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Case Report

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Post-streptococcal glomerulonephritis complicated with reversible posterior leukoencephalopathy syndrome

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Abstract

Introduction: Reversible posterior leukoencephalopathy syndrome(RPLS) is a clinical condition with headache, altered behavior, visual symptoms and seizures. It is associated with characteristic neuroimaging which shows vasogenic edema at occipital and parietal region commonly. Post-streptococcal glomerulonephritis(PSGN) is uncommonly associated with RPLS.

Case Presentation: We report a case of 15 years old boy who presented with recurrent seizure and progressive visual impairment ten days following a sore throat, subsequently diagnosed to have PSGN with MRI evidence of RPLS. He was commenced on oral phenoxymethylpenicillin and oral verapamil. Repeat MRI after six weeks showed complete resolution of previous changes. This is a rare case of RPLS associated with PSGN.

Keywords: Reversible posterior encephalopathy syndrome (RPLS), Post streptococcal glomerular nephritis (PSGN), Transient Visual loss

INTRODUCTION

Reversible posterior leukoencephalopathy syndrome (RPLS) is a clinico-neuro-radiological condition with symmetrical subcortical vasogenic oedema in occipital and parietal regions[1]. We describe a young boy who developed RPLS as a result of hypertension secondary to post-streptococcal glomerulonephritis (PSGN).

CASE REPORT

A 15-year-old boy was transferred from a peripheral hospital with a history of seizures and sudden onset loss of vision. He had been well 10 days prior to admission, when he had developed sore throat with pain and fullness of the left ear. His mother noticed mild swelling of face and bilateral ankles two days prior to admission. On the tenth day, he developed two witnessed generalized tonic-clonic seizures (GTCS). On admission he developed two more GTCS. He was noted to have a blood pressure of 150/90 mmHg for which he had been kept under observation. He had bilateral early papilloedema with rest of the central nervous system (CNS) examination being normal. Later on his vision had got worse gradually. Patient was transferred to the tertiary care hospital for MRI.

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Serial examination of vision showed that he had blurring of vision which progressed eight hours later to worsening of visual acuity (VA) to "hand movements only" bilaterally. On examination at the tertiary care center, there was mild hyperemia in the throat without pustules. Both ears were normal. He had mild pedal oedema. There was no neck stiffness, and Kernig's sign was negative. His heart rate was 80 bpm with a blood pressure of 140/90 mmHg, respiratory rate of 12 /min and an oxygen saturation of 100% on air. Lung fields were clear. There was no nystagmus, with normal pupillary light reflexes bilaterally. He had early papilledema. Visual acuity was low bilaterally. Rest of the nervous system examination including motor and sensory examination of limbs was normal.

CSF was found to have normal cell counts, protein and glucose. Urine full report revealed red cells with 70% dysmorphic cells and a subnephrotic range proteinuria (UPCR-1025 mg/g). Serum creatinine was normal. Serum C3 level was low 27.9 (90-180) mg/dl and C4 level was normal at 24.1(10-40) mg/dl. Anti-streptolysin O titre (ASOT) was very high with a value of 800 IU/ml. MRI brain showed bilateral symmetrical cortical and subcortical T2/FLAIR high signal intensities in the posterior parietal and occipital regions which suggested a diagnosis of RPLS. (Figure 1)

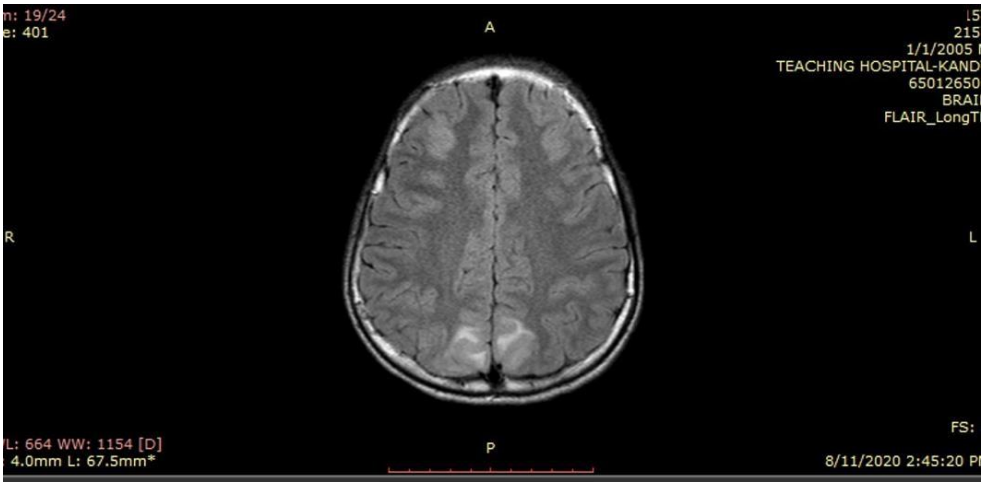

Figure 1: MRI brain showed bilateral symmetrical cortical and subcortical T2/FLAIR high signal intensities in the posterior parietal and occipital regions which suggested a diagnosis of RPLS

Absence of ANA, dsDNA ruled out autoimmune pathology. C-ANCA, P-ANCA and cerebral MRA revealed no abnormalities to suggest vasculitis.
He was commenced on oral phenoxymethylpenicillin 500mg which was given 6 hourly to eradicate any remaining streptococci. BP was controlled with oral verapamil. Examinations of VA fourteen hours later showed marked improvement to "counting fingers" but he was unable to see colours distinctly. Twenty-four hours later there was further improvement with Snellen VA reaching 6/12 and clarity of colour returning to normal. Repeat assessment after one week using the Snellen chart showed it to be 6/6 bilaterally.

After six weeks his visual acuity returned to 6/6, complements normalized (C3-104, C4-28.4) Urine full report revealed trace albumin and UPCR 138 mg/g. MRI brain done eight weeks later showed complete resolution of previous changes. Considering the entire clinical picture, a diagnosis of hypertension associated with post-streptococcal glomerulonephritis (PSGN) complicated with RPLS was made.

DISCUSSION

RPLS is a clinico-neuro-radiological condition with headache, altered behavior, visual symptoms and convulsions[2]. Heterogenous etiologies include high blood pressure, eclampsia, renal impairment, vasculitis being most commonly reported[3,4]. Cyclosporin is well known cause

of drug induced RPLS[5]. The prevalence of RPLS following PSGN is not known. Only few cases have been reported with FSGS[6].
The pathophysiology of RPLS is unclear and it appears to be related to auto- regulatory failure and hypertension leading to brain hyper perfusion resulting in breakdown of the blood-brain barrier, allowing extravasation of fluid and blood products into the brain parenchyma[5].

Neuroimaging is mandatory to diagnose RPLS. MRI usually reveals high signal intensities in T2- weighted FLAIR sequences[1,4]. Common radiological presentation is posterior cerebral oedema with sparing of calcarine and paramedian parts of the occipital lobe[1]. Diffusion-weighted imaging (DWI) helps distinguish RPLS from infarct. MRI will demonstrate complete resolution within few weeks. Treatment of RPLS depends on the etiology, among which hypertension is commonly seen.

RPLS should be considered as a possibility in a patient with PSGN having visual symptoms, seizures with elevated blood pressure. Most of the cases were associated with GTCS[3]. Children are vulnerable even in lower levels of blood pressure[4,9]. Early MRI with T2-weighted FLAIR and DWI sequences help in the diagnosis. Prompt diagnosis of precipitant and targeted treatment including antihypertensives is vital to prevent progression to irreversible brain damage. Unattended hypertension and prolong seizures may result in permanent neurological deficit[9]. There were few incidences of permanent infarct. Successful treatment may result Complete resolution of neuroimaging which is characteristic of RPLS as in our case[10].

CONCLUSION

RPLS, as a possibility, should be considered in patient with PSGN who has visual symptoms, seizures and elevated blood pressure. Children are vulnerable in lower levels of blood pressure. Early MRI with T2 FLAIR, DWI sequences helps in prompt diagnosis and treatment.

Abbreviations

RPLS: Reversible posterior encephalopathy syndrome
PSGN: Post streptococcal glomerularonephritis
NCCT: Non contrast computed tomography
MRI: magnetic resonance imaging
MRA: Magnetic resonace angiography
VA:visual acuity
SOT: antistreptolysin O titre
DWI: diffusion weightened images
ANA: anti-nuclear antibodies
P-ANCA: myeloperoxidase antineutrophilic cytoplasmic antibodies
C-ANCA:proteinase 3 antineutrophilic cytoplasmic antibodies
ESR-Erythrocyte sedimentation rate
LP: lumbar puncture

Author declaration
Author contributions
KK wrote the case report, drafted and edited the manuscript, ILMS contributed to write the case report and edited the manuscript, SA contributed to write the case report and edited the manuscript and revised final manuscript.

Conflict of interest
There is no conflict of interest

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Consent to participate
Informed written consent was obtained from the patient and mother for publication of this case report

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