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Case Report

Post Infectious Glomerular Nephritis (PIGN) leading to Posterior Reversible Encephalopathy Syndrome (PRES); an uncommon presentation.

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Abstract

Posterior Reversible Encephalopathy Syndrome (PRES) is an acute encephalopathy characterized by headache, altered consciousness, visual symptoms and convulsions. Diagnosis is supported by Magnetic resonance imaging (MRI) of the Brain. Here we report a case of PRES in a 24-year-old-male who showed complete recovery with the control of blood pressure. Finally Post Infectious Glomerular Nephritis (PIGN) was found to be the underlying etiology of hypertension to develop PRES.

Introduction

PRES is characterized by headache, altered consciousness, visual symptoms, convulsions and symmetrical subcortical white matter vasogenic edema predominantly in posterior cerebral hemispheres (1). Hypertensive crisis is the common cause for PRES (2). Developing hypertensive encephalopathy and PRES following PIGN is known but uncommon presentation.

Key words

PIGN, PRES, MRI-Brain

Case presentation

A 24-year-old male was transferred from peripheral hospital to emergency unit with the history of two episodes of generalized tonic clonic seizures with loss of consciousness. He was drowsy. His Glascow Coma Scale was 13/15 (Eye-4, Verbal-4, Motor-5). He did not have signs of meningeal irritation. His limited neurological examination was normal including ophthalmic fundoscope. Bilateral ankle oedema with infected lower limb eczema and periorbital oedema were noted. His blood pressure was 180/120 mmHg. Rest of the examination was unremarkable.

When further questioning from the sibling, we found that he had chronic lower limb eczema for more than 2 years. He was fever free, but he complained headache with intermittent vomiting for one week and treatment was taken at peripheral hospital. There were no visual symptoms neither urinary symptoms, but urine output was low. There were no other systemic symptoms. He occasionally takes alcohol. There was no history of smoking, drug abuse and high-risk sexual behavior. He was having allergy to tomatoes and prawns.

His urinalysis revealed moderately field full red cells with dysmorphic red cells of 75% suggestive of glomerular origin, and mild proteinuria. His serum creatinine was 118micromol/L with blood urea of 8.9mmol/L and normal electrolytes. He had mild leukocytosis with neutrophil predominance. His CRP was 57mg/L and ESR was 32mm/1st hour. ANA was negative. His blood and urine cultures revealed no growth. Non contrast computed tomography (NCCT) of brain revealed bilateral occipital white matter hypodensity. He was further subjected to MRI, which showed high signal intensities on T2 weighted and Fluid-attenuated inversion recovery(FLAIR) images in the subcortical white matter of bilateral parietal, occipital, cerebellar hemispheres and dorsal aspect of pons and normal intensities in Diffusion-weighted imaging(DWI) consistent with the radiological diagnosis of PRES (Figure 1). Cerebral Magnetic Resonance Angiography revealed no abnormalities to suggest vasculitis. His CSF analysis was within normal limits. His anti streptolysin O titre (ASOT) was 400 U/ml(<200 U/mL).

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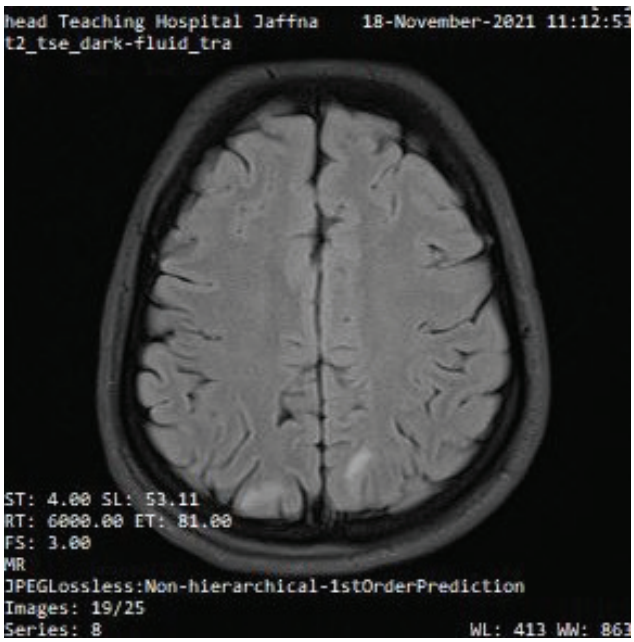

Figure 1: MRI Brain showed high signal intensities on T2, FLAIR images in parieto-occipital regions bilaterally

He was diagnosed to have PRES due to PIGN following lower limb skin sepsis. His blood pressure was well controlled with oral amlodipine. Antiepileptics were not started as he was fits free. Oral phenoxy methyl penicillin was commenced to eradicate the remaining infection.

He made a complete neurological recovery. His serum creatinine came down to 74micromol/L during ward stay. He was discharged with the antihypertensive and followed up at clinic. Dermatology follow-up also arranged for the management of eczema. After 3 weeks, his repeat urinalysis was completely normal and antihypertensive was gradually tailed off.

Discussion

PRES is a neurological condition, primarily characterized by headache, altered consciousness, visual symptoms and convulsions clinically and vasogenic oedema primarily in subcortical white matter areas, predominantly in posterior cerebral hemispheres (parieto-occipital regions), also in cerebellum and brainstem radiologically (1). Hypertensive crisis is the commonest cause but also occurs with cytotoxic immuno suppressive therapy, renal disease and auto immune disorders (2). Children are more vulnerable even in the low blood pressure.

Pathogenesis is unclear, but appears to be related to disordered cerebral auto regulation and endothelial dysfunction leading to brain hyper perfusion and break down of the blood brain barrier allowing extravasation of fluid and blood products in to brain parenchyma (3, 4). Prevalence of PRES following PIGN is known but rare and only few cases reported. (4).

Neuro imaging is mandatory in the diagnosis of PRES. It usually reveals white matter oedema typically in both posterior cerebral hemisphere. But relative sparing of calcarine and paramedian parts of occiptal lobes and cortical gray matter help to distinguish from infarction. MRI typically shows high signal intensities on T2 weighted images and FLAIR. Hypo or iso intense signal on DWI helps to differentiate from basilar stroke which causes hyper intensity (5, 6).

It is typically reversible condition but can lead to true ischemia and infarction or intracranial hemorrhage if left untreated. Treatment depends on aetiology, but commonly due to hypertension. Successful treatment causes resolution on neuro imaging within days to weeks (6).

Conclusion

PRES should be considered in hypertensive patients who have headache, altered consciousness, visual symptoms and convulsions.

MRI-Brain helps in prompt diagnosis and treatment.

Consent

Consent was taken from patient for publication.

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