Case Presentation:
A 19-year-old gentleman with underlying nephrotic-nephritic syndrome since 2013 admitted for acute kidney injury. He presented with dyspnea, facial puffiness and leg swelling for one week. His urea was 32 mmol/L, creatinine was 1567 ìmol/L and bicarbonate was 4.2 mmol/L. Ultrasound kidneys showed no evidence of obstructive uropathy. He was treated with intravenous methylprednisolone of 500 mg daily for 3 days and intermittent haemodialysis. He was discharged well after 10 days in hospital.

2 days later, he presented again to hospital with status epilepticus and altered sensorium. On arrival to hospital, his Glasgow coma scale (GCS) was E1V1M4, blood pressure was 170/116 mmHg. He was intubated for airway protection. Chest radiography showed right upper lobe consolidation. Urgent non-contrasted computed tomography of brain showed symmetrical white matter hypodensity at the bilateral occipital, bifrontal, left parietal and left cerebellum region consistent with posterior reversible encephalopathy syndrome (PRES) Contrasted brain imaging showed no evidence of venous thrombosis.

Abstract:
Posterior reversible encephalopathy syndrome (PRES) is a rare neurological complication. It can occur as a complication of hypertensive disease of pregnancy, autoimmune disorder or after immunosuppressant. Typical imaging features of PRES are bilateral symmetrical involvement of parietal and occipital lobe. Here, we reported a case of PRES after methylprednisolone.

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Fig.-1 : Non contrasted Computed tomography of the brain
Fig.-2: Contrast-enhanced Computed tomography of the brain

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He was admitted to intensive care unit (ICU) and treated with supportive management. His high dose steroid was withheld. He was treated with intravenous antibiotics for aspiration pneumonia, anti-epileptics and intermittent haemodialysis. His GCS subsequently improved to normal after one week in ICU. He was discharged home with plan of slow reintroduction of steroid.

PRES is a clinical-radiological syndrome characterized by seizures, headache, altered consciousness and visual disturbances[1]. Various conditions are associated with PRES, which include sepsis, Guillain-Barre syndrome, autoimmune diseases, cytotoxic agent, steroid and pregnancy related complication. Severe hypertension is the most common etiology of PRES. The exact pathophysiology is still uncertain, but it’s believed to be due to dysfunctional cerebral autoregulation.

In computed tomography, PRES lesions are thought to be due to vasogenic edema and tend to be symmetrical, affecting both hemispheres. The commonest (≥90%) site involved are parietal and occipital lobes. However, important to note PRES lesions can also occur in watershed zones, which are frontotemporal region. Magnetic resonance imaging remained the gold standard in diagnosing PRES[2].

In term of treatment, treatment of PRES is mainly supportive, which includes optimal blood pressure control, withdrawal of offending drug, treatment of underlying etiology and seizure control[3]. Most patient with PRES have good neurological recovery. In conclusion, PRES is a rare neurological complication after methylprednisolone. Recognizing the typical imaging features will help in achieving the diagnosis.

References: