

Original Research Paper

Pediatrics

PRESS SYNDROME IN POST STREPTOCOCCAL GLOMERULONEPHRITIS

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ABSTRACT

Background: Posterior reversible encephalopathy syndrome(PRES) is characterized by hypertension, seizure, headache, altered mental status, visual disturbance, and diagnosed in presence of typical

lesions on brain imaging.

Case: We report 14-yr girl presented with features suggestive of PRES. Computerized tomography of brain showed multiple ill defined, non-enhancing hypodensities in frontoparietal and left occipital area due to corticomedullary oedema suggestive of

Result: After management with iv fluid, antibiotics, antiepileptics and antihypertensive patient improved.

Conclusion: If recognised and treated early the clinical syndrome resolves within a week. High index of clinical suspicion and prompt treatment can reduce morbidity, mortality and helps in early recovery.

KEYWORDS: critical care, hypertension, posterior reversible encephalopathy syndrome, seizures.

INTRODUCTION:

Severe hypertension in children usually results from secondary causes, with renovascular diseases constituting 5-10% of all paediatric cases of hypertension.[1]. Developing hypertensive encephalopathy following post-streptococcal glomerulonephritis (PSGN) is a known but uncommon manifestation in children and developing PRES in such a situation is very rare.[2] Posterior reversible encephalopathy syndrome is characterized by headache, altered mental status, visual disturbances, and seizures, with typical radiographic findings of vasogenic oedema involving posterior cerebral region.[3] Even though the pathophysiology is still unclear, various associated underlying conditions such as renal diseases, autoimmune diseases, or cancers have been implicated. [3,4] In addition, there are several well identified trigger factors, like hypertension, cytotoxic agents or corticosteroid. [5,6]

CASE:

A 14-year-old girl presented with complaints of generalised oedema, headache, vomiting, blurring of vision, haematuria and oliguria. There was history of sore throat, fever 3-4 weeks back. On examination, blood pressure (BP) was160/100mmof Hg, pallor was present along with generalised oedema was noted. At admission patient had seizure Generalised Tonic Clonic followed by post-ictal drowsiness minutes. On CNS examination patient was drowsy, a Glasgow coma scale (GCS) 13/15, no s/o meningitis present. Signs of raised intra cranial tension were present. Fundus examination revealed papilledema. Laboratory investigations showed a haemoglobin of 10.6gm%, total protein was 5.1mg/dl and albumin 2.1mg/dl. Serum creatinine was 0.7mg/dl. Urinalysis revealed urine protein of 3+ (300mg/dL) and haematuria. Spot urine protein to creatinine ratio (Up/Uc) was 3.89.Abdominal ultrasound showed bilateral increased cortical renal echogenicity. In view PSGN, ASO and C3 were sent and C3 was decreased 80.7mg/dland ASO titre 236.92 (increased).

As the child presented with acute glomerulonephritis (AGN) with hypertensive emergency, was started on depine retard (0.3mkd). To control raised intracranial pressure symptoms mannitol were given; which also helps to decrease blood pressure. Seizures were controls by injection levetiracetam. Hydration was maintained with iv fluid therapy.

Computed tomography of brain revealed multiple ill-defined non enhancing hypodensities seen involving frontoparietal and left occipital regions due to corticomedullary oedema suggestive of PRES.[Figure 1]With current management patient improved and got discharged on day 10 after admission.

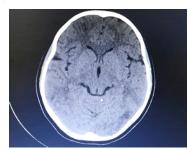


Figure 1. Computerised Tomography of brain showing multiple, ill defined, non-enhancing hypodensities involving bilateral frontoparietal and left occipital region due to corticomedullary oedema

DISCUSSION:

The clinical manifestations of PRES include headache, nausea and vomiting, altered mental status, decreased alertness, seizures, cortical blindness, and transient motor deficits. In the patients with PRES, seizures are common at the onset of neurologic symptoms but can also develop later. The seizures are usually GTC type and multiple. Temporary restlessness and agitation may alternate with lethargy. Stupor and coma may develop. The patients are often confused and there may be some abnormalities of vision such as hemianopia, blurred vision, and cortical blindness. [5,7]

In a study about PRES, Kwonet et al. [5] reported 12 patients who presented with seizures (42%), visual disturbances (33%), headache (17%), or altered mental status (8%). In all of their patients, most common clinical features as seizure (6/9), headache (6/9), and altered consciousness (4/9). The other symptoms were nausea and vomiting and blurred vision.

Many predisposing factors have been proposed including hypertension, immunosuppressive drugs, eclampsia, and renal dysfunction. Hypertension has often been emphasized as a common feature of PRES-associated conditions. Onder

et al. [4] detected hypertensive crisis as the most common trigger of PRES in 59%. Although hypertensive encephal opathy is the most common cause of PRES, cases have occurred in the absence of severe hypertension. Considering the rare frequency of arteriosclerosis and good plasticity of vessel walls in children, the vulnerability of vessel walls to hypertension was decreased in childhood.[8] D. N. Gera et al,[9] in study including 11 patients with PRES; Initial cranial imaging revealed fairly symmetrical areas of oedema not only involving occipito-parietal white matter regions in majority of cases but also extended to cortical gray matter in nine cases (81%) and extended to fronto-temporal area in five cases (45%), involved basal ganglia also in two patients; whereas cerebellum and medulla were also involved in one patient.

PRES related to hypertension might be due to sudden elevation of blood pressure causing disruption of the autoregulatory mechanisms of the central nervous system vasculature, leading to vasoconstriction and vasodilatation, and breakdown of the blood-brain barrier. Hypertensive encephalopathy following post-streptococcal glomerulone phritis (PSGN) is a known but uncommon manifestation in childrenand developing PRES in such a situation is very rare.

CONCLUSION:

It is important to consider diagnosis of PRES in children presenting with seizures and altered sensorium in anappropriate clinical setting. If recognised and treated early the clinical syndrome resolves within a week. High index of clinical suspicion and prompt treatment can reduce morbidity, mortality and helps in early recovery.

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