

Posterior Reversible Encephalopathy Syndrome (PRES) Secondary to Severe Pneumonia

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Abstract

Posterior reversible encephalopathy is a rare neurological disease. The association between Posterior reversible encephalopathy syndrome and severe pneumonia is not well described in the available medical literature. We describe a case of a 51 year old female presented to our hospital with sudden loss of vision in both eyes in the background history of pneumonia who recovered completely with treatment. This case report elaborates the very rare entity of posterior reversible encephalopathy syndrome secondary to severe pneumonia.

Keywords: Neurological manifestations; Posterior reversible encephalopathy syndrome; PRES; Severe pneumonia

Introduction

Posterior reversible encephalopathy syndrome is also referred as PRES. PRES was first described in the medical literature in 1996. It is a distinct clinical condition, but considered as a stroke like syndrome due to sharing some of the clinical features of stroke [1,2]. PRES can be resulted due to many conditions including acute hypertension, autoimmune conditions, drugs, chronic inflammatory conditions and severe infections [1]. Usually PRES has an excellent outcome once the causative factor is controlled. Neurological involvement of pneumonia is already known fact. Especially atypical pneumonia caused by Mycoplasma pneumonia and Legionella pneumophila are more potential to associate with classic neurological manifestations such as meningoencephalitis, aseptic meningitis, myelitis, cranial nerve palsies. But severe pneumonia causes PRES as a neurological sequelae is a very rare association [3].

Case Presentation

A 51 year old female presented with sudden loss of vision in both eyes for 4 hours. No pain or redness of the eyes was reported. She had a five day duration of febrile illness with cough, nasal stuffiness and runny nose prior to this presentation. She was treated by a general practitioner with oral azithromycin 500 mg once a day for 3 days. On the fifth day of the illness, she reported loss of vision by anecdotal evidence stated by her husband. It was mentioned that she was not able to see anything around 9 am in a well lit room when she tried to move around in the house. The situation did not improve when she was assisted to be outdoors. Interestingly, her primary complaint at the moment was weakness rather than loss of vision as she sought for mobilizing assistance in spite of absence of limb weakness or unsteadiness. Her speech was slurred. She did not experience any similar events in the past. She denied headache, giddiness, vertigo or vomiting. Her consciousness was preserved and oriented. She had no

seizures. No preceding chest pain or palpitation was noted. She could not recall any trauma to head and neck. She did not suffer from arthralgia, rashes or oral ulcers. She didn't complain any constitutional symptoms like anorexia, malaise or weight loss. No significant bleeding tendency was noted in the history. Her first pregnancy was uneventful. She had pregnancy induced hypertension during her second pregnancy 7 years back. She had undergone 2 caesarean sections at 37 and 36 weeks during her two pregnancies respectively. She took antihypertensive medications until 2 years following the birth of the second child. She did not follow any outpatient clinics thereafter. Family history was not significant except the fact that her mother had a stroke at the age of 63 years.

Her physical examination indicated Glasgow coma scale score (GCS) was 15/15. She was mildly confused and afebrile on admission. Visual acuity was limited to the perception of light in both eyes (PL-both eyes). Visual field could not be tested. She had preserved direct and consensual light reflexes. Accommodation reflex was unable to perform. Fundoscopy revealed a normal retina, discs and a vascular appearance. Other cranial nerves were clinically intact.

Motor system and sensory system examination revealed normal findings. She was tachycardic with pulse rate of 116 bpm and regular in rhythm. Blood pressure was recorded as 180/100 mmHg. There were no murmurs or bruits heard on auscultation. Respiratory rate was 22 breath cycles per minute. Her oxygen saturation in ambient air was 73% and 85% with 5 litres of supplementary oxygen. Lung auscultation revealed vesicular breathing with bilateral crackles (more prominent in left side) and rhonchi.

Non contrast Computer tomography (CT) brain was done on admission as shown in Figure 1. It showed hypodensified areas in both occipital lobes and right posterior temporal lobes suggestive of an infarction. CT venogram concluded the absence of cerebral venous sinus thrombosis. Magnetic resonance imaging (MRI) of brain was done after 2 weeks and it revealed normal bilateral occipital lobes which supported the diagnosis of PRES.

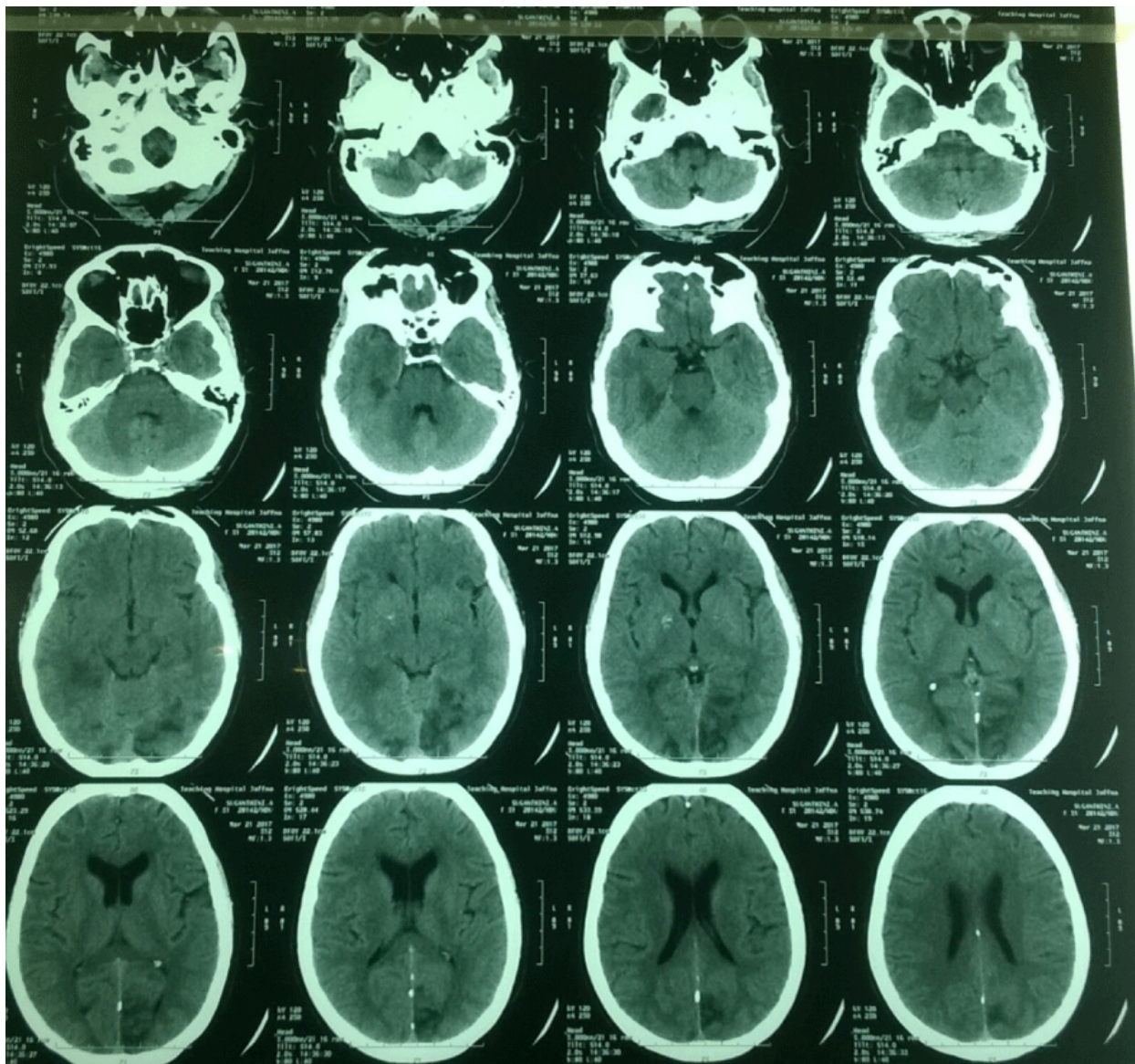


Figure 1: NCCT brain scan which was done on admission illustrated hypodensed areas in bilateral occipital lobes and right posterior temporal lobes.

She had been evaluated for possible thromboembolism. Electrocardiogram recorded a sinus rhythm and no significant left ventricular hypertrophy noted. Cardiac sources of thromboembolism were excluded by a 2D echocardiogram. Her clotting profile was within the normal limits. Arterial blood gas analysis was consistent with hypoxaemic respiratory failure. Arterial blood gas analysis showed pH 7.38 (7.35-7.45), $p\text{CO}_2$ 29 mmHg (35-45), $p\text{O}_2$ 60 mmHg (80-100), HCO_3^- 19.8 mEq/l (22-26), BE-7.9. Complete blood count was in the normal limits including white blood cells $8.65 \times 10^9/l$ (4.00-10.00), neutrophils 63.1% (40-80), lymphocytes 23.5% (20-40), monocytes 9.4% (2-10), eosinophils 3.5% (1-6), basophils 0.5% (0.0-2), haemoglobin level 13.7 g/dl (13-16), platelets $275 \times 10^9/l$ (150-410). C reactive protein level was 19.2 mg/dl (<6) and ESR 65 mm/first hour. Blood was negative for influenza antigen in reverse transcription

polymerase chain reaction. Antinuclear factor was within the normal range.

Clinical diagnosis of Posterior reversible encephalopathy syndrome (PRES) was made. Patient's high blood pressure was treated with oral losartan 50 mg twice a day. On the following day patient became more dyspnoeic and developed respiratory distress. Patient was admitted to the intensive care unit and she did not require endotracheal intubation and ventilation. She was given oral oseltamivir 75 mg twice a day for 5 days and intravenous ceftazidime 1 g twice a day for 10 days according to the sensitive pattern of sputum culture which was positive for *Pseudomonas* species. Her visual loss slowly improved and visual assessment was performed periodically. Patient was continued to be tachycardic and had persistent high blood pressure which prompted us to upgrade antihypertensives with oral hydrochlorothiazide 25 mg once a

day and oral nifedipine 20 mg twice a day. Over the next few days, patient's clinical condition was improved gradually as shown in Figures 2 and 3. She was discharged home on the 16th day of the illness and her corrected visual acuity in both eyes 6/24 with N14 near vision and had a right side homonymous hemianopia. Chest X-ray showed a resolution of inflammatory shadows.

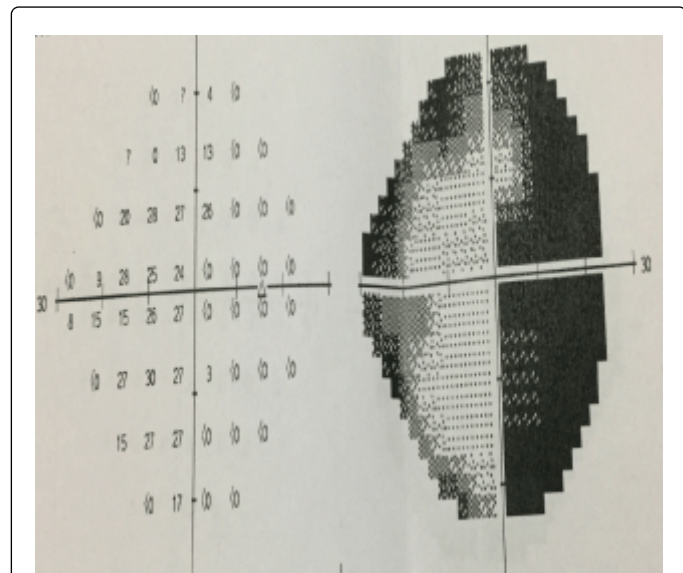


Figure 2: Right side visual field of the patient two weeks after admission.

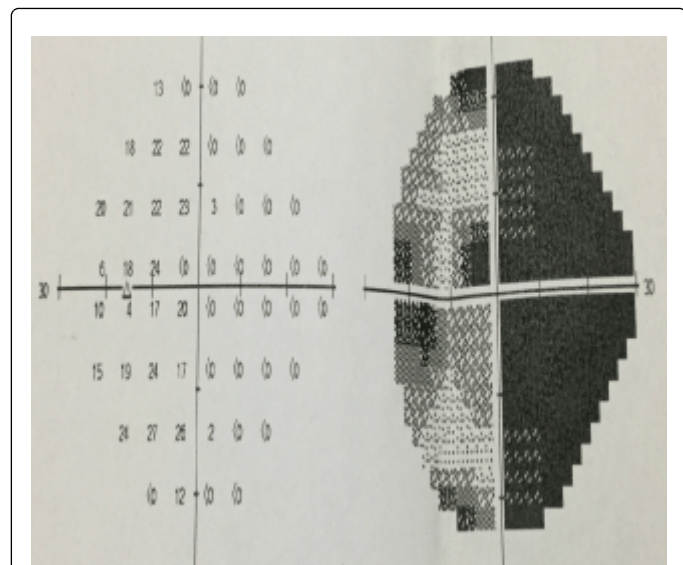


Figure 3: Left side visual field of the patient two weeks after admission.

Discussion

PRES is a relatively new terminology in the field of medicine as it was first described in 1996. PRES is increasingly recognized in the current practice which is believed to be due to the evolution of modern neuroimaging facilities [4,5]. During the last two decades, MRI scan

has allowed for increased number of case detection in PRES [6]. Common MRI findings of PRES are bilaterally symmetrical subcortical or cortical hyperintensity in T2 weighted images and the evidence of vasogenic oedema [4,6]. Among the reported case of PRES, its common presentations included confusion, seizures, visual impairment, headache, vomiting [2]. However PRES is a complex clinical condition which is not fully understood yet. PRES has been noted to have a good clinical outcome. Thus PRES has numerous associations; it has been proved that acute hypertension has an immense impact on PRES [6]. Toxins, drugs, eclampsia, autoimmune diseases, malignancies are also known as causative factors [1,2,7]. Although there have been reported cases with PRES and severe infection and sepsis, there are no definitive cases found in the literature in order to severe pneumonia and PRES [7]. Infection related PRES was 8-24%. When PRES is associated with sepsis, it is more likely to be due to gram positive bacteria preferably by gram positive cocci [7]. We assume that PRES in a case with infection caused by gram negative bacteria is very rare. *Mycoplasma pneumoniae*, *Legionella pneumophila* are the commonest bacteria which are more capable of causing numerous extra pulmonary manifestations included neurological syndromes such as meningoencephalitis, aseptic meningitis, cerebellitis, cranial nerve palsies, polyradiculopathy [3]. But severe pneumonia causes PRES as a neurological consequence is not found in the current medical literature. Neurological manifestations of pneumonia are frequently categorized under two groups such as neurological symptoms which occur parallel with respiratory symptoms and neurological symptoms which appear later following a gap [3]. Mechanisms of underlying PRES in pneumonia and sepsis have not yet been elucidated [4,7]. But there are some theories which have been used to explain this association. The postulated theory of infection, sepsis, shock and associated PRES would also explain the pathogenesis of pneumonia and PRES. The pathophysiology of PRES is multifactorial in sepsis. Body response to any bacterial infection is mediated by both exotoxins and endotoxins [1,7]. These chemical mediators are also strong stimulants to initiate an inflammatory response. Vascular instability, direct invasion, neurotoxicity are complex mechanisms of pathophysiology in PRES in sepsis. Endothelin 1 is one of the very potent vasoconstrictor which also plays a key role [6].

This case had a severe pneumonia and the diagnosis is supported by clinical findings, hypoxaemic respiratory failure, extensive chest X-ray shadows, high inflammatory markers. Antibiotic treatment prior to admission would have resulted normal complete blood count. This patient presented during the period of seasonal influenza infection was its peak. But it could not be proved as influenza antigen became negative. However the underlying untreated hypertension also thought to be contributed to PRES in this case or may be coincidental.

According to the MRI scan of brain which was done two weeks following the occurrence of symptoms had a resolution and clinical improvement would strongly suggest the diagnosis of PRES. It took 2 to 3 weeks for the patient's vision to be restored. She followed the medical clinic and attended regular ophthalmological assessments. She had a good adherence to treatment too. Four weeks after the initial presentation, she resumed her previous job as a lecturer.

This case prompts clinicians to consider PRES in the clinical practice, even in a case with pneumonia and the importance of early diagnosis. This will be the first case report with PRES in severe pneumonia responded effectively with treatment. Still more evidence is needed in the future in regards to this association.

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