain–Barré syndrome; AMSAN; *Plasmodium vivax*; malaria

POST-INFECTIOUS ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY (AMSAN) FOLLOWING SUSPECTED PLASMODIUM VIVAX MALARIA WITH MENINGOENCEPHALITIS

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Abstract ID: 413

Aim/Background: Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that may occur after infectious illnesses. The acute motor and sensory axonal neuropathy (AMSAN) variant is uncommon and can cause rapidly progressive weakness with substantial functional morbidity. Recognition may be challenging when neurological manifestations occur during or after severe systemic infection and encephalopathy.

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Conclusion: This case highlights the diagnostic complexity of acute neurological deterioration following a febrile illness. A post-infectious axonal GBS phenotype should be considered when new flaccid weakness develops after improvement in systemic and encephalopathic features. Serial neurological examination and electrodiagnostic testing are particularly important for identifying evolving AMSAN, especially when the initial presentation is dominated by fever and altered sensorium.

Keywords: Guillain–Barré syndrome; AMSAN; *Plasmodium vivax*; malaria; meningoencephalitis; axonal polyneuropathy; post-infectious neuropathy; IVIG

CERVICAL MENINGOCELE WITH SYRINGOMYELIA AND LUMBOSACRAL MENINGOCELE WITH TETHERED FILUM TERMINALE: A REPORT OF TWO INFANT CASES

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Abstract ID: 103

Aim/ Background: Spinal dysraphism comprises a heterogeneous group of congenital spinal malformations resulting from abnormal neurulation. Although meningocele is generally associated with a favorable prognosis, occult anomalies such as syringomyelia or tethered cord may significantly influence management and neurological outcomes. We report two infant cases demonstrating distinct anatomical presentations and associated abnormalities.

Materials & Methods: A retrospective review was performed of two infants with congenital spinal swellings managed surgically at a tertiary-level neurosurgical center. Clinical examination, magnetic resonance imaging (MRI), intraoperative findings, surgical management, and postoperative outcomes were analyzed.

Results: Case 1 involved a six-month-old female with a posterior cervical swelling. MRI demonstrated a C3–C4 cervical meningocele with an associated C5–D2 syrinx. Microsurgical excision with C5 laminectomy confirmed the absence of neural tissue within the sac and no cord tethering. Case 2 involved a four-month-old female with a lumbosacral swelling. MRI revealed an L5–S1 meningocele associated with a tethered filum terminale. Surgical excision with detethering and watertight dural closure was performed successfully. Both patients had intact neurological function preoperatively and experienced uneventful postoperative recovery without new neurological deficits.

Conclusion: These cases highlight the spectrum of associated anomalies encountered in meningoceles despite preserved neurological status. Comprehensive preoperative MRI and meticulous microsurgical exploration remain essential to identify occult pathology, guide definitive surgical management, and prevent delayed neurological deterioration. Early intervention offers excellent functional outcomes, particularly before the onset of irreversible neurological deficits.

Keywords: Spinal Dysraphism, Cervical Meningocele, Syringomyelia

MORE THAN JUST A CROOKED SMILE

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Abstract ID: 64

Aim/ Background: Recurrent idiopathic facial nerve palsy is uncommon and warrants evaluation for alternative etiologies beyond Bell's palsy. We report a rare case of recurrent lower motor neuron facial nerve palsy associated with menstrual cycles, highlighting the role of hormonal fluctuations in the reactivation of latent viral infections.

Materials & Methods: A 24-year-old female presented with right-sided deviation of the angle of the mouth, inability to close the left eye, inability to frown, absent forehead wrinkling on the left side, and inability to blow from the left side of the mouth. Clinical examination revealed left lower motor neuron seventh cranial nerve paresis corresponding to House–Brackmann Grade IV.

The patient had experienced approximately 50 episodes of left facial palsy over the preceding 2.5 years. Detailed history and examination were undertaken to exclude alternative causes. There was no history suggestive of autoimmune disease, sarcoidosis, neuropathy, stroke, or Melkersson–Rosenthal syndrome.

Investigations included nerve conduction studies with facial nerve assessment, magnetic resonance imaging of the brain, serum angiotensin-converting enzyme levels with high-resolution computed tomography of the chest, antinuclear antibody testing, rheumatoid factor, perinuclear anti-neutrophil cytoplasmic antibody, cytoplasmic anti-neutrophil cytoplasmic antibody, and serological testing for Herpes simplex virus, Cytomegalovirus, Epstein–Barr virus, and Rubella virus.

Results: Neurological examination confirmed recurrent left lower motor neuron facial nerve palsy without other cranial nerve involvement, sensory deficits, motor weakness, or deep tendon reflex abnormalities. During follow-up, the patient reported that most episodes coincided with the commencement of menstruation.

Serological evaluation suggested previous viral exposure consistent with Herpes simplex virus and Rubella virus infection. After exclusion of other etiologies, a diagnosis of recurrent left lower motor neuron seventh nerve paresis secondary to Herpes simplex virus reactivation with catamenial association was established.

Conclusion: Catamenial recurrence of Bell's palsy is exceedingly rare. Hormonal withdrawal at the onset of menstruation, particularly the decline in estrogen and progesterone levels, may alter cytokine balance and reduce cell-mediated immunity through transient suppression of CD8-positive T-cell and natural killer cell activity. This immunological shift can facilitate reactivation of latent viruses within the geniculate ganglion.

Subsequent viral replication leads to inflammatory neuritis, nerve edema, entrapment within the facial canal, ischemia, and segmental demyelination, manifesting clinically as recurrent facial nerve palsy.

Recognition of menstrual association in recurrent Bell's palsy is important, as targeted management with perimenstrual antiviral prophylaxis may reduce recurrence and improve patient outcomes. This case emphasizes the need for careful history-taking and consideration of uncommon triggers in patients presenting with recurrent facial nerve palsy.

Keywords: Bell's Catamenial Herpes Reactivation Menstruation

PREDICTORS OF TRIPTAN FAILURE IN MIGRAINE PATIENTS: A PROSPECTIVE CROSS-SECTIONAL STUDY FROM A QUATERNARY CARE HOSPITAL IN INDIA

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Abstract ID: 101

Aim/ Background: Triptans remain the cornerstone of acute migraine therapy, but a substantial proportion of patients fail to achieve adequate response, and data on predictors of triptan failure from India remain limited. This study aimed to determine the rate and predictors of triptan failure in migraine patients attending a quaternary care hospital in India.

Materials & Methods: In this cross-sectional prospective study, adult migraine patients diagnosed using ICHD-3 criteria and treated with at least one triptan were enrolled. Clinical and demographic variables were recorded, and predictors of triptan non-response were identified using univariate analysis followed by multivariable logistic regression.

Results: Of 300 patients, 240 (80.0%) were triptan responders and 60 (20.0%) were triptan failures, comprising 47 (15.7%) non-responders, 9 (3.0%) triptan-resistant, and 4 (1.3%) triptan-refractory patients. The data was verified by two neurologists in blinded manner. On multivariable analysis, higher HIT-6 score, photophobia/phonophobia, headache duration >2 hours, attack duration >4 hours, limitation of daily activities, headache frequency >15 days/month, and NPRS pain score >7/10 were independent predictors of triptan failure.

Conclusion: One in five migraine patients failed triptan therapy in this cohort. Higher HIT-6 scores, photophobia/phonophobia, longer headache/attack duration, activity limitation, high attack frequency, and severe pain intensity independently predicted failure, highlighting the need for early identification and tailored acute treatment in high-risk patients.

Keywords: Migraine; Triptan failure; Triptan resistance; HIT-6

GENETICALLY CONFIRMED CAPN3-ASSOCIATED LIMB-GIRDLE MUSCULAR DYSTROPHY (LGMDR1) IN AN ADOLESCENT

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Abstract ID: 93

Aim/ Background: Limb-girdle muscular dystrophies (LGMDs) are a group of diseases that affect the muscles. Among these are limb-girdle muscular dystrophy type R1 (LGMDR1), previously known as LGMD2A. LGMDR1 is caused by mutations in the CAPN3 gene. The diagnosis of these diseases is often confirmed through genetic testing for the specific mutation in the CAPN3 gene. We report the clinical and molecular findings of an adolescent with LGMDR1.

Materials & Methods: A 15-year-old boy who exhibited steady difficulty rising from a squatting position and increasing lower-limb weakness was examined in a complete clinical assessment, neurological examination, serum creatine phosphokinase (CPK) testing and molecular genetic analysis. The genetic variant that was identified in this patient was classified according to the American College of Medical Genetics and Genomics (ACMG) guidelines.

Results: The examination of the child revealed weakness of the hip flexor muscles as well as calf pseudohypertrophy. The levels of the serum CPK enzyme were markedly elevated at 6,154 U/L. Also, genetic analysis of the child's DNA revealed a homozygous splice-site variant at the 3' splice acceptor site of intron 18 of the CAPN3 gene. According to the ACMG criteria for pathogenic variants (PVS1_Very Strong, PM2_Moderate and PP1_Strong), the variant was classified as pathogenic and the child was diagnosed with CAPN3-related LGMDR1.

Conclusion: This case highlights the importance of recognizing the condition of calpainopathy in adolescents who present with muscle weakness in the proximal muscles, calf pseudohypertrophy and increased levels of creatine kinase in their serum. By diagnosing this condition through molecular testing, it becomes possible to manage the condition appropriately for the individual, to counsel their family members and to conduct genetic testing on family members who are at risk of developing the condition. Such cases also contribute to an increased understanding of the genetic condition of LGMDR1

Keywords: Limb-girdle muscular dystrophy type R1 (LGMDR1); Calpainopat

WHEN THE PRESSURE DROPS : A CASE OF POSTPARTUM SPONTANEOUS INTRACRANIAL HYPOTENSION

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Abstract ID: 3

Aim/ Background: Postpartum headache is a common clinical entity with diverse etiologies. is a rare but important cause, often under-recognized and misdiagnosed as . Early identification is essential to ensure appropriate management and avoid complications.

Materials & Methods: We report a case of a postpartum female presenting with orthostatic headache. Clinical evaluation, laboratory investigations, and neuroimaging were performed. Differential diagnoses including and were systematically excluded.

Results: The patient presented with severe headache aggravated on standing and relieved on lying down, associated with nausea and neck pain. There was no history of recent dural puncture. MRI brain revealed diffuse pachymeningeal enhancement and features of brain sagging, suggestive of intracranial hypotension. The patient showed partial response to conservative management and significant improvement following epidural blood patch.

Conclusion: should be considered in postpartum patients with orthostatic headache, particularly in the absence of neuraxial procedures. Differentiation from is crucial. MRI plays a key role in diagnosis, and epidural blood patch remains an effective treatment in refractory cases. Early recognition leads to favorable outcomes.

Keywords: Postpartum headache, intracranial hypotension, CSF leak, epi

WHEN DOPAMINE STOPS : A LIFE THREATENING PARKINSONIAN CRISIS

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Abstract ID: 4

Aim/ Background: It is a rare but life-threatening neurological emergency seen in patients with Parkinson's disease following abrupt withdrawal or reduction of dopaminergic therapy. It closely mimics and requires early recognition to reduce morbidity and mortality.

Materials & Methods: We report a case of a known Parkinson's disease patient presenting with acute onset fever, rigidity, and altered sensorium. Clinical history, drug compliance review, laboratory investigations, and supportive imaging were performed. Differential diagnoses including sepsis, , and metabolic causes were considered and excluded.

Results: The patient presented with hyperpyrexia, severe generalized rigidity, altered mental status, and autonomic instability. Laboratory findings revealed elevated creatine kinase levels suggestive of rhabdomyolysis. History revealed recent interruption of dopaminergic medications. Prompt reinstitution of levodopa via nasogastric route, along with supportive care including intravenous fluids and temperature control, led to significant clinical improvement.

Conclusion: This should be suspected in Parkinson's disease patients presenting with fever and rigidity, especially with recent drug withdrawal. Early differentiation from and other mimics is crucial. Immediate reinstitution of dopaminergic therapy is the cornerstone of management and can be lifesaving.

Keywords: Parkinson's disease, hyperpyrexia, dopaminergic withdrawal,

A CASE OF SUBACUTE SCLEROSING PAN ENCEPHALITIS: A DIAGNOSTIC CONUNDRUM

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Abstract ID: 94

Aim/ Background: Subacute sclerosing pan encephalitis(SSPE) is a rare, progressive, fatal neurological complication of mutant measles virus infection characterised by inflammation and extensive degeneration of brain tissue. With increasing number of measles cases in areas with suboptimal vaccine coverage, SSPE poses huge threat to public health.

Materials & Methods: At a tertiary care centre, an extensive history and clinical examination was done, following which requitted investigations were performed after informed consent of the parents of the patient. A poster was then created for this case report presentation.

Results: A 6 year old, unvaccinated child with preterm birth and congenital hydrocephalus history presented with rapidly progressive decline in cognition, myoclonic jerks, ataxia, bowel-bladder incontinence and behavioural changes. His mother was diagnosed to have measles infection prior to her gestational period. Initial blood investigations were unremarkable. MRI brain showed Peri ventricular leukomalacia sequelae to perinatal hypoxic insult. EEG showed B/L fronto- temporal central region quasi periodic epileptiform discharges. CSF analysis was unremarkable. With SSPE as possible diagnosis IgG antibody titres for measles were sent which were positive in both CSF and serum. Hence the diagnosis of SSPE was made and patient was started on Isoprinosine and discharged.

Conclusion: Typical presentation of SSPE is 7 to 10 years after measles infection. But this case highlights that low threshold for suspicion of SSPE needs to be kept for children with congenital hydrocephalus or non vaccination history presenting with myoclonic jerks in early age of life for timely diagnosis and intervention.

Keywords: SSPE, Measles, Congenital Hydrocephalus, Myoclonic jerks

ROLE OF GUT MICROBIOTA DYSBIOSIS ON THE FUNCTIONAL OUTCOMES IN ISCHEMIC AND HAEMORRHAGIC STROKE PATIENTS

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Abstract ID: 128

Aim/ Background: Stroke is a leading cause of death and long-term disability worldwide, yet the biological determinants of post-stroke recovery remain incompletely understood. Recent evidence implicates the gut microbiome as a key regulator of neuroinflammation, metabolic signalling, and immune response through microbiota-gut brain axis. However, the extent to which gut microbial dysbiosis influences neurological severity and functional recovery across ischemic and haemorrhagic stroke remains unclear.

Materials & Methods: We performed a comprehensive analysis of published clinical studies to investigate associations between gut microbiome alterations and stroke outcomes. Studies indexed in major biomedical databases up to July 2025 were evaluated, yielding 23 eligible cohorts comprising 2,951 participants. Microbial diversity, taxonomic composition, and microbiota-derived metabolites were analysed in relation to standardized clinical measures, including neurological severity (NIHSS) and functional outcomes (modified Rankin Scale and Barthel Index).

Results: Stroke was consistently associated with significant disruption of gut microbial ecology. Reduced alpha diversity and marked shifts in beta diversity were observed across cohorts, indicating loss of microbial richness and community restructuring. Depletion of short-chain fatty acid-producing commensals, including *Roseburia*, *Faecalibacterium*, and *Coprococcus*, was accompanied by enrichment of opportunistic and pro-inflammatory taxa such as *Enterobacteriaceae*, *Enterococcus*, and *Escherichia/Shigella*. Metabolic profiling revealed reduced SCFA production alongside enhanced endotoxin-related and trimethylamine-N-oxide (TMAO) pathways. These alterations correlated with greater neurological severity, increased disability, higher infection risk, and poorer functional recovery.

Conclusion: Gut microbial dysbiosis represents a consistent and biologically relevant feature of stroke, characterized by ecological disruption and metabolic imbalance. These findings support a mechanistic role for the microbiota-gut-brain axis in modulating stroke outcomes and highlight microbiome-based biomarkers as promising tools for early risk stratification and targeted therapeutic intervention.

Keywords: Gut microbiota, Stroke recovery, Dysbiosis

SOY MILK-ASSOCIATED GUT MICROBIOTA AND FRONTOTEMPORAL DEMENTIA RISK: A MENDELIAN RANDOMIZATION STUDY

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Abstract ID: 21

Aim/ Background: Dietary factors and gut microbiota may contribute to neurodegenerative disorders, but their causal relevance to specific dementia subtypes remains unclear. This study investigated whether genetically predicted soy milk intake is associated with frontotemporal dementia (FTD) risk and explored potential mediation by gut microbiota.

Materials & Methods: We performed a two-sample Mendelian randomization analysis using genome-wide association study summary data for milk subtype intake, gut microbiota, and dementia subtypes. Six milk-related phenotypes were evaluated against five dementia outcomes. Inverse variance weighting was used as the primary method, with weighted median, MR-Egger, MR-PRESSO, heterogeneity testing, and leave-one-out analyses used for sensitivity assessment. A two-step Mendelian randomization framework was applied to explore gut microbiota-mediated pathways.

Results: Among the evaluated milk subtypes, genetically predicted soy milk intake showed a suggestive inverse association with FTD risk (OR = 0.261, 95% CI: 0.108–0.635, P = 0.003). Other milk subtypes showed no consistent association with dementia outcomes. In exploratory mediation analysis, soy milk intake was associated with several gut microbial taxa and pathways. *Faecalibacterium prausnitzii* was identified as a candidate mediator, accounting for 14.75% of the total association between soy milk intake and FTD risk. Sensitivity analyses did not indicate substantial heterogeneity or directional pleiotropy.

Conclusion: These findings provide preliminary genetic evidence linking soy milk intake to lower FTD risk, potentially involving gut microbiota-related pathways. Further validation in independent cohorts and mechanistic studies is warranted.

Keywords: Soy milk, Milk subtype, Dementia, Frontotemporal dementia, G

WHOLE MILK INTAKE, CEREBROSPINAL FLUID TMAO, AND INTRACEREBRAL HEMORRHAGE RISK: A MENDELIAN RANDOMIZATION STUDY

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Abstract ID: 22

Aim/ Background: The relationship between whole milk intake and stroke risk remains uncertain, partly because observational studies are susceptible to confounding and reverse causation. Trimethylamine N-oxide (TMAO), a gut microbiota-derived metabolite detectable in cerebrospinal fluid (CSF), may represent a diet-related pathway linking whole milk intake to cerebrovascular injury. This study evaluated the association between genetically predicted whole milk intake and stroke subtypes and explored whether CSF TMAO mediates this relationship.

Materials & Methods: We performed a two-sample Mendelian randomization analysis using genome-wide association study summary data for whole milk intake, CSF TMAO, and stroke outcomes. Univariable Mendelian randomization was used to assess associations with stroke subtypes, including intracerebral hemorrhage (ICH), subarachnoid hemorrhage, embolic stroke, embolic stroke related to atrial fibrillation, and all-cause stroke. Multivariable and two-step Mendelian randomization analyses were conducted to evaluate the potential mediating role of CSF TMAO.

Results: Genetically predicted whole milk intake was associated with increased ICH risk (OR = 1.196, 95% CI: 1.050–1.361, $P = 0.007$), but not with other stroke subtypes. After adjustment for whole milk intake, genetically predicted CSF TMAO was independently associated with ICH risk (OR = 1.043, 95% CI: 1.004–1.083, $P < 0.001$). Mediation analysis suggested that CSF TMAO accounted for approximately 23.6% of the total association. Sensitivity analyses showed no clear evidence of horizontal pleiotropy.

Conclusion: Genetically predicted whole milk intake was associated with increased ICH risk (OR = 1.196, 95% CI: 1.050–1.361, $P = 0.007$), but not with other stroke subtypes. After adjustment for whole milk intake, genetically predicted CSF TMAO was independently associated with ICH risk (OR = 1.043, 95% CI: 1.004–1.083, $P < 0.001$). Mediation analysis suggested that CSF TMAO accounted for approximately 23.6% of the total association. Sensitivity analyses showed no clear evidence of horizontal pleiotropy.

Keywords: Whole milk consumption, Intracerebral hemorrhage, Trimethyla

CCL20–CCR6-ASSOCIATED RECRUITMENT OF α -SYNUCLEIN-REACTIVE T CELLS PROMOTES NEUROINFLAMMATION IN PARKINSON'S DISEASE MODELS

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Abstract ID: 23

Aim/ Background: Adaptive immune responses to α -synuclein have been implicated in Parkinson's disease (PD), but the migratory phenotype and recruitment mechanisms of α -synuclein-reactive T cells remain incompletely defined. This study investigated whether the CCL20–CCR6 chemotactic axis contributes to their recruitment and neuroinflammatory effects in PD models.

Materials & Methods: α -Synuclein-reactive T cells were generated by co-culturing murine splenic T cells with α -synuclein-loaded, LPS/IFN- γ -matured DC2.4 cells. T-cell activation, cytokine profile, and CCR6 expression were assessed by flow cytometry. Transwell assays evaluated migration toward α -synuclein-stimulated BV2 microglial conditioned medium and recombinant CCL20. CCL20 expression in BV2 cells was measured by RT-qPCR and ELISA. A53T mice received adoptive transfer of α -synuclein-reactive T cells with or without CCR6 inhibition, followed by behavioral and immunofluorescence analyses.

Results: α -Synuclein-reactive T cells exhibited T-cell activation, an IL-17A-skewed inflammatory phenotype, increased CCR6 expression, and enhanced migration toward α -synuclein-stimulated BV2 conditioned medium and CCL20. α -Synuclein stimulation increased CCL20 expression and secretion in BV2 cells. In A53T mice, adoptive transfer of α -synuclein-reactive T cells was associated with increased brain-associated T-cell accumulation, enhanced microglial activation, reduced tyrosine hydroxylase-positive neurons in the substantia nigra, and aggravated motor impairment. CCR6 inhibition attenuated T-cell chemotaxis, brain-associated T-cell accumulation, neuroinflammation, and dopaminergic injury-related changes.

Conclusion: α -Synuclein-reactive T cells acquire an IL-17A/CCR6-associated inflammatory migratory phenotype. The CCL20–CCR6 axis may link adaptive immune activation to neuroinflammatory amplification in PD models.

Keywords: Parkinson's Disease, α -Synuclein-Reactive T Cells, CCL20–CCR

FOCAL CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY OF THE BRACHIAL PLEXUS: A CASE REPORT HIGHLIGHTING DIAGNOSTIC CHALLENGES IN RARE VARIANTS

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Abstract ID: 416

Aim/ Background: Focal CIDP is a rare variant characterized by motor and sensory dysfunction confined to a single limb and can pose significant diagnostic challenges due to its localized presentation and failure to meet conventional electrodiagnostic criteria.

Materials & Methods: A 33-year-old woman with hypothyroidism presented with progressive left upper limb weakness. Clinical examination, cerebrospinal fluid analysis, nerve conduction studies, and MR neurography were performed. Treatment response to intravenous methylprednisolone and subsequent intravenous immunoglobulin was assessed.

Results: The patient had a five-year history of wrist drop followed by a static phase and worsening proximal weakness over three months. Examination showed marked wasting, hypotonia, areflexia, and severe left upper limb weakness (MRC 1/5), with an INCAT disability score of 5. CSF was unremarkable. Nerve conduction studies showed multifocal demyelinating abnormalities with conduction block involving multiple nerves of the affected limb. MR neurography demonstrated diffuse thickening and T2/STIR hyperintensity of the left C5–C7 roots extending into the brachial plexus. Methylprednisolone produced minimal improvement, whereas intravenous immunoglobulin resulted in significant recovery, with power improving to 4/5 and INCAT score from 5 to 2.

Conclusion: Focal CIDP should be considered in chronic progressive monomelic neuropathy, even without sensory symptoms or classical CSF abnormalities. Electrodiagnostic evidence of demyelination, supportive MRI findings, and objective response to immunotherapy are important for diagnosis. Early recognition and treatment with intravenous immunoglobulin can result in substantial functional recovery.

Keywords:

REDUCTION IN CAREGIVER BURDEN FOLLOWING STRUCTURED TRANSITIONAL REHABILITATION: A LITERATURE-BASED REVIEW

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Abstract ID: 35

Aim/ Background: Caregiver burden is a significant challenge among patients recovering from neurological and disabling conditions such as stroke, traumatic brain injury, and spinal cord injury. Following hospital discharge, caregivers frequently experience psychological stress, physical exhaustion, social isolation, and financial strain. Transitional Care Centres (TCCs) provide structured multidisciplinary rehabilitation and caregiver training aimed at improving patient independence and continuity of care during the transition from hospital to home. Recent literature emphasizes the importance of caregiver-centered rehabilitation models in improving long-term outcomes. This study aim to evaluate the impact of structured transitional rehabilitation on caregiver burden and functional recovery based on current literature.

Materials & Methods: A literature-based review was conducted using studies published between 2020 and 2024 from PubMed, Scopus, and Google Scholar databases. Articles focusing on transitional rehabilitation, caregiver burden, neurorehabilitation, and continuity of care were reviewed. Studies assessing caregiver outcomes using validated scales such as the Zarit Burden Interview (ZBI) and patient functional outcomes using the Functional Independence Measure (FIM) were included.

Results: Current evidence demonstrates that structured transitional rehabilitation significantly reduces caregiver burden by improving caregiver preparedness, confidence, and psychosocial support. Multidisciplinary interventions including physiotherapy, occupational therapy, speech therapy, psychological counselling, and caregiver education were associated with improved patient functional independence and smoother community reintegration. Several studies also reported reduced hospital readmissions and enhanced quality of life for both patients and caregivers.

Conclusion: Recent literature supports the growing role of Transitional Care Centres in bridging acute hospitalization and home-based recovery. Caregiver-centered rehabilitation models contribute to reduced stress, improved continuity of care, and better functional outcomes. However, heterogeneity in rehabilitation protocols and outcome measures highlights the need for larger prospective studies evaluating long-term caregiver and patient outcomes.

Keywords: Transitional Neuro Care Centre; Caregiver Burden;

TWO ORGANS, ONE TRIGGER: A RARE CASE OF CONCURRENT TRALI AND PRES FOLLOWING PRBC TRANSFUSION

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Abstract ID: 15

Aim/ Background: Transfusion-related acute lung injury (TRALI) is a major cause of transfusion-associated mortality, whereas neurological complications after blood transfusion are rare and underrecognized. Posterior reversible encephalopathy syndrome (PRES) has occasionally been linked to transfusion, but concurrent occurrence with TRALI has rarely been described. We report a rare case of simultaneous TRALI type II and transfusion-related PRES following packed red blood cell (PRBC) transfusion.

Materials & Methods: A 65-year-old man with carcinoma ascending colon receiving adjuvant chemotherapy was admitted with severe sepsis, bilateral bronchopneumonia, chemotherapy-induced enteropathy, pancytopenia, and hypoalbuminemia. Following stabilization, he developed severe anemia (hemoglobin 6.9 g/dL) and received one unit PRBC transfusion. Clinical findings, laboratory investigations, chest imaging, echocardiography, MRI brain, and cerebrospinal fluid analysis were evaluated.

Results: Within three hours of transfusion, the patient developed acute hypoxemic respiratory failure with bilateral pulmonary infiltrates, hypotension, and tachycardia. Cardiac causes were excluded, establishing TRALI type II. Subsequently, he developed agitation, headache, visual disturbances, gaze deviation, and abnormal behavior. MRI brain revealed bilateral asymmetric T2/FLAIR hyperintensities with sulcal effacement and patchy diffusion restriction, consistent with PRES. Cerebrospinal fluid examination was normal. Supportive management resulted in marked clinical improvement, and follow-up MRI showed near-complete radiological resolution.

Conclusion: This case highlights a rare concurrence of TRALI and transfusion-related PRES, suggesting a shared mechanism involving systemic inflammation, endothelial dysfunction, and impaired autoregulation. Early recognition of neurological complications associated with transfusion reactions is crucial, as timely supportive care may lead to favorable outcomes.

Keywords: TRALI, PRES, Blood transfusion, Endothelial Dysfunction

CLINICAL PROFILE, AETIOLOGY AND OUTCOME OF LUMBOSACRAL RADICULOPLEXONEUROPATHY: A SINGLE-CENTER PROSPECTIVE CASE SERIES

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Abstract ID: 53

Aim/ Background: Lumbosacral radiculoplexoneuropathy (LS-RPN) is an uncommon but disabling disorder involving the lumbosacral roots, plexus, and peripheral nerves. It comprises a heterogeneous group of conditions with diverse etiologies, including diabetes mellitus, inflammatory neuropathies, vasculitis, malignancy, infections, and radiation injury. Data on its clinical profile and outcomes in Indian patients are limited.

Materials & Methods: This prospective observational case series included 32 consecutive patients with clinically suspected and electrophysiologically and/or radiologically confirmed LS-RPN enrolled at a tertiary-care neurology center over 18 months. All patients underwent detailed clinical evaluation, laboratory investigations, nerve conduction studies, electromyography (EMG), and magnetic resonance imaging (MRI). Functional outcomes were assessed at baseline, 3 months, and 6 months using the Medical Research Council (MRC) sum score, modified Rankin Scale (mRS), pain score, and ambulatory status.

Results: The mean age was 51.8 ± 13.6 years, with male predominance (68.8%). Pain (87.5%), weakness (93.8%), and sensory symptoms (65.6%) were common presentations, with unilateral onset in 75% of patients. Diabetes mellitus was the most frequent associated condition (43.8%). Etiologies included diabetic LS-RPN (37.5%), idiopathic inflammatory LS-RPN (25%), vasculitic neuropathy (12.5%), neoplastic plexopathy (9.4%), post-infectious LS-RPN (9.4%), and radiation-induced plexopathy (6.2%). Electrodiagnostic abnormalities were detected in 90.6%, while MRI abnormalities were present in 68.8%. At 6 months, 34.4% achieved near-complete recovery, 50% had partial recovery, and 15.6% showed minimal improvement. Severe weakness, extensive axonal loss, and neoplastic etiology predicted poorer outcomes.

Conclusion: LS-RPN is a heterogeneous disorder in which diabetes is the leading cause. Electrodiagnostic studies remain central to diagnosis, while MRI provides complementary information. Despite overall improvement, residual disability is common, emphasizing the importance of early diagnosis and etiology-directed management.

Keywords: Lumbosacral radiculoplexoneuropathy, diabetic amyotrophy, lu

POST STROKE RECRUDESCENCE

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Christian Medical College, Ludhiana,

Abstract ID: 90

Aim/ Background: Post-stroke recrudescence (PSR): transient worsening of the neurological deficits, associated with an old stroke, without evidence of new stroke on neuro-imaging.

An under-recognized, under—reported and poorly characterized phenomenon.

May be falsely attributed to seizure phenomenon, and sometimes patients are started on anti-seizure medications.

Aim:

To describe 2 patients, who presented with acute worsening of old neurological deficits, without any neuroimaging evidence of an acute stroke, diagnosed to have PSR.

Materials & Methods:

70/M,

Large RT MCA infarct 1 year back.

Presented with acute worsening of his left hemiparesis with difficulty in swallowing and drooling of saliva.

MRI: chronic right cerebral infarct, without any diffusion restriction.

Urine culture grew *P. aeruginosa*. Other investigations normal.

56/M , LT MCA infarct 18 months back.

Presented with sudden worsening of speech(motor aphasia) and right hemiparesis.

MRI brain and CT brain showed chronic left MCA infarct, without new lesion.

Blood and urine investigations were normal.

Further evaluation revealed maxillary sinusitis, for which he was started on antibiotics.

Results:

After antibiotics, reported symptomatic improvement within 3 days of hospital admission.

Conclusion: Distinguishing PSR from similar conditions such as seizure phenomenon, TIA , migraines, amyloid spells, or functional disorders is crucial due to the distinct therapeutic, prognostic implications involved.

MRI with DWI is essential especially for patients within the window period, to decide thrombolytic and re-perfusion therapy.

Additionally, EEG (Electroencephalogram) is necessary to exclude the possibility of a seizure phenomenon.

Keywords: Post-stroke recrudescence; Old stroke; Stroke mimic

OUTCOME AND PREDICTORS OF CARDIOPULMONARY RESUSCITATION AMONG PATIENTS ADMITTED IN INTENSIVE CARE UNIT OF A TERTIARY CARE HOSPITAL

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Abstract ID: 110

Aim/ Background:

CPR outcomes and predictors in Indian ICU patients remain poorly characterised, as most existing evidence derives from Western populations with differing comorbidity profiles and resources. This study aimed to determine the outcome of CPR among ICU patients and identify predictors of return of spontaneous circulation (ROSC).

Materials & Methods:

A one-year prospective observational study was conducted in the Medical and Pulmonary ICUs of a tertiary care hospital. Patients aged >18 years undergoing CPR as per 2020 AHA BLS/ACLS guidelines were enrolled, and demographic, clinical and biochemical parameters were compared between ROSC-positive and ROSC-negative patients.

Results:

Of 132 patients who underwent CPR, 28 (21.2%) achieved ROSC while 119 (90.2%) expired. Most were male (70.5%) and aged over 45 years (65.9%). Infections were the commonest diagnosis (97%), predominantly respiratory (75%) and urinary tract (41.6%). Hypertension (40.2%), diabetes (32.6%), cardiac disease (24.2%) and AKI (20.4%) were frequent, with AKI significantly more common among ROSC-positive patients. pH (admission and arrest), potassium and bicarbonate at arrest, and ESR were significantly higher among ROSC-positive patients. Shorter ICU stay and CPR duration correlated with ROSC.

Conclusion:

CPR outcomes in the ICU remain poor, with 90.2% mortality. ROSC was more frequent among older males with infectious and renal involvement, and was associated with pH, potassium, bicarbonate and ESR. Early recognition, correction of reversible causes, and targeted interventions, particularly infection control and renal monitoring, may improve survival in high-risk ICU patients undergoing CPR

Keywords: CPR, Cardiac arrest, ROSC, ICU

MENTAL CALCULATION RELATED REFLEX EPILEPSY- A CASE OF EPILEPSIA ARITHMETICES

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Abstract ID: 47

Aim/ Background: REFLEX EPILEPSIES ARE SEIZURE DISORDERS CONSISTENTLY TRIGGERED BY SPECIFIC STIMULI, AFFECTING 4–7% OF EPILEPSY PATIENTS AND UP TO 21% OF THOSE WITH IDIOPATHIC GENERALIZED EPILEPSY (IGE). COMPLEX COGNITIVE TASKS, PARTICULARLY MENTAL CALCULATION (EPILEPSIA ARITHMETICES), ARE RARE BUT RECOGNIZED TRIGGERS OF GENERALIZED SEIZURES, TYPICALLY MANIFESTING AS MYOCLONIC JERKS OR TONIC–CLONIC CONVULSIONS, MOST OFTEN WITHIN THE JUVENILE MYOCLONIC EPILEPSY SPECTRUM.

Materials & Methods: A 22-YEAR-OLD WOMAN HAD AN 8-YEAR HISTORY OF GENERALIZED TONIC–CLONIC SEIZURES PREDOMINANTLY PROVOKED BY SOLVING MATH PROBLEMS. SEIZURES BEGAN WITH AN AURA OF UNEASINESS AND TACHYCARDIA, WITH LEFT-HAND POSTURING AND HEAD/EYE DEVIATION TO THE LEFT, FOLLOWED BY BRIEF BILATERAL CLONIC JERKS (<1 MIN) AND POSTICTAL CONFUSION WITH CRYING. FREQUENCY WAS 1–2/MONTH, OFTEN DURING INTENSIVE MATHEMATICAL WORK. NEUROLOGIC EXAMINATION WAS NORMAL. PRIOR MRI AND ROUTINE EEG WERE NONDIAGNOSTIC. CURRENT EPILEPSY-PROTOCOL MRI SHOWED AN ENLARGED PITUITARY (POSSIBLE RATHKE’S CLEFT CYST) WITHOUT CORTICAL LESIONS. VIDEO-EEG CAPTURED GENERALIZED 3 HZ SPIKE-WAVE DISCHARGES PRECIPITATED BY MENTAL ARITHMETIC TASKS. THESE FINDINGS SUPPORTED A DIAGNOSIS OF PRIMARY GENERALIZED EPILEPSY WITH A REFLEX (COGNITIVE) COMPONENT.

Results: THE PATIENT CONTINUED ANTISEIZURE MEDICATIONS (LACOSAMIDE, CLOBAZAM, PERAMPANEL) AND WAS COUNSELED. ON FOLLOW-UP, SHE REMAINED SEIZURE-FREE, WITH SOME MINOR EPISODES TRIGGERED BY MENSTRUATION.

Conclusion: THIS CASE ILLUSTRATES EPILEPSIA ARITHMETICA, A REFLEX EPILEPSY WHERE COMPLEX CALCULATION TRIGGERS GENERALIZED SEIZURES. THE MECHANISM INVOLVES PARIETAL COGNITIVE NETWORKS PRODUCING GENERALIZED DISCHARGES. MANAGEMENT PARALLELS STANDARD TREATMENT OF GENERALIZED EPILEPSY, COMBINING APPROPRIATE ANTISEIZURE MEDICATION WITH AVOIDANCE OR MITIGATION OF KNOWN TRIGGERS. TASK-BASED EEG CAN GUIDE TARGETED EVALUATION AND IMPROVE PATIENT CARE.

Keywords: REFLEX EPILEPSY, EPILEPSIA ARITHMETICES.

TITLE: THE SILENT SPINE: UNDERDIAGNOSED SPINAL CORD INVOLVEMENT IN ATYPICAL POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) PRESENTING WITH CLINICAL-RADIOLOGICAL DISSOCIATION

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Abstract ID: 155

Aim/ Background: Posterior Reversible Encephalopathy Syndrome (PRES) is a clinico-radiological entity characterized by reversible vasogenic edema, classically involving the parieto-occipital regions. Spinal cord involvement is an exceptionally rare manifestation and may remain clinically silent. We report an atypical case of PRES with extensive brainstem and cervical spinal cord involvement demonstrating marked clinical-radiological dissociation.

Materials & Methods: A 43-year-old hypertensive male presented with acute blurring of vision and headache during hypertensive emergency (BP 210/110 mmHg). Neurological examination revealed no focal deficits or signs of myelopathy. MRI brain and cervical spine, cerebrospinal fluid analysis, and laboratory investigations were performed. The patient was managed conservatively with aggressive blood pressure control and supportive therapy.

Results: MRI demonstrated confluent T2/FLAIR hyperintensities involving the basal ganglia, thalami, brainstem, cerebellar peduncles, and a longitudinal non-enhancing T2 hyperintense lesion extending from C3 to C7, consistent with vasogenic edema. Despite extensive spinal cord involvement, the patient had no motor, sensory, autonomic, or pyramidal deficits. Clinical stabilization was achieved following antihypertensive therapy without neurological deterioration.

Conclusion: This case highlights the rare phenomenon of clinically silent spinal cord involvement in atypical PRES. Recognition of PRES-SCI is crucial to avoid misdiagnosis as inflammatory or demyelinating myelopathy and unnecessary investigations. Cervical spine imaging should be considered in patients with brainstem-predominant PRES, even in the absence of myelopathic signs.

Keywords: Posterior Reversible Encephalopathy Syndrome; PRES

A BRAIN ASLEEP, A BODY AWAKE: A CASE OF COMPLEX SLEEP BEHAVIOURS

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Abstract ID: 2

Aim/ Background: Zolpidem, a non-benzodiazepine benzodiazepine receptor agonist (Z-drug), is widely prescribed for the short-term treatment of insomnia because of its rapid onset of action and perceived safety compared with benzodiazepines. However, increasing evidence suggests that zolpidem may cause complex sleep behaviours (CSBs), including sleepwalking, sleep-related eating, sleep driving, and other automatic behaviours performed without subsequent recall. Recognition of this uncommon but potentially hazardous adverse effect remains limited, leading to frequent misdiagnosis.

Materials & Methods: A 55-year-old woman with hypertension, hypothyroidism, and chronic kidney disease on maintenance haemodialysis was prescribed zolpidem 5 mg, later increased to 10 mg, for insomnia. Approximately 4-5 months after initiation, she developed recurrent nocturnal episodes of sleepwalking, sleep-related eating, wandering outside the house, attempting to cook during the night, and one episode resulting in a fall. During these episodes, she appeared awake, became aggressive when interrupted, and had complete amnesia the following day while maintaining normal daytime occupational functioning. She was initially diagnosed elsewhere with REM sleep behaviour disorder, restless legs syndrome, and depression, and was treated with clonazepam, melatonin, pramipexole, and amitriptyline while zolpidem was continued, resulting in worsening symptoms. Following neurological evaluation, a detailed medication review suggested zolpidem-induced NREM parasomnia. Brain MRI demonstrated only a small chronic right gangliocapsular infarct, while EEG and cerebrospinal fluid examination were normal. Zolpidem was discontinued.

Results: At one-month follow-up, all abnormal nocturnal behaviours had completely resolved without recurrence.

Conclusion: This case highlights zolpidem as an important reversible cause of complex sleep behaviours that may mimic primary parasomnias or psychiatric disorders. Careful medication history is essential in patients presenting with new-onset nocturnal behaviours. Prompt recognition and withdrawal of zolpidem can result in complete clinical recovery and prevent potentially serious injuries.

Keywords: parasomnia, sleep,

AN UNUSUAL PRESENTATION OF MOG-IGG AND NEUROFASCIN-186 ANTIBODY-ASSOCIATED CENTRAL-PERIPHERAL DEMYELINATION

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Abstract ID: 82

Aim/ Background: Combined central and peripheral demyelination (CCPD) is a rare immune-mediated disorder characterized by simultaneous involvement of the central and peripheral nervous systems. We report an atypical antibody-associated demyelinating syndrome presenting as acute post-febrile lumbosacral plexopathy followed by delayed contralateral upper motor neuron involvement.

Materials & Methods: A previously healthy adult developed high-grade fever followed by acute flaccid right lower limb weakness. Two weeks later, progressive stiffness with extensor spasms developed in the left lower limb. Evaluation included nerve conduction studies, electromyography, MRI brain, spine and lumbosacral plexus, cerebrospinal fluid analysis, visual evoked potentials, and autoimmune antibody testing.

Results: Nerve conduction studies and electromyography localized the lesion to the right lumbosacral plexus with active denervation. MRI demonstrated thickening and T2 hyperintensity of the right lumbosacral plexus with denervation changes in the right gluteal muscles, without brain, optic nerve or spinal cord demyelinating lesions. CSF and infectious work-up were unremarkable. VEP performed twice demonstrated bilateral prolonged latencies. Serum anti-MOG IgG was mildly positive on a cell-based indirect immunofluorescence assay, while anti-neurofascin-186 antibody was elevated (798; reference 0–366). Treatment with intravenous methylprednisolone, rituximab, oral corticosteroids and rehabilitation resulted in improvement of spasticity, although right lower limb weakness persisted.

Conclusion: This case highlights an unusual post-infectious neurological syndrome with objective evidence of peripheral demyelination and subclinical central pathway involvement supported by persistent VEP abnormalities and MOG-IgG positivity. The coexistence of MOG-IgG and neurofascin-186 antibodies broadens the evolving spectrum of antibody-associated central-peripheral demyelinating disorders and emphasizes the need to consider immune-mediated overlap syndromes in atypical post-febrile monoparesis.

Keywords: MOG-IgG, Neurofascin-186, Lumbosacral Plexopathy

ANTIBODY-NEGATIVE AUTOIMMUNE ENCEPHALITIS WITH SUPER-REFRACTORY STATUS EPILEPTICUS

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Abstract ID: 18

Aim/ Background: Limbic encephalitis is an inflammatory disorder of the medial temporal lobes presenting with seizures and cognitive dysfunction. Differentiating autoimmune from infectious causes is challenging, especially in antibody-negative cases.

Materials & Methods: A 23-year-old female presented with recurrent seizures progressing to super-refractory status epilepticus and altered sensorium. MRI brain showed bilateral medial temporal lobe FLAIR hyperintensities involving hippocampal and parahippocampal regions with diffusion restriction and leptomeningeal enhancement. HHH serology was negative. EEG demonstrated extreme delta brush pattern.

CSF revealed mild lymphocytic pleocytosis (5 cells/mm³), elevated protein (70 mg/dL), normal glucose, low ADA, and negative Gram stain, culture, TB-PCR, and malignant cytology. Autoimmune encephalitis panel was negative. Contrast-enhanced CT showed bilateral lower lobe ground-glass opacities, ascites, omento-mesenteric fat stranding, and hepatic lesions suggestive of possible granulomatous/infective etiology. Ovarian teratoma was excluded.

Results: Management and Outcome:

She required anesthetic infusions (midazolam, ketamine, thiopentone) and multiple antiseizure medications (levetiracetam, phenytoin, lacosamide, clobazam, topiramate, perampanel) without seizure control. First-line immunotherapy with intravenous methylprednisolone and IVIG (100 g) was ineffective. Subsequent treatment with two doses of rituximab resulted in cessation of seizures and improvement in sensorium, allowing gradual tapering of antiseizure medications.

Conclusion: This case highlights the diagnostic dilemma in antibody-negative limbic encephalitis with overlapping infective features. Early escalation to second-line immunotherapy such as rituximab may be crucial in refractory cases.

Keywords: Autoimmune, Encephalitis, status epilepticus

PULSELESS DISEASE UNMASKED AS STROKE: A CASE OF TAKAYASU ARTERITIS PRESENTING WITH ACUTE ISCHEMIC STROKE

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Abstract ID: 19

Aim/ Background: Takayasu arteritis is a chronic inflammatory disease involving the aorta and its major branches, predominantly affecting young women. Neurological manifestations are uncommon but may include ischemic stroke due to progressive vascular stenosis or occlusion.

Materials & Methods: A 36-year-old female presented with headache for 7–8 months, followed by acute onset left upper limb weakness and slurring of speech for 3 days. Headache was intermittent, initially right-sided, later diffuse, and associated with episodes of blurring of vision.

She was a known case of hypothyroidism with no history of hypertension, diabetes, or tuberculosis. Menstrual history revealed irregular cycles with menorrhagia. Obstetric history was significant for two spontaneous abortions.

On examination, pulse was absent and blood pressure was non-recordable in the left upper limb. Neurological examination showed left-sided weakness.

Results: The patient was managed with low molecular weight heparin and dual antiplatelet therapy for acute stroke. Immunosuppressive therapy was initiated with intravenous methylprednisolone followed by oral steroids and mycophenolate mofetil. Rheumatology consultation was sought for further management.

Conclusion: This case highlights an uncommon presentation of Takayasu arteritis as acute ischemic stroke in a young female. Clinical clues such as pulse and blood pressure discrepancy along with vascular imaging are critical for early diagnosis. Prompt immunosuppressive therapy is essential to prevent disease progression and recurrent cerebrovascular events.

Keywords: vasculitis, takayasu arteritis, stroke

TITLE: THE RED EYES THAT WEREN'T INFLAMMATORY: AN ELUSIVE INDIRECT CAROTID–CAVERNOUS FISTULA PRESENTING WITH BILATERAL OCULAR CONGESTION

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Abstract ID: 154

Aim/ Background: Indirect carotid–cavernous fistulas are low-flow dural arteriovenous shunts that may present with subtle and atypical manifestations, often mimicking inflammatory ophthalmic disorders. We report a case of unilateral indirect CCF presenting with bilateral red eyes, resulting in delayed diagnosis.

Materials & Methods: A 53-year-old postmenopausal woman presented with bilateral ocular redness for six months, associated with headache, progressive visual impairment, and mild proptosis. She had initially received treatment for allergic conjunctivitis and diffuse anterior scleritis without improvement. Clinical examination, neuroimaging, and digital subtraction angiography (DSA) findings were reviewed.

Results: MRI orbit with CT angiography and venography demonstrated bilateral dilatation of the superior ophthalmic veins without evidence of thrombosis, aneurysm, or arteriovenous malformation. DSA revealed a right-sided indirect low-flow carotid–cavernous fistula supplied by meningeal branches of the cavernous segment of the right internal carotid artery and dural branches of the right middle meningeal artery. Venous drainage occurred into the right cavernous sinus with cross-flow through intercavernous venous channels to the contralateral cavernous sinus, resulting in bilateral superior ophthalmic vein engorgement and bilateral conjunctival congestion.

Conclusion: Indirect CCFs are frequently misdiagnosed because of their indolent presentation. Bilateral ocular manifestations arising from a unilateral fistula are uncommon and occur due to transmission of elevated venous pressure through intercavernous venous communications. Recognition of this mechanism is essential to avoid diagnostic delay and prevent visual morbidity.

Conclusion : Persistent bilateral red eyes, particularly when associated with headache, proptosis, or visual symptoms, prompts evaluation for underlying vascular etiology. DSA remains the gold standard for diagnosis of indirect CCF and facilitates timely management.

Keywords: DSA, CCF, Proptosis

ONE GENE, THREE JOURNEYS: A CASE SERIES OF DOPA-RESPONSIVE DYSTONIA.

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Lokmanya Tilak Municipal Medical College and Lokmanya Tilak Municipal General Hospital, Mumbai, Maharashtra

Abstract ID: 412

Aim/ Background:

Background: Dopa-responsive dystonia (DRD) is an uncommon yet highly treatable movement disorder that is frequently under-recognized due to its variable clinical presentation. Early diagnosis is essential, as patients may show a dramatic and sustained response to low-dose levodopa.

Objective: To describe the clinical spectrum, diagnostic challenges, and treatment response in three patients with DRD, including two affected siblings.

Materials & Methods: We present a retrospective case series of three patients diagnosed with DRD at a tertiary neurology center. Clinical characteristics, family history, investigations, neuroimaging, levodopa responsiveness, and follow-up outcomes were reviewed.

Results: Three patients demonstrated variable phenotypic presentations of DRD. Two were siblings, highlighting the familial nature of the disorder, while the third appeared sporadic. Clinical manifestations included childhood- or adolescent-onset dystonia with gait impairment and variable diurnal fluctuation. Diagnostic evaluation was often extensive, contributing to delayed recognition. All three patients showed sustained improvement following initiation of low-dose levodopa, with significant functional recovery and minimal adverse effects.

Conclusion: DRD should be considered in patients with early-onset dystonia, particularly when gait disturbance, diurnal variation, or a positive family history is present. Recognition of this treatable disorder can prevent diagnostic delay, reduce disability, and substantially improve quality of life.

Keywords:

CORRELATION BETWEEN EEG LOCALIZATION AND PERCEIVED STIGMA IN PATIENTS WITH EPILEPSY: A CLINICAL OBSERVATIONAL STUDY

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Abstract ID: 48

Aim/ Background: Epilepsy is a chronic neurological disorder often associated with social and psychological stigma. The perception of stigma may vary depending on seizure type, behavioral manifestations, and electroencephalographic (EEG) localization. Temporal lobe epilepsy is frequently associated with emotional and behavioural symptoms that may contribute to increased social stigma.

Materials & Methods: Epilepsy is a chronic neurological disorder often associated with social and psychological stigma. The perception of stigma may vary depending on seizure type, behavioral manifestations, and electroencephalographic (EEG) localization. Temporal lobe epilepsy is frequently associated with emotional and behavioral symptoms that may contribute to increased social stigma.

Results: Among the 50 patients evaluated, a higher level of perceived stigma was observed in patients with temporal lobe epilepsy compared to those with frontal lobe involvement. Patients with temporal lobe epileptiform discharges reported greater emotional distress, social embarrassment, and fear of discrimination. Frontal lobe epilepsy patients demonstrated relatively lower stigma perception. The findings suggest a possible relationship between EEG localization and psychosocial impact in epilepsy.

Conclusion: Temporal lobe epilepsy appears to be associated with greater perceived stigma compared to frontal lobe epilepsy. EEG localization may therefore play an important role not only in diagnosis and treatment planning but also in understanding psychosocial outcomes in epilepsy patients. Further large-scale studies are recommended to validate these findings and improve holistic epilepsy care.

Keywords: Epilepsy, EEG, stigma, temporal lobe, epilepsy,

NOVEL CYTOKINE RATIOS AS DIAGNOSTIC MARKERS FOR PRIMARY CNS LYMPHOMA

Dr. Sofia Singh, Dr. Aastha Takkar Kapila, Dr. Karthik Vinay Mahesh, Prof. Gaurav Prakash, Prof. Vivek Lal

PGIMER, Chandigarh

Abstract ID: 124

Aim/ Background: The aggressive extranodal diffuse large B-cell lymphoma of the CNS, primary CNS lymphoma (PCNSL), accounts for 3–4% of primary brain cancers. Delays in diagnosis are common due to radiologic overlap with gliomas and demyelinating diseases. Stereotactic brain biopsy is the gold standard, which is highly invasive and corticosteroid-sensitive. Hence, minimally invasive, biological diagnostics are needed. This study investigated PCNSL's unique multiparametric CSF cytokine–chemokine profile and its diagnostic potential.

Materials & Methods: This prospective case-control study at PGIMER, Chandigarh, funded by DST SERB, included 160 participants in four groups (n=40 each): biopsy proven PCNSL, CNS inflammatory diseases, other CNS malignancies, and non-neurological controls. CSF and serum cytokines (IL-10, IL-6, IL-8, IL-17A, IL-9, TNF- α) were measured following lumbar puncture. ROC and Kruskal-Wallis with Dunn's correction were used for diagnostic accuracy and statistical analysis.

Results: CSF IL-10 levels were substantially higher in PCNSL patients (median 55.86 pg/mL) than controls. The most reliable individual cytokine-derived marker was the IL-10:IL-6 ratio with 85% sensitivity and 92.5% specificity, and the novel IL-10:IL-8 ratio with 82.5% sensitivity and 95% specificity. Serum levels, however, overlapped across the study groups.

Conclusion: CSF IL-10 was markedly elevated in PCNSL, but ratio-based markers outperformed IL-10 alone. Most notably, the novel IL-10:IL-8 ratio achieved the highest specificity (95%) with strong sensitivity (82.5%), suggesting IL-8 co-elevation may better isolate the PCNSL-specific signal than IL-6 and identifying this ratio as a new candidate biomarker for further study. Serum cytokines showed no discriminatory value, confirming the diagnostic signal is CSF-compartmentalized. These findings support CSF-based, steroid-independent testing as a minimally invasive alternative to biopsy pending external validation.

Keywords: Primary CNS lymphoma, Interleukin, diagnostic biomarker, CSF

NEUROMYOTONIA : A GREAT MASQUEDER OF MND

Dr. Sonali Paul

Tirupati, Andhra Pradesh

Abstract ID: 123

Aim/ Background: 68-year-old male, no prior comorbidities, presented with a history of generalized body ache, with more pain over bilateral soles and the dorsum of the foot for the last three months. He had unexplained weight loss of around 8 kg for the last three months. He also had a significant history of thinning of muscles over bilateral upper limbs, lower limbs, and diffuse wasting of muscles. He had twitching of muscles over bilateral upper limbs, thigh, trunk, and sometimes the abdomen for the last two and a half months.

He came to us for further evaluation and treatment.

Materials & Methods: -

Results: On examination, we noticed he had:

- Tone in MAS 1+ in bilateral upper limbs and 2+ in bilateral lower limbs
- DTR being 3+ in both upper limbs and 2+ in both lower limbs
- Fasciculations were predominant over bilateral triceps, trunk, pectoralis, and also in bilateral lower limbs, in the vastus medialis, soleus, gastrocnemius, and also at tibialis anterior muscle
- Sensory was normal, but he was complaining more of neuropathic pain over bilateral soles, not localizing to any kind of dermatome

We had a suspicion of MND in view of the fasciculations, and the EMG finding was very significant for myokymia . we sent a serum autoimmune panel showed CASPAR 2 positive, with Pet-CT suggestive of an anterior mediastinal mass. This raised the suspicion of Neuromyotonia in view of the EMG findings showing myokymia The background of CASPAR 2 and an anterior mediastinal mass again correlated with the underlying Neuromyotonia.

We started plasmapheresis for this patient, and he showed significant improvement. Thus, the conclusion is that Neuromyotonia is a great masquerader for motor neuron disease and treatment changes

Conclusion: Fasciculations with twitching and wasting disproportionate to weakness

With Emg findings of myokymia predominant always suspicion of neuromyotonia

Keywords: Neuromyotonia

ANODAL TDCS OVER THE LEFT DLPFC AND ITS PRELIMINARY EFFECT ON MEMORY IN ALZHEIMER'S DEMENTIA: A SINGLE CASE OBSERVATION

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Abstract ID: 41

Aim/ Background: Alzheimer's disease is a major public health concern in India. In this setting, MoCA-Hindi, MMSE, and bedside MSE help assess cognition across diverse educational and language backgrounds. tDCS is a safe, non-invasive neuromodulatory technique, and anodal stimulation of the left DLPFC may support memory processing in AD.

Materials & Methods: To preliminarily observe and document the effect of a standardised 10-session anodal tDCS protocol targeting the left DLPFC on memory and cognitive function in a single case of mild Alzheimer's Dementia in the North Indian clinical context, assessed through MoCA-Hindi, MMSE, and structured bedside MSE pre- and post-intervention.

Results: A 63-year old right-handed female homemaker from Ghaziabad, Uttar Pradesh, with 10 years of education, was diagnosed with mild Alzheimer's dementia per ICD-11 and was on stable donepezil 10 mg/day with limited benefit. Baseline MSE showed psychomotor slowing, anomie speech, impaired orientation, absent delayed recall, partial insight, and impaired judgement. Baseline MoCA-Hindi was 18/30 and MMSE was 20/30. She received 10 weekday sessions of anodal tDCS over F3 (left DLPFC) at 2 mA for 20 minutes per session, with donepezil continued unchanged.

Conclusion: Post-intervention at session 10, the patient showed improvement on MoCA-Hindi, MMSE, and bedside MSE, with better recall, orientation, speech spontaneity, and affect. The intervention was well tolerated, suggesting that anodal tDCS over the left DLPFC may be a useful adjunct for memory improvement in mild Alzheimer's Dementia.

Keywords: tDCS, Alzheimer's, MoCA, Neuromodulation, Intelligence

IMMEDIATE EARLY GENE DEPENDENT MODULATION OF NEUROPLASTICITY IN THE VENTROMEDIAL HYPOTHALAMUS

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Abstract ID: 84

Aim/ Background: Experience dependent plasticity enables innate behavioural circuits to adapt to prior social encounters. The ventromedial hypothalamus (VMH) undergoes functional remapping following social defeat, yet the molecular mechanisms linking neuronal activity to long-term circuit reorganization remain poorly understood. We investigated whether Serum Response Factor (SRF), a master regulator of immediate-early gene transcription, mediates experience-dependent plasticity in glutamatergic and GABAergic VMHvl neurons. Broadly, this is necessary for uncovering the neural basis of maladaptive states such as social anxiety, depression, and PTSD.

Materials & Methods: Conditional Srf mice were subjected to a resident-intruder social defeat paradigm. SRF was selectively deleted from Vglut2⁺ or Vgat⁺ VMHvl neurons using an intersectional viral strategy with stereotactic AAV delivery. Defensive behaviours, including social investigation, shelter occupancy, flight, and risk assessment, were quantified before and after defeat. To determine whether SRF-dependent transcription influences circuit function, optogenetic activation of VMHvl neurons was performed following social defeat.

Results: Social defeat induced robust and persistent defensive adaptations, including reduced social investigation and increased shelter occupancy, flight, and risk assessment. SRF deletion in glutamatergic VMH neurons did not significantly alter defeat-induced behavioural adaptations, suggesting that excitatory neuron SRF is not essential for behavioural expression. In contrast, optogenetic activation of SRF deficient GABAergic VMHvl neurons revealed significant alterations in experience dependent behavioural responses, identifying an essential role for inhibitory circuit plasticity in mediating defeat-induced behavioural modulation.

Conclusion: Our findings reveal a cell type specific requirement for SRF dependent transcription in hypothalamic plasticity. While excitatory VMHvl neurons retain the capacity to support defeat-induced defensive behaviour in the absence of SRF, inhibitory GABAergic neurons are required for experience dependent modulation of VMHvl circuit output. These results identify inhibitory transcriptional plasticity as a key mechanism by which social experience reshapes innate defensive circuits and provide new insight into molecular pathways underlying adaptive responses to social stress.

Keywords: Neuroplasticity, Hypothalamus, Optogenetics, Transcription

TO READ OR NOT TO READ

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Abstract ID: 149

Aim/ Background: Transient isolated alexia is a rare higher cortical dysfunction, most commonly described with dominant posterior circulation infarction. Its occurrence as the sole manifestation of transient cerebral ischemia is uncommon and may delay diagnosis. We report a patient with recurrent transient alexia secondary to intracranial vasculopathy.

Materials & Methods: In hospital case.

Results: A 43-year-old right-handed gentleman presented with sudden-onset inability to read. He had difficulty identifying letters and comprehending written words despite preserved vision, speech, and consciousness. The first episode lasted approximately 20 minutes with complete recovery. Three months later, he experienced a similar 10-minute episode associated with jumbling of words and selective difficulty recognising letters such as b, d, and j. MRI brain, including diffusion-weighted imaging, showed no acute or chronic infarction. MR angiography demonstrated irregular narrowing of the left M2 segment with multiple collateral vessels. Digital subtraction angiography was suggestive of Moyamoya disease. An extensive work-up, including vasculitis profile, ANA, HPLC, peripheral blood smear, and infective markers, was unremarkable. He was started on antiplatelet therapy.

Conclusion: Reading depends on an extensive dominant hemispheric network involving the occipitotemporal cortex and its connections. Transient ischemia affecting this network can produce isolated alexia even in the absence of diffusion restriction. Although migraine aura and focal seizures remain important differentials, recurrent stereotyped episodes with angiographic evidence of intracranial vasculopathy strongly support a vascular etiology. This case highlights that isolated transient alexia may represent a TIA equivalent and should prompt urgent vascular imaging and institution of secondary stroke prevention, even when conventional MRI is normal.

Keywords: Alexia, Moya-moya disease

PREDICTORS OF SYMPTOMATIC INTRACEREBRAL HAEMORRHAGE FOLLOWING REPERFUSION THERAPY IN ACUTE ISCHEMIC STROKE: A PROSPECTIVE OBSERVATIONAL STUDY

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Abstract ID: 38

Aim/ Background: Symptomatic intracerebral hemorrhage (sICH) remains a devastating complication following reperfusion therapy in acute ischemic stroke (AIS), adversely affecting functional outcomes and mortality. Early identification of high-risk patients may improve treatment selection and safety.

Materials & Methods: This prospective observational study included 250 AIS patients undergoing reperfusion therapy at Apollo BGS Hospitals, Mysore, between July 2022 and July 2024. Patients received IVT (n=165), MT (n=58), or IVT+MT (n=27). Clinical variables, imaging parameters, treatment timelines, and outcomes were analyzed. A cumulative risk-factor score was assigned based on the number of predefined risk factors. sICH was defined according to SITS-MOST criteria. Multivariate logistic regression identified independent predictors of sICH.

Results: sICH occurred in 8.4% (n=21), while 14.8% (n=37) developed asymptomatic ICH. Significant predictors of sICH included NIHSS >20 ($p<0.001$), systolic BP >145 mmHg ($p<0.001$), hyperglycemia >180 mg/dL ($p=0.0356$), prior dual antiplatelet use ($p=0.0084$), anticoagulant therapy ($p=0.0087$), and ASPECTS <6 ($p<0.001$). Delayed reperfusion (IVT >3 hours; MT >4.5 hours), >5 total risk factors, risk-factor score >8, and combined IVT+MT were also significantly associated with sICH. sICH was linked to significantly higher in-hospital mortality ($p<0.001$).

Conclusion: A multidimensional risk-stratification model incorporating clinical, imaging, and treatment variables may help predict sICH and optimize reperfusion strategies in AIS.

Keywords: • sICH • IV Thrombolysis • Mechanical Thrombectomy

AUTOIMMUNE GFAP ASTROCYTOPATHY – AN UNDER-RECOGNISED CAUSE OF FULMINANT NEUROINFLAMMATION AND A CHAMELEON IN NEUROLOGICAL PRACTICE

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Abstract ID: 39

Aim/ Background: Autoimmune glial fibrillary acidic protein (GFAP) astrocytopathy is a recently recognized autoimmune neuroinflammatory disorder characterized by GFAP-IgG positivity in cerebrospinal fluid (CSF). Owing to its diverse clinical and radiological manifestations, it is frequently misdiagnosed as infectious, paraneoplastic, or other autoimmune CNS disorders.

Materials & Methods: A 32-year-old male presented with fever, headache, vomiting, progressive paraparesis, urinary retention, and altered sensorium. MRI revealed longitudinally extensive transverse myelitis (LETM) with leptomeningeal enhancement suggestive of inflammatory myeloencephalitis. CSF analysis demonstrated lymphocytic pleocytosis, elevated protein, and GFAP-IgG positivity, confirming autoimmune GFAP astrocytopathy. Despite empirical antimicrobial and anti-tubercular therapy, neurological deterioration necessitated mechanical ventilation. Following diagnosis, the patient received high-dose intravenous methylprednisolone followed by rituximab, resulting in significant neurological recovery with improvement in motor and autonomic function.

Results: GFAP astrocytopathy is an important differential diagnosis in unexplained neuroinflammatory syndromes presenting with encephalitis, myelitis, autonomic dysfunction, or psychiatric manifestations. Characteristic MRI findings, including radial perivascular enhancement and longitudinal spinal cord lesions, provide crucial diagnostic clues. Early recognition and prompt immunotherapy are associated with favorable outcomes, while delayed diagnosis may lead to severe neurological disability.

Conclusion: GFAP astrocytopathy should be considered in patients with atypical meningoencephalomyelitis or LETM unresponsive to conventional antimicrobial therapy. Early antibody testing and aggressive immunotherapy can significantly improve clinical outcomes and reduce long-term neurological sequelae

Keywords: GFAP astrocytopathy, LETM , rituximab, neuroinflammation.

COMPLETE AND RAPID RECOVERY IN A CASE OF FULMINANT CHILDHOOD NMDAR ENCEPHALITIS WITH PARANEOPLASTIC AETIOLOGY

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Abstract ID: 40

Aim/ Background: Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis is the most common autoimmune encephalitis in children and may rapidly progress to severe neuropsychiatric dysfunction, refractory seizures, autonomic instability, and respiratory failure. Early recognition of underlying paraneoplastic etiology is crucial for optimal outcomes.

Materials & Methods: A previously healthy 14-year-old girl presented with acute psychosis, behavioral disturbances, hallucinations, and agitation, followed by generalized tonic-clonic seizures and rapid neurological decline culminating in akinetic mutism within one week. She developed refractory seizures requiring intensive care support, mechanical ventilation, and tracheostomy. MRI brain was normal, while cerebrospinal fluid analysis showed mild lymphocytic pleocytosis with negative infectious meningoencephalitis panel. Autoimmune and paraneoplastic evaluation revealed serum NMDAR antibody positivity. Contrast-enhanced CT abdomen identified a left ovarian teratoma, confirming paraneoplastic anti-NMDAR encephalitis.

Results: The patient received early high-dose intravenous methylprednisolone and IV immunoglobulin, followed by surgical excision of the ovarian teratoma on day 10. Rituximab therapy was initiated subsequently with four weekly cycles. Marked neurological recovery ensued, with successful ventilator weaning, tracheostomy decannulation by day 30, and discharge on day 34 with independent functional recovery.

Conclusion: This case highlights the importance of early diagnosis, aggressive immunotherapy, and prompt tumor removal in paraneoplastic anti-NMDAR encephalitis. A multidisciplinary approach can result in rapid and complete neurological recovery even in fulminant pediatric presentations.

Keywords: Anti-NMDAR encephalitis, ovarian teratoma ,IVIG , rituximab,

BASELINE 18F-FDOPA PET UPTAKE PREDICTS RECOVERY IN DISORDERS OF CONSCIOUSNESS FOLLOWING SEVERE TRAUMATIC BRAIN INJURY: A LONGITUDINAL PILOT STUDY

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Abstract ID: 75

Aim/ Background: Disorders of consciousness (DOC) following severe traumatic brain injury (sTBI) are associated with disruption of dopaminergic pathways involved in arousal and awareness. Amantadine is one of the few pharmacological interventions with evidence supporting its use in DOC; however, the relationship between dopaminergic integrity and recovery remains poorly understood. This study explored the utility of 18F-FDOPA PET as a biomarker of recovery and response to amantadine.

Materials & Methods: Nine patients with DOC following sTBI were enrolled in this longitudinal pilot study (amantadine group, n=4; control group, n=5). After neurological stabilization, participants underwent baseline clinical assessment using the Coma Recovery Scale–Revised (CRS-R) and Disability Rating Scale (DRS), along with 18F-FDOPA PET imaging. Patients were randomized to amantadine or control groups and reassessed clinically and radiologically after 4 weeks. Striatal standardized uptake value ratios (SUVr) were calculated using the cerebellum as the reference region. Group comparisons and Spearman correlation analyses were performed.

Results: Baseline demographic and clinical characteristics were comparable between groups. No significant between-group differences were observed in clinical recovery measured by change in CRS-R ($p=0.266$) and DRS ($p=0.217$), or in longitudinal changes in FDOPA uptake ($p=0.730$). Exploratory analyses demonstrated moderate associations between baseline FDOPA uptake and subsequent changes in CRS-R ($\rho=0.647$, $p=0.060$) and DRS ($\rho=-0.588$, $p=0.096$).

Conclusion: Although significant treatment-related differences were not observed, higher baseline FDOPA uptake was associated with greater clinical improvement. These preliminary findings support the biological rationale for dopaminergic interventions such as amantadine and warrant validation in a larger cohort.

Keywords: TBI, Disorders of consciousness, 18F-FDOPA PET, Amantadine

THE MASQUERADER: WHEN NEURODEGENERATION MEETS AUTOIMMUNITY

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Abstract ID: 52

Aim/ Background: One of the rare autoimmune neurological disorder with overlapping neurodegenerative and autoimmune features is IgLON 5 . It typically presents with sleep disturbances, bulbar symptoms, and movement disorders, often leading to delayed diagnosis. We report a case highlighting clinical recognition and response to immunotherapy.

Materials & Methods: A middle-aged diabetic patient presented with subacute encephalopathy, myoclonic jerks and parasomnias. Evaluation included neuroimaging, cerebrospinal fluid (CSF) analysis, metabolic workup, autoimmune & paraneoplastic panel. Serum and CSF IgLON5 antibodies were detected using IFA. Initial management involved antifungal therapy for BAL proven pulmonary candidiasis, followed by immunotherapy after confirmation of autoimmune etiology.

Results: MRI brain and EEG . Serum and CSF IgLON5 antibodies were positive, confirming the diagnosis. The patient exhibited myoclonus, hyper somnolence , bulbar dysfunction and parasomnias. Treatment with MPS pulse , intravenous immunoglobulin (IVIG) followed by Rituximab led to significant clinical improvement, including resolution of myoclonus and stabilization of neurological status. Antifungal therapy was given for 14 days and stopped due to metabolic derangements with Fluconazole.

Conclusion: Anti-IgLON5 disease presents with heterogeneous features, often mimicking neurodegenerative and sleep disorders. Early diagnosis is critical, as prompt immunotherapy may improve outcomes despite underlying tauopathy. This case underscores the importance of considering autoimmune encephalitis in atypical presentations and supports the role of early immunotherapy in modifying disease progression.

Keywords: Hypersomnolence, bulbar dysfunction, myoclonus , parasomnias

AN UNUSUAL CASE OF BILATERAL PROPTOSIS: IGG4-RELATED OPHTHALMIC DISEASE

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Abstract ID: 28

Aim/ Background: IgG4-related disease is a chronic fibroinflammatory condition that can affect multiple organs and often presents as mass-like lesions. Orbital involvement, can mimic other inflammatory or autoimmune conditions.

Materials & Methods: A 52-year-old man presented with long-standing periorbital swelling and bilateral proptosis for 8 years. Over the last 20 days, he noticed a rapid increase in swelling, along with bilateral temporal headache. MRI showed diffuse soft tissue thickening and edema involving the scalp and face, along with bilateral proptosis, retrobulbar soft tissue thickening, and preseptal inflammatory changes in the orbits.

Given the clinical picture, autoimmune and thyroid-related causes were initially suspected, but all relevant investigations were negative. This prompted further evaluation, and serum IgG4 levels were found to be markedly elevated (>330 mg/dL). Based on the overall clinical, radiological, and laboratory findings, a diagnosis of possible IgG4-related orbital pseudotumor was considered.

Results: A diagnosis of possible IgG4-related orbital pseudotumor was considered. The patient was started on systemic corticosteroids, with gradual tapering. He showed significant improvement in symptoms on follow-up and was subsequently maintained on immunomodulatory therapy.

Conclusion: This case highlights the importance of considering IgG4-related disease in patients with unexplained orbital inflammation. Early recognition can lead to effective treatment and help prevent irreversible fibrosis and end stage organ dysfunction.

Keywords: IgG4-related Ophthalmic Disease

NIVOLUMAB-INDUCED MYOCARDITIS, MYOSITIS, AND MYASTHENIA GRAVIS (MMM) OVERLAP SYNDROME IN UROTHELIAL CARCINOMA: A RARE NEUROLOGICAL IMMUNE-RELATED ADVERSE EVENT

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Hyderabad, Telangana

Abstract ID: 186

Aim/ Background: Immune checkpoint inhibitors (ICIs) have transformed cancer therapy but can rarely cause life-threatening immune-related adverse events (irAEs). The simultaneous occurrence of myocarditis, myositis, and myasthenia gravis (MMM overlap syndrome) is an uncommon but highly fatal complication that requires early diagnosis and prompt immunosuppressive therapy.

Materials & Methods: A 62-year-old man with hypertension, type 2 diabetes mellitus, and high-grade urothelial carcinoma, status post robotic radical cystectomy and adjuvant gemcitabine/carboplatin, presented five days after his second cycle of nivolumab with Drooping Eyelids, diplopia, generalized weakness, myalgias. Neurological examination revealed bilateral ptosis, complete ophthalmoplegia, proximal limb weakness (predominantly lower limbs), and a Single Breath Count of 10. Serum creatine phosphokinase peaked at 4,945 U/L and Troponin I at 5,413 ng/L. Anti-acetylcholine receptor antibodies were negative, whereas anti-MuSK was positive. Electrocardiography demonstrated atrial fibrillation followed by left bundle branch block with prolonged QT interval, while echocardiography showed regional wall motion abnormalities with a left ventricular ejection fraction of 40–45%.

Results: Nivolumab-induced MMM overlap syndrome was diagnosed, and treatment with five sessions of plasmapheresis followed by intravenous methylprednisolone (1 g/day for 5 days) was initiated promptly. The patient demonstrated marked biochemical recovery (CPK 606 U/L) and improved limb strength, with persistent ptosis and partial improvement in ophthalmoplegia.

Conclusion: MMM overlap syndrome is a rare but devastating neurological irAE associated with ICIs. Early recognition, multidisciplinary management, and prompt initiation of plasmapheresis with high-dose corticosteroids are critical to reducing cardiac and respiratory morbidity and improving clinical outcomes.

Keywords: Nivolumab; MMM Syndrome; Myasthenia; Urothelial Cancer

FROM FEVER TO FIRESTORM: A CASE OF FIRES.

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Abstract ID: 187

Aim/ Background: Febrile infection-related epilepsy syndrome (FIRES) is a subtype of new onset refractory status epilepticus (NORSE) characterized by a recent antecedent febrile illness.

Materials & Methods: 17-Year-old Female with no known comorbidities presented with uprolling of eyes and tonic posturing of all 4 limbs following febrile illness. She was intubated and started on multiple Antiepileptics, Initial CSF analysis, MRI Brain were Grossly Normal. EEG showed extreme delta brush. She was started on pulse steroids and later IVIG given for 5 days. After 3 days of IVIG she has got good control of seizures. Repeat MR Brain done three days after seizure onset showed Bilateral Symmetrical diffusion restriction and FLAIR Hyperintensities in External Capsule, Insular cortex. Serum and csf Autoimmune Encephalitis panel was negative. Patient was discharged on antiepileptics and tapering doses of oral steroids.

Results: Functionally independent and no recurrence of seizures. Repeat MRI done two months after discharge showed Complete Resolution of FLAIR changes and EEG showed Generalized Epileptiform Discharges.

Conclusion: A suspicion of FIRES should be confirmed as early as possible to adjust the status epilepticus therapy and initiate targeted immunotherapy as It has extremely long periods of Epileptogenicity.

Keywords: FIRES; Delta Brush; Autoimmune Encephalitis

ASSOCIATION OF TRIGLYCERIDE GLUCOSE INDEX WITH EARLY NEUROLOGICAL DETERIORATION IN ACUTE ISCHEMIC STROKE PATIENTS TREATED WITH INTRAVENOUS THROMBOLYSIS: A COMPARATIVE STUDY IN A TERTIARY CARE CENTER

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Abstract ID: 118

Aim/ Background: Early Neurological Deterioration (END) and poor functional outcomes remain major challenges after intravenous thrombolysis (IVT) for acute ischemic stroke (AIS). The Triglyceride-Glucose (TyG) index, a surrogate marker of insulin resistance, may predict these outcomes.

Objectives: To evaluate the association of the TyG index with END and 90-day functional outcome in AIS patients treated with IVT and determine its predictive value and optimal cutoff.

Materials & Methods: In this prospective observational study, 130 AIS patients treated with recombinant tissue plasminogen activator were categorized into low and high TyG groups using a cutoff of 8.96. END was assessed by change in NIHSS at 72 hours, and 90-day outcome by modified Rankin Scale (mRS), with good outcome defined as mRS 0–2. Multivariate logistic regression and ROC curve analyses were performed

Results: END occurred in 45.4% of patients and was more frequent in the high TyG group (61.5% vs. 29.2%, $p=0.001$). TyG index independently predicted END (OR 4.24, 95% CI 1.77–10.14; $p=0.001$), along with diabetes and dyslipidemia. Good 90-day outcome was achieved in 51.5% of patients. High TyG index was associated with poor outcome (64.6% vs. 32.3%, $p=0.001$) and independently predicted poor outcome (OR 2.93, 95% CI 1.22–6.99; $p=0.016$). ROC analysis showed moderate predictive accuracy for END (AUC 0.697) with an optimal cutoff of 8.89

Conclusion: The TyG index independently predicts END and poor 90-day outcome after IVT. A cutoff of approximately 8.9 may aid early risk stratification in AIS.

Keywords: Triglyceride-Glucose index, insulin resistance, acute ischem

A TWISTED BEGINNING: TONIC SPASMS AS THE UNUSUAL DEBUT OF NMOSD

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Abstract ID: 119

Aim/ Background: Tonic spasms are an uncommon but recognized manifestation of neuromyelitis optica spectrum disorder (NMOSD) and may mimic seizures or functional neurological disorder (FND), resulting in delayed diagnosis.

Materials & Methods: A woman in her early fifties presented with progressive paresthesia, gait imbalance, and recurrent stereotyped tonic spasms. Four years earlier, she had developed ascending paresthesia followed by brief episodes of limb stiffness and abnormal posturing with preserved consciousness. She was diagnosed elsewhere with psychogenic non-epileptic seizures (PNES) and adjustment disorder, and her symptoms initially improved. Seven months before presentation, she developed recurrent paresthesia, hoarseness, left foot dragging, and reappearance of tonic spasms, which worsened with emotional stress. Neurological examination revealed bilateral lower-limb spasticity, brisk reflexes, cranial nerve involvement, and impaired proprioception. MRI demonstrated brainstem and cervical spinal cord demyelinating lesions. CSF showed oligoclonal bands, and serum aquaporin-4 IgG was positive, confirming NMOSD.

Results: The patient underwent eight sessions of plasmapheresis with marked resolution of tonic spasms and neurological deficits, followed by rituximab for relapse prevention.

Conclusion: Tonic spasms in NMOSD are attributed to ephaptic transmission in demyelinated pathways and may be mistaken for PNES or FND. This case highlights how persistent functional labeling can delay recognition of autoimmune demyelination. Careful reassessment when new objective neurological signs emerge is essential.

Tonic spasms can be the presenting manifestation of NMOSD. Clinicians should maintain a high index of suspicion for NMOSD in patients with recurrent stereotyped tonic spasms and evolving neurological deficits, as early diagnosis and immunotherapy can prevent irreversible disability.

Keywords: NMOSD, tonic spasms, PNES, functional neurological disorder,

MANY FACES OF GUYONS CANAL ULNAR NEUROPATHY

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Abstract ID: 147

Aim/ Background: To describe the diverse clinical presentations of Guyon's canal syndrome.

To localize the site of ulnar nerve compression using nerve conduction studies/electromyography (NCS/EMG)

To identify the underlying etiologies and anatomical subtypes of Guyon's canal syndrome

Materials & Methods: Retrospective analysis of eight patients with clinically and electrophysiologically confirmed GCS. Clinical findings, NCS/EMG, imaging, etiology, and anatomical subtype were reviewed

Results: Eight patients (7 males, 1 female; age range 22–60 years) demonstrated diverse presentations of Guyon's canal syndrome. Etiologies included ganglion cyst, repetitive occupational wrist compression, cyclist's handlebar palsy, prolonged crutch use, vascular malformation, and idiopathic compression. Clinical manifestations ranged from isolated motor weakness to pure sensory involvement and mixed sensorimotor deficits. 2 type1, 2 type2, 2 type3, 1 type 4, 1 type 5.

Conclusion: Guyon's canal syndrome exhibits considerable clinical and electrophysiological heterogeneity. Accurate localization using NCS/EMG combined with imaging facilitates identification of the underlying etiology and guides appropriate management, including surgical decompression where indicated. Awareness of

atypical subtypes is essential to avoid misdiagnosis and optimize outcomes.

Keywords: Guyons ulnar neuropathy

EXPLORING BLOOD AND CEREBROSPINAL FLUID BIOMARKERS IN IDIOPATHIC INTRACRANIAL HYPERTENSION

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Abstract ID: 138

Aim/ Background: Idiopathic intracranial hypertension (IIH) is a neurological disorder characterized by elevated intracranial pressure without an identifiable underlying cause. Emerging evidence suggests that BBB dysfunction and neuroinflammatory mechanisms may contribute to its pathophysiology; however, molecular evidence remains limited. Identification of biomarkers such as Aquaporin-1 (AQP1), Aquaporin-4 (AQP4), renin, angiotensin, heat shock proteins, and CD146 may provide insights into disease mechanisms and facilitate the development of novel diagnostic and therapeutic strategies.

Materials & Methods: In this prospective case-control study, 40 patients with IIH diagnosed according to Friedman's criteria, 40 age- and sex-matched individuals undergoing spinal anaesthesia for non-neurological elective surgery (CSF controls) were recruited at a tertiary care centre in North India between March 2023 and September 2024. Biomarker levels were measured using enzyme-linked immunosorbent assay (ELISA), while gene expression analyses were performed using Real time-PCR.

Results: The mean age of IIH patients was 35.73 ± 9.75 years, with a mean BMI of 28.15 ± 5.16 kg/m². CSF AQP4 levels were significantly reduced in patients with IIH ($p=0.016$), whereas CSF renin levels were significantly elevated ($p=0.008$). No significant differences were observed in AQP1 or angiotensin levels. CSF expression of HSP60 ($p=0.038$) and CD146 ($p=0.008$) was also significantly higher in IIH patients.

Conclusion: Patients with IIH demonstrated reduced CSF AQP4 levels, elevated CSF renin levels, and altered expression of HSPs, supporting the involvement of neuroinflammation and BBB dysfunction in disease pathophysiology. These biomarkers may serve as promising candidates for future diagnostic applications and targeted therapeutic interventions in IIH.

Keywords: IIH, Biomarkers, Neuroinflammation

CORRELATION OF INFLAMMATORY & HEMATOLOGICAL BIOMARKERS WITH SEVERITY AND PROGNOSIS IN ACUTE STROKE: A PROSPECTIVE OBSERVATIONAL STUDY FROM A TERTIARY CARE CENTRE IN CENTRAL INDIA

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Abstract ID: 13

Aim/ Background: Aims:

To assess the correlation between NLR, serum ferritin, CRP, MPV, and platelet count with severity and prognosis of acute stroke, and to evaluate their association with established stroke severity scales.

Background:

Stroke is a leading cause of mortality and long-term disability worldwide. Inflammation plays a pivotal role in the pathophysiology of both ischemic and hemorrhagic stroke. Easily available inflammatory biomarkers such as neutrophil-to-lymphocyte ratio (NLR), serum ferritin, C-reactive protein (CRP), and mean platelet volume (MPV) may serve as useful predictors of stroke severity and prognosis.

Materials & Methods: This prospective observational study was conducted in tertiary care centre of Central India. A total of 250 patients aged 18–80 years with acute ischemic or hemorrhagic stroke were included. Clinical assessment was performed using GCS, NIHSS, CSS, mRS, and ICH score. Laboratory parameters including NLR, serum ferritin, CRP, MPV, and platelet indices were measured at admission and follow-up. Statistical analysis was done using SPSS-25, with $p < 0.05$ considered significant.

Results: Of the 250 patients, 68.4% had ischemic stroke and 31.6% hemorrhagic stroke. Mortality was 13.6%. Mean NLR, serum ferritin, and CRP levels were significantly higher in non-survivors compared to survivors ($p < 0.05$). Serum ferritin and CRP showed strong correlations with stroke severity scales (GCS, NIHSS, mRS, ICH) in both ischemic and hemorrhagic strokes. NLR showed moderate correlation with severity and outcome. MPV demonstrated weak and inconsistent associations with stroke severity and prognosis.

Conclusion: NLR, serum ferritin, and CRP are significant, inexpensive, and easily available biomarkers that correlate well with stroke severity and prognosis. These parameters may aid in early risk stratification and prognostication of acute stroke patients

Keywords: Stroke, Prognosis, Inflammatory markers, stroke mortality

BALLOON-ASSISTED STENTING OF A CRITICALLY STENOSED VERTEBRAL ARTERY IN QUADRUPLE EXTRACRANIAL CEREBROVASCULAR OCCLUSION

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Abstract ID: 415

Aim/ Background: Severe multivessel extracranial cerebrovascular occlusive disease poses a major hemodynamic challenge. We report a rare case of bilateral internal carotid artery (ICA) occlusion with critical stenosis of the dominant vertebral artery, successfully managed by endovascular revascularization.

Materials & Methods: A 59-year-old chronic smoker presented with sudden-onset aphasia followed by progressive right-sided weakness over three days. Clinical examination, MRI brain, CT angiography, and digital subtraction angiography were evaluated to assess cerebrovascular anatomy and collateral circulation.

Results: MRI demonstrated an acute left perisylvian infarct. CT angiography and digital subtraction angiography revealed complete occlusion of both ICAs at their origins, a hypoplastic right vertebral artery, and critical V1-segment stenosis of the left vertebral artery. Collateralization was present. External carotid–ophthalmic collaterals partially supported the anterior circulation, while the left vertebral artery served as the sole effective cerebral inflow, supplying bilateral middle and anterior cerebral artery territories through posterior communicating arteries. A 4 × 18 mm balloon-mounted stent was deployed across the left V1 stenosis, restoring robust antegrade flow without complications. The patient remained stable and was discharged on dual antiplatelet and statin therapy.

Conclusion: This case highlights the remarkable resilience of cerebral collateral circulation in extensive occlusive disease. Detailed angiographic assessment is essential to identify the critical remaining inflow pathway. Targeted revascularization of a single severely stenosed vertebral artery can restore cerebral perfusion and may prevent further hemodynamic ischemia in selected patients.

Keywords:

TWO NEGATIVE WORK-UPS, ONE ANSWER: UNMASKING DADA2 A DECADE LATER

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Abstract ID: 150

Aim/ Background: Deficiency of Adenosine Deaminase 2 (DADA2) is a monogenic vasculopathy caused by biallelic loss-of-function mutations in the ADA2 (formerly CECR1) gene. It classically presents in childhood with polyarteritis nodosa-like vasculitis, livedo racemosa, and recurrent lacunar strokes affecting the deep grey and posterior fossa structures.

Materials & Methods: We report a young man with two temporally distant ischemic strokes, both lacunar and both involving the brainstem, in whom an exhaustive conventional stroke work-up was unrevealing and the diagnosis was ultimately established by whole-exome sequencing.

Results: A previously healthy male suffered a first ischemic stroke at age 21 years, with imaging localizing the infarct to the medulla. Standard etiological evaluation — including cardiac, vascular, haematological, and autoimmune work-up — was negative, and the event was labelled cryptogenic. He remained well until age 30, when he presented with a second ischemic stroke, this time localized to the midbrain, again with lacunar morphology. Repeat comprehensive stroke work-up was again negative. Given the recurrent, young-onset, posterior-circulation lacunar stroke pattern with a negative standard evaluation, whole-exome sequencing was pursued and revealed biallelic pathogenic variants in ADA2, confirming a diagnosis of DADA2-associated vasculitis.

Conclusion: This case underscores that DADA2 should be considered in any young patient with recurrent, unexplained lacunar strokes — particularly those involving the brainstem — even in the absence of the more classically taught cutaneous or systemic vasculitis features. Genetic testing should be incorporated early into the diagnostic algorithm for cryptogenic young-onset stroke, as a specific diagnosis carries direct implications for targeted therapy (anti-TNF agents) and family screening.

Keywords: DADA2, ADA2 deficiency, recurrent young stroke

AUTONOMIC DYSFUNCTION AS A PARANEOPLASTIC SYNDROME IN HODGKIN'S LYMPHOMA: A CASE REPORT

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Abstract ID: 151

Aim/ Background: Hodgkin's lymphoma is a B-cell malignancy of the lymphatic system characterized histologically by the presence of Reed-Sternberg cells. While paraneoplastic syndromes are known complications of malignancy, autonomic dysfunction as an initial and primary presentation is extremely rare in the context of Hodgkin's lymphoma.

Materials & Methods: We report a case of an 82-year-old male presenting with a three-month history of progressive autonomic dysfunction symptoms, including severe orthostatic hypotension, anhidrosis, gastrointestinal disturbances, and gait instability with recurrent falls. Notably, the patient exhibited no cognitive decline. Initial differential diagnoses included rapidly progressive extrapyramidal syndromes; however, extensive workup including cerebrospinal fluid analysis and paraneoplastic and autoimmune panels yielded negative results.

Results: Whole-body Positron Emission Tomography (PET) scanning subsequently revealed multiple enlarged generalized lymphadenopathies. Biopsy confirmed the diagnosis of Hodgkin's lymphoma.

Conclusion: This case underscores the critical importance of considering lymphoproliferative disorders in the differential diagnosis of unexplained subacute autonomic dysfunction, particularly in the elderly population.

Keywords: autonomic dysfunction, paraneoplastic syndrome, hodgkins

CLINICAL PROFILE, CSF PATTERN, NEUROIMAGING FINDINGS AND OUTCOMES OF CEREBRAL CRYPTOCOCCOSIS: A RETROSPECTIVE CASE SERIES FROM A TERTIARY CARE CENTRE

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Abstract ID: 43

Aim/ Background: Central nervous system cryptococcosis is a life-threatening fungal infection traditionally associated with HIV infection; however, increasing cases are being reported among non-HIV immunocompromised individuals, particularly transplant recipients and patients receiving immunosuppressive therapy. Data on the clinical profile, cerebrospinal fluid (CSF) characteristics, neuroimaging findings, and outcomes of cerebral cryptococcosis in Indian patients remain limited. This study aimed to evaluate the clinical profile, CSF characteristics, neuroimaging findings, and outcomes of patients with cerebral cryptococcosis.

Materials & Methods: A retrospective case series was conducted at a tertiary care centre including patients diagnosed with cerebral cryptococcosis between 2021 and 2026. Demographic, clinical, laboratory, cerebrospinal fluid (CSF), neuroimaging, and outcome data were retrieved from medical records and analyzed descriptively. Variables assessed included comorbidities, transplant status, immunosuppressive therapy, presenting symptoms, CSF characteristics, radiological findings, and clinical outcomes.

Results: A total of 26 patients were included with a mean age of 49.3 years; 73% were males. Diabetes mellitus was present in 50%, while 31% were transplant recipients and 35% were on immunosuppressive therapy. HIV positivity was observed in only 15% of cases. Headache (65%) was the most common presenting symptom, followed by fever (46%) and altered sensorium (35%). CSF cryptococcal antigen was positive in all patients, whereas fungal culture and India ink positivity were seen in 62% and 12% respectively. Neuroimaging commonly showed leptomeningeal involvement, with a few patients demonstrating cryptococcoma-like lesions. Mortality among patients with known outcomes was 36%, with altered sensorium and elevated CSF inflammatory markers associated with poorer outcomes.

Conclusion: Cerebral cryptococcosis predominantly affected non-HIV immunocompromised individuals, particularly patients with diabetes, transplant recipients, and those receiving immunosuppressive therapy. Mortality remained high, with altered sensorium and elevated CSF inflammatory markers suggesting poorer outcomes. Early recognition and prompt diagnosis may improve clinical outcomes.

Keywords: Cerebral Cryptococcosis, Cryptococcal meningitis