pleocytosis of 144 x10^9/L with normal cultures and negative pan-viral PCR multiplex. Broad anti-infective treatment was continued. MRI Brain and spine revealed intracranial and conus medullaris leptomeningeal enhancement. Standard anti-neuronal antibody and limbic encephalitis panel testing was negative. Due to persistent fever, lower limb weakness and neurogenic bladder a PET study confirmed on-going meningo-myeloencephalitis despite broad anti-infective therapy. At further request, extended anti-neuronal antibody testing by immunopathology supported a diagnosis of glial fibrillary acidic protein (GFAP) astrocytopathy. Immunotherapy with IVIG, mycophenolate and initial pulse steroids with a slow oral taper were able to induce clinical remission after 4 months.

Conclusion GFAP astrocytopathy can mimic an infective meningomyeloencephalitis. Early broader immunologpathological consideration is key to diagnosis.

2839 IMMUNE MYOPATHY WITH PERIMYSIAL PATHOLOGY (IMPP) IN A PATIENT WITH AN UNUSUAL CLINICAL PHENOTYPE AND ANTI-MI-2 ANTIBODY

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Background IMPP have myopathological features that are distinct from dermatomyositis (DM) and are usually associated with extramuscular manifestations.[1 2] There is also some evidence that the pathophysiology in Anti-Mi-2 DM may be more similar to phenotypes with IMPP than DM associated with other myositis antibodies.[3]

Methods/Results We present a case of a 71-year-old woman with a six-month history of progressive muscle weakness. Her father had Paget's disease of the bone and Dementia in his 70's. Examination revealed distal weakness in the upper limbs including the deep finger flexors and proximal weakness in the lower limbs. She also had oropharyngeal dysphagia and axial weakness. There were no nailbed or skin changes. Investigations revealed creatinine kinase level of 3579U/L, anti-nuclear antibody of 1:2560 with positive Mi-2a and Mi-2b antibodies. Nerve conduction studies were normal. Electromyography revealed mixed myopathic and neurogenic motor unit potentials in weak muscles with fibrillation potentials. Muscle biopsy of the left vastus lateralis showed features of a chronic immune mediated myopathy consistent with a chronic IMPP. Given her atypical presentation and family history of Pagets disease, Pathwest comprehensive neuromuscular genetic panel was also sent off. Extensive evaluation including FDG-PET was negative for any malignancy. She was treated with a combination of intravenous methylprednisolone and immunoglobulin. After two months of treatment the patient remained similarly disabled.

Conclusions This case highlights the clinical and pathological heterogeneity that can be seen in patients with acquired immune and inflammatory myopathies and provides further evidence of the overlap between IMPP and Anti-Mi-2 DM.

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2843 CEREBRAL VENOUS SINUS THROMBOSISPRESENTING WITH HEMIAGUSIA: A LESSON IN VASCULAR ANATOMY

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Introduction Cerebral venous sinus thrombosis (CVST) typically presents with headaches and intracranial complications of venous congestion including.

Methods Single case report.

Results A 67-year-old female presented with a 6 week history of taste change affecting the left side of the tongue and a rubbery sensation in her left cheek noted when chewing. MRI identified loss of flow voids in the left transverse sinus and no evidence of stroke or other intracranial pathology. CT venogram demonstrated an extensive filling defect in the left anterior transverse sinus extending to the left jugular bulb consistent with CVST. The patient commenced anticoagulation. Two months later her taste and buccal sensation were improving.

Discussion Taste sensation to the anterior two thirds of the tongue is supplied by the chorda tympani, a branch of the facial nerve that joins the lingual nerve. Sensation of the buccal mucosa is supplied by the long buccal nerve (branch of V3). The chorda tympani, lingual nerve and long buccal nerve traverse the infratemporal fossa in close anatomical association with the pterygoid venous plexus (2). Valveless emissary veins including the sphenoidal emissary vein and emissary vein of the foramen ovale, drain blood from the pterygoid plexus to the ipsilateral sphenoid sinus via the cavernous sinus (3). We postulate that obstruction of the usual venous outflow due to left transverse and sigmoid venous sinus thrombosis, resulted in dilatation of the left pterygoid venous plexus causing mechanical impingement of the chorda tympani and buccal nerve, with subsequent loss of taste and buccal sensation localised to the left. No alternative plausible explanation for the symptoms could be identified centrally based on clinical anatomical localisation or imaging findings.

Conclusion This case expands the spectrum of clinical presentation of CVST. It highlights the potential diversity of presentations related to the impact of venous congestion on central and peripheral neurological function.

2844 ANTI-IGLON-5 ENCEPHALITIS: A CASE SERIES

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Anti-Iglon-5 disease has wide array of manifestations.[1] Those can range from sleep disorders with parasomnia, PSP-like presentations, cognitive decline, bulbar dysfunction, or movement disorders.[2]

We present 3 cases of Anti-Iglon-5 encephalitis where patients had variable presentations demonstrating the