disorders of connective tissues. Nasal CSF leakage is extremely rare.¹

Methods and results We present the case of a 40-year-old woman presented to hospital for few days history of postural headache associated with clear intermittent discharge from right nostril without any signs of meningism. There was no history of trauma. She has a background history of Marfan syndrome with associated complications of ASD repair at age 2, mechanical Aortic and Mitral valve replacement, aortic root repair, previous ST elevation MI with LV dysfunction, automated implantable cardioverter-defibrillator in situ, atrial fibrillation, and Hashimoto's thyroiditis. Her regular medications are warfarin, bisoprolol and thyroxine. The clear nasal discharge was positive for β -2 transferrin confirming cerebrospinal fluid. Her CT Brain did not reveal any clear site of CSF leak. She had a flexible nasendoscopy which showed normal nasal passageway, no defect in nasal mucosa and no active CSF leakage. She was managed conservatively with strict bed rest and advised to minimise strenuous activity and heavy lifting.

Conclusion Spontaneous cerebrospinal fluid leak is uncommon condition and frequently associated with hereditary disorders of connective tissues. Nasal CSF leakage is extremely rare.¹ Testing β -2 transferrin has high sensitivity and specificity.² Initial treatment may include bed rest, oral or intravenous hydration, oral caffeine or corticosteroids.^{3–4} If conservative therapy fails, surgical repair with nasal endoscopic approach is recommended.^{2–5}

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076

A RARE CASE OF ANTI-GLUTAMATE DECARBOXYLASE ANTIBODY SYNDROME PRESENTING WITH RECURRENT VOMITING

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Introduction Anti-glutamate decarboxylase antibody (anti-GAD) has been linked with various neurological syndromes including stiff-person syndrome, limbic encephalopathy, cerebellar ataxia, eye movement disorders and epilepsy (collectively known as 'anti-GAD positive neurological syndromes').¹ We describe a very atypical phenotypic presentation of anti-GAD syndrome with unexplained vomiting and weight loss.

Case A 46 years old lady with no past medical or family history of note, presented with 6 months history of severe headaches and recurrent attacks of episodic vomiting (4–6 episodes of multiple vomiting daily) with no identified precipitant and complete normality in between the episodes with no other associated symptoms. She reported 15 kg of unintentional

weight loss. Neurological examination and investigations including MRI brain, CT angiogram and liver enzymes, immunoglobulins, thyroid function, vasculitic screen were normal. Upper GI endoscopy, gastric emptying studies, CT imaging of chest, abdomen and pelvis and whole body PET scan were unremarkable. Serum autoimmune antibody screen was positive with high titre of anti-GAD antibody (1200 kU/liter). The cerebrospinal fluid anti-GAD antibody titre was raised at 103.7 kU/liter with otherwise normal parameters including negative oligoclonal bands. The nerve conduction studies did not show continuous motor activity or spasmodic reflex myoclonus (seen in stiff-person syndrome).² A therapeutic trial of immunosuppression was introduced with moderate improvement in symptoms.

Conclusion Anti-GAD neurological syndromes are rare and this is a unique presentation of the same. It is not completely understood why the presence of one antibody causes varied syndromes. The hypothesis is that the recurrent vomiting is possibly due to diaphragmatic spasms.

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077

AN UNUSUAL PRESENTATION OF ANTIBODY NEGATIVE AUTOIMMUNE ENCEPHALITIS: A CASE REPORT

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Introduction Immune-mediated encephalitis can occur in the absence of measurable pathogenic antibodies in CSF or serum.

Methods and results We present the case of a 29-year-old Sudanese woman presented with four days history of meningism. She is three months post-partum with her fifth child and does not have significant past medical history. She is a current smoker with history of methamphetamine and heroin use until 5 years ago. There were no focal neurological deficits on examination. CT brain was normal and her CSF had $420 \times 10^6/L$ leucocytes (Polymorphs 18, Mononuclears 402). Three days later, she deteriorated clinically with GCS 11 and developed left sided hemiplegia requiring intubation and ventilation in intensive care.

The CSF culture along with autoimmune encephalitis panel, anti-neuronal antibodies, HSV, VZV, Enterovirus, cryptococcal antigen, TB quantiferon, HIV, Malaria were negative. Her immunology tests were unremarkable including rheumatoid factor, complements, anti-nuclear antibodies, dsDNA, PR3, MPO, RNP, Smith, Ro, La, Scleroderma 70 and Jo-1 antibodies. The MRI Brain showed persistent changes in right hemispheric cortical and subcortical gyriiform FLAIR hyperintensities which were also present in DWI images. The EEG showed right temporo-occipital region 7–8 Hz rhythmic discharges with generalised diffuse slowing. She had right parietal lobe brain biopsy which shows perivascular and intramural inflammation by mononuclear cells almost comprised of CD3 T-cells consistent with encephalitis associated with secondary lymphocytic vasculitis. She was treated with intravenous pulsed methylprednisolone and IVIg