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Acute Neurology/Oncology
May 7, 12:30 - 13:30
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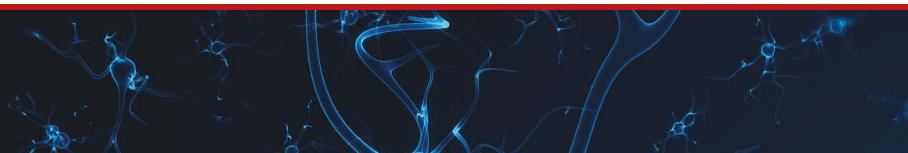

Rapid Deterioration on ITU: A Devastating Combination of Genetics and a Viral Infection

Michal Chinn, Linda Lei, Farrah Jabeen, Talal Al-Mayhani

Royal Free Hospital

Fat embolism syndrome (FES) is a rare but frequently fatal complication of sickle cell disease (SCD), disproportionately reported in patients with heterozygous HbSC rather than homozygous HbSS disease when triggered by parvovirus B19–associated aplastic crisis.

We describe a woman with HbSC disease who presented with bilateral flank pain and rapidly deteriorated with aplastic crisis secondary to parvovirus B19 infection. She subsequently developed acute respiratory failure, encephalopathy, and profound bilateral weakness.

Computed tomography pulmonary angiography demonstrated acute on chronic pulmonary emboli, and she required intensive care admission with multi-organ support. Biochemical findings were striking, with ferritin rising from ~1,000 to 47,000 µg/L within 48 hours alongside thrombocytopenia, strongly suggestive of FES. Neurologically, she deteriorated to a Glasgow Coma Scale (GCS) of 3/15 with preserved brainstem reflexes.

Initial red cell exchange produced minimal improvement. Escalation to five days of plasma exchange resulted in haemodynamic stabilisation and gradual neurological recovery over months. Susceptibility-weighted imaging (SWI) MRI brain demonstrated the characteristic "starfield" appearance, highly suggestive of widespread cerebral fat emboli.

This case is notable given the limited literature describing neurological recovery following catastrophic cerebral FES. It highlights the particular vulnerability of HbSC patients and supports early consideration of plasma exchange in refractory cases.

Labrune syndrome without cysts: a genetically confirmed case highlighting diagnostic challenges.

Musab Eltahir, Luke Canham, Timothy Harrower

Severn Deanery

Labrune syndrome, a rare genetic microangiopathy caused by SNORD118 mutations, is classically characterised by leukoencephalopathy, parenchymal calcifications, and cyst formation. We report the second published case of genetically confirmed Labrune syndrome presenting without cysts, highlighting diagnostic challenges and emerging management strategies.

A 21-year-old female presented with progressive headache, nausea, vomiting, blurred vision, and seizures. Neuroimaging revealed multifocal intracranial lesions with extensive calcifications in the parietal lobe, thalamus, cerebellum, basal ganglia, and dentate nuclei, without cyst formation. She underwent multiple rounds of brain biopsy and extensive serological and cerebrospinal fluid testing, which had not yielded a definitive diagnosis. Following discussion at the neuro-inflammatory multidisciplinary team (MDT), the presence of multifocal white matter changes with calcifications raised concern for Labrune syndrome. Genetic testing subsequently confirmed a pathogenic SNORD118 mutation. The patient experienced multiple admissions for seizure control requiring escalation of antiepileptic therapy (lamotrigine 100 mg BD, brivaracetam 125 mg BD) and was referred for consideration of bevacizumab therapy.

This case highlights the phenotypic variability of Labrune syndrome, demonstrates that cysts are not mandatory for diagnosis, and underscores the importance of early genetic testing to avoid invasive procedures and guide emerging therapeutic strategies.

Delayed Post-Hypoxic Leukoencephalopathy after Overdose: A Case of Biphasic Neurologic Decline with Unexpected Recovery

Aiknaath Jain, James Dias, Ruth Wood, Aref Rastegar, Guru Kumar

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A 55-year-old woman presented with acute encephalopathy and transient hypoxia following an intentional mixed drug overdose of amitriptyline, tramadol and alcohol. She required intubation but was discharged at neurological baseline within one week

Two weeks later, she re-presented with rapidly progressive cognitive decline. Examination revealed akinetic mutism, bilateral pyramidal signs and abnormal paroxysmal movements. CSF was normal and EEG demonstrated moderate to severe encephalopathy. Infectious, metabolic and autoimmune screening was unrevealing, however she was empirically treated with corticosteroids. MRI showed diffuse, symmetrical confluent white matter changes with globus pallidus involvement, consistent with delayed post-hypoxic leukoencephalopathy (DPHL).

Her second admission was protracted with seizures, spasticity and a prolonged disorder of consciousness. At one month she was minimally responsive (WHIM 1, CRS-R 0). She was transferred to a specialist neurorehabilitation unit. Surprisingly, over subsequent months, she demonstrated remarkable recovery with WHIM scores improving to 50. At 1-year follow-up she was independently mobile with only mild residual attentional difficulties.

DPHL is a rare biphasic complication of hypoxic brain injury, characterised by delayed neurological deterioration after initial improvement. This case demonstrates meaningful functional recovery is possible despite severe early disability, underscoring the need to avoid premature prognostic pessimism even after prolonged disorders of consciousness.

Increasing use of immune-checkpoint inhibitors: need for increased awareness, and implications for service development

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Background: The use of immune checkpoint inhibitors (ICIs) in cancer therapy has increased substantially over the past decade, leading to greater recognition of life-threatening ICI-associated neurotoxicities. However, further work is required to better understand the epidemiology and management of these conditions, with implications for service development.

Method: Retrospective case note review of ICI-treated patients in south east Wales (demographic population 1.7 million, 5000 new cancer diagnoses per year) by keyword screening of electronic health records, from 2017-2025. Cases of interest: 3M syndrome (Myasthenia Gravis, Myositis, Myocarditis), encephalitis, cytokine release syndrome (CRS), haemophagocytic lymphohistiocytosis (HLH) and Guillain-Barre syndrome.

Results: Between 2018 and 2025, annual ICI use almost tripled (2018: 105, 2019: 207, 2020: 140, 2021: 206, 2022: 243, 2023: 282, 2024: 364, 2025: 286). Within 2025, one patient had clinical features suggestive of 3M syndrome, with a further case showing a possible dual overlap (myositis with myocarditis); three patients had suspected isolated myocarditis, four had myositis and two had CRS.

Conclusion: Although ICI- associated neurotoxicities are rare, cases are likely to increase further as ICI use continues to expand. This highlights the need for greater clinical awareness and early multidisciplinary collaboration, particularly neurology input, to optimise recognition and management.

A rare cause of bilateral arm weakness in an older adult

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Case presentation: A 60-year-old previously well man presented with acute, painful, progressive bilateral upper limb weakness following a two-week viral prodrome. Weakness was initially asymmetric and progressed over seven days to severe flaccid paralysis of both upper limbs with marked functional impairment. Upper limb reflexes were absent, with preserved sensation.

Cranial nerve and lower limb examinations were normal.

Investigations demonstrated lymphocytosis with atypical mononuclear cells, markedly deranged liver function tests, splenomegaly, and cervical lymphadenopathy. MRI of the brain, cervical spine, and brachial plexus revealed no structural abnormality. Cerebrospinal fluid analysis was normal. EBV serology demonstrated positive viral capsid antigen IgM and IgG, with persistently elevated serum EBV PCR confirming primary infection; CSF EBV PCR was negative. Neurophysiology supported a diagnosis of bilateral brachial neuritis.

Management and outcome: The patient was treated with intravenous immunoglobulin following multidisciplinary discussion, alongside multimodal analgesia and rehabilitation. Partial improvement in pain and strength was observed, though significant weakness persisted at follow-up.

Conclusion: This case challenges the assumption that primary Epstein–Barr virus infection does not occur in older adulthood, demonstrating its presentation as severe bilateral brachial neuritis in an immunocompetent patient and highlighting a key diagnostic consideration.

Development and Validation of the NOVA Score for Surgical Triage in Metastatic Spinal Cord Compression

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Objectives: To develop and validate the NOVA Score for predicting 12-month survival in patients with metastatic spinal cord compression (MSCC), and to compare its performance to established prognostic scores.

Methods: Two independent cohorts of patients with MSCC referred for surgical consideration were included from a tertiary spine center: a derivation cohort (n=184) and a validation cohort (n=100). The final NOVA Score incorporated three clinical parameters: Karnofsky Performance Status (KPS), primary tumor category, and presence of extraspinal metastases. The score ranges from 0–10, with a threshold of ≥7 used to predict >12-month survival. Predictive performance was assessed using accuracy, F1-score, area under the receiver operating characteristic curve (AUC), Cohen's kappa, and calibration plots.

Results: In the validation cohort, the NOVA Score achieved an accuracy of 71.0%, AUC of 0.693, F1-score of 47.3% for >12-month survival, and Cohen's kappa of 0.28. Calibration plots showed good agreement in the mid-to-high probability range. Compared to the Revised Tokuhashi Score, OSRI, Modified Bauer Score, and oncologist estimates, NOVA demonstrated superior sensitivity for identifying long-term survivors.

Conclusions: The NOVA Score is a simple tool for early prognostication in MSCC. Its minimal input requirements and balanced performance support its utility for triage. Further external validation is warranted.

Human Versus Machine: Comparative Accuracy of Survival Prediction in Metastatic Spinal Cord Compression

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Background: Accurate survival prediction in metastatic spinal cord compression (MSCC) is critical for treatment planning but remains difficult. Traditional scoring systems and physician estimates often perform inconsistently. This study compared the predictive accuracy of oncologist judgment, surgeon-calculated Tokuhashi scores, and ChatGPT-based assessments against actual survival outcomes.

Methods: Ninety-nine anonymized MSCC cases were retrospectively analyzed. Clinical summaries included demographic, oncological, imaging, and functional data. Survival predictions (<6 months,6–12 months,>12 months) were obtained from oncologists, spine surgeons (Tokuhashi), and ChatGPT using both Tokuhashi-based and literature-informed estimations.

Results: ChatGPT-assisted Tokuhashi scoring achieved the highest overall accuracy (54%), followed by surgeon Tokuhashi (50%) and oncologist judgment (48%). Short-term survival (<6 months) was best predicted by surgeon Tokuhashi (71% recall) and ChatGPT Tokuhashi (69%). Prediction of intermediate survival (6–12 months) was poor across all methods. Oncologists achieved the highest recall for >12 months survival (75%). Machine learning analyses consistently highlighted Karnofsky Performance Status and patient age as the strongest predictors, whereas MSCC status and ASIA grade had limited influence.

Conclusions: Structured prognostic tools, particularly Tokuhashi scoring augmented by AI, improve short-term survival prediction in MSCC. Functional status and age emerged as the most robust predictors, supporting the need for individualized models that emphasize functional reserve.

Beyond Infection and Infiltration: Autoinflammatory Neurological Disease in Chronic Myelomonocytic Leukaemia

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Background: Neurological complications in people with haematological malignancies present significant diagnostic and management challenges due to non-specific symptoms and multiple potential mechanisms including malignant infiltration, opportunistic infection and autoimmune phenomena. Early identification of the underlying aetiology is essential as treatment strategies differ substantially.

Case report: A 76-year-old man with chronic myelomonocytic leukaemia (CMML) and Rosai–Dorfman disease presented with a two-month history of fluctuating encephalopathy, visual and auditory impairment, lumbar radiculopathy and bi-cytopenia. MRI demonstrated smooth left parietal dural enhancement and abnormal signal of the right lateral femoral cutaneous nerve. Cerebrospinal fluid (CSF) analysis revealed persistent mild pleocytosis with hypoglycorrhachia. Extensive work-up for infectious, inflammatory, and infiltrative causes was negative, including autoantibody testing and CSF metagenomics. CMML-associated systemic autoinflammatory disease (SAID) was diagnosed, supported by hypocomplementaemia, elevated inflammatory markers, myeloid inflammatory infiltrates on bone marrow biopsy and prior CMML-associated immune thrombocytopenia. Treatment with intravenous methylprednisolone and immunoglobulins resulted in clinical and haematological improvement.

Conclusion: SAIDs result from innate immune dysregulation, often driven by somatic myeloid mutations. Although cytopenias, vasculitis and serosal inflammation can occur in 20% of CMML patients, neurological involvement is rare and underdiagnosed. Corticosteroids remain first-line therapy, with evolving evidence supporting the use of hypomethylating agents in this disorder.

Dysarthria and Dysphagia: When a Tongue Tumour Mimics Central Causes

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Background: Isolated lingual dysarthria typically arises from central neurological or neuromuscular pathology, resulting in lingual monoparesis. However, peripheral structural pathology can produce similar findings. Tongue tumours may impair articulation by disrupting lingual mobility, making dysarthria an early or isolated symptom. Recognising this pattern helps avoid misattributing symptoms solely to central pathology.

Case: A 67 year old man presented with subacute dysarthria and dysphagia. Initial MRI/MRA, myasthenia antibodies, and EMG were normal, and symptoms briefly improved. One month later he re presented with worsening symptoms. Examination showed globally reduced tongue movements without atrophy and lingual dysarthria without fatigability or other neurological deficits. Repeat imaging revealed a tongue mass infiltrating intrinsic muscles. Biopsy confirmed P16 positive squamous cell carcinoma of the tongue base, and he commenced curative chemoradiotherapy.

Discussion: This case emphasises the need to consider local tongue pathology when dysarthria is confined to lingual movements. Tumours can mimic isolated hypoglossal dysfunction, and subtle dysarthria with early dysphagia may precede mass related symptoms. Thorough oral examination remains essential.

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Gut Instinct: When Gastrointestinal Symptoms Signal a Neurological Storm

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Background: Acute Motor and Sensory Axonal Neuropathy (AMSAN), a severe Guillain-Barré Syndrome (GBS) variant, can present atypically with isolated gastrointestinal symptoms due to autonomic involvement. We report a case where colonic pseudo-obstruction was the initial and predominant troublesome manifestation of AMSAN.

Case Summary: A 72-year-old male was admitted following collapse after a diarrhoeal illness and vomiting. Bloods showed severe hypokalaemia and CT imaging revealed marked large bowel dilatation, managed conservatively. Despite some improvement of pseudo-obstruction, he developed progressive limb weakness, dysarthria, and type II respiratory failure requiring intubation and tracheostomy. Neurophysiology confirmed severe, sensory-motor axonal neuropathy, consistent with AMSAN variant of GBS; MRI spine was unremarkable. He received a 5-day IVIg course. His prolonged admission was complicated by pulmonary embolism, atrial flutter, ventilator-associated pneumonia, acute kidney injury secondary to pseudo-obstruction, C. difficile infection, and grade 3 sacral pressure ulcer. At five months, he had objectively improved (MRC sum score 62/80) and was transferred for specialist neurorehabilitation.

Conclusion: This case highlights pseudo-obstruction as a rare initial and persistently troublesome presentation of AMSAN, underscoring the importance of considering GBS variants in patients with unexplained gastrointestinal dysmotility and evolving neuromuscular weakness. Early recognition and multidisciplinary management are crucial for improving outcomes.