Abstract 375 Table 1 Table of investigations

	Prior chemotherapy	D2 post Chemotherapy	D3 post chemotherapy	D6 post chemotherapy	D11 post chemotherapy	D19 post Chemo therapy
Platelet	33	7	42	142		152
AST	40	5381	7978	938	100	79
ALT	16.4	2035	3524	1829	610	127
Bilirubin	4.9	29.6	49.9	34.5	37.8	19.9
Ammonia		125	84			
PT		33.7	34.5	18.8		

brain revealed no intracranial bleed and ultrasound abdomen showed gross ascites. Multidisciplinary teams discussion led to diagnosis of HTS.

She was closely monitored in intensive care unit with vigorous supportive care included high flow nasal cannula, judicious fluid and urine output monitoring.

She showed tremendous improvement, regained full GCS within 24 hours of admission. Her platelet count had marked increment by second day of admission and normalized within one week. All the blood parameters showed improvement within a week.

She received subsequent chemotherapy with reduction of actinomycin D dose with no recurrence of HTS.

Results The onset of HTS occurred within 10 weeks of diagnosis and usually occurred 1 to 7 days post actinomycin D administration. HTS can last up to 12 days and resolve with supportive treatment. After recovery, most children can tolerate actinomycin D at a reduction dosage without recurrence of the syndrome.

Therapy of HTS is mainly vigorous supportive care. However, defibrotide, which is an antithrombotic has emerged as an effective therapy in view of histopathologic marker of HTS is obliteration of hepatic vein.

Conclusions Early detection and recognition of syndrome will prompt immediate action resulting extremely favourable outcome. Further studies regarding risk factors and promising drugs should be carried out to reduce mortality of HTS.

379

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN ACUTE POST-STREPTOCOCCAL GLOMERULONEPHRITIS

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Background Acute post streptococcal glomerulonephritis (PSGN) is well known sequel of group A β -hemolytic streptococcus infections and may be complicated with hypertensive encephalopathy followed by Posterior reversible encephalopathy syndrome (PRES). PRES is also known as acute hypertension encephalopathy, usually present with seizures, hypertension, headache, encephalopathy and/or visual disturbance. The pathophysiology behind PRES is poorly understood. It is usually associated with hypertensive encephalopathy, kidney disease, autoimmune disease, immunosuppressive drug and organ transplantation. Contrast-enhanced computed tomography imaging allows for fast assessment which shows features

of edema of the posterior cerebral hemisphere, especially at the parieto-occipital region. PRES can be a presenting sign of PSGN and vice versa.

Objectives To study relationship between PSGN and PRES

Methods Two case reports PSGN with PRES, based on contrast-enhanced computed tomography (CECT) brain.

Results We reported two cases of PSGN with PRES.

First case is a 11 years old boy, who presented with status epilepticus, following one-day history of headache and vomiting. He was afebrile, no history of epilepsy or recent trauma and any drug intake. He had history of left facial cellulitis secondary to odontogenic infection one-week prior the onset of seizure. He was initially diagnosed with PRES after CECT brain.

Second case is a 6 years old boy, who presented with multiple infected impetigo lesions over bilateral lower limb, fever and vomiting. He also developed blurry vision and recurrent vomiting, requiring ICU care due to hypertensive crisis. CECT brain was performed and reported as PRES.

Similarly, both children had persistent microscopic hematuria and hypertension, and investigations show low C3 and normal C4 levels with raised Anti-streptolysin O titer (ASOT), fulfilling the criteria of PSGN.

Conclusions The incidence of PRES is rare in children, but should always be a diagnosis to be considered in children with PSGN presenting with hypertensive crisis and/or rapidly progressive neurological manifestations, e.g. status epilepticus. Early recognition and prompt treatment are important measures in reducing morbidity and mortality.

380

THINK OUT OF THE BOX! LYMPHADENOPATHY, SKIN RASHES AND SKULL LESIONS; ALL ENDED UP WITH LANGERHANS CELL HISTIOCYTOSIS ?

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Background Langerhans cell histiocytosis (LCH), previously known as histiocytosis X, is a rare paediatric disorder. The abnormal proliferation of specialized dendritic cells found in skin and mucosa, which resembles langerhans cells, account for the underlying pathology of LCH (1). The vast clinical presentations of LCH, from self-limiting lesion to fulminant disseminated disease, make its diagnosis a challenge for doctors. Its incidence stands at around 1–5 per million children. Some literatures showed that children of parents who were exposed to certain chemicals such as benzene have an