

## Case Report

# A case of post-splenectomy posterior reversible encephalopathy syndrome: a reversible event

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### ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a disease characterized by features of clinical and neuroimaging findings. The basics of recognizing the entity include a combination of clinical features and radiological findings in the presence of triggering factors. Clinically the patient can have various presentations like nausea, vomiting, blurring of vision, seizures, altered consciousness, transient motor deficits, or cortical blindness. Radiologically the findings can be picked up by computed tomography (CT), magnetic resonance imaging (MRI), or diffusion-weighted imaging. The usual factors that can trigger the PRES include blood pressure fluctuations, eclampsia/preeclampsia, renal failure, cytotoxic agents, and autoimmune conditions. With vigilant suspicion and prompt treatment, patients can be saved from serious complications. Here we wish to report a case of a 22-year male patient who developed PRES syndrome after undergoing an emergency splenectomy and recovered with conservative medical management for the condition. This syndrome can be managed well with an early diagnosis which needs keen suspicion and prompt treatment.

**Keywords:** Post-splenectomy, PRES syndrome, Posterior reversible encephalopathy syndrome

### INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a disease characterized by features of clinical and neuroimaging findings. In the literature Hinchey in the year 1996 has described this for the first time.<sup>1</sup> The basics of recognizing the entity include a combination of clinical features and radiological findings in the presence of triggering factors. Clinically the patient can have various presentations like nausea, vomiting, blurring of vision, seizures, altered consciousness, transient motor deficits, or cortical blindness.<sup>2</sup>

Radiologically the findings can be picked up by computed tomography (CT), magnetic resonance imaging (MRI), or diffusion-weighted imaging. It is characterized by vasogenic oedema involving the cortical/subcortical regions which are bilateral affecting the parietal and

occipital regions but atypically there can be involvement of other regions, cortical involvement, restricted diffusion, contrast enhancement.<sup>3-5</sup> The usual factors that can trigger the PRES include blood pressure fluctuations, eclampsia/preeclampsia, renal failure, cytotoxic agents, and autoimmune conditions.<sup>6,7</sup> Several other etiological factors and features have been recently recognized. Early recognition of the condition is essential for prompt treatment and reversibility.<sup>7</sup>

Here we wish to report a case of a 22-year male patient who developed PRES syndrome after undergoing an emergency splenectomy.

### CASE REPORT

A 22-year-old male presented to our emergency room with a history of stab injury by a hard and sharp object over the

left flank region through which the omentum was seen herniating. The patient was conscious and well oriented to time and place. His pulse rate was 118 beats per minute, blood pressure measured over his right arm in supine position was 90/60 mm of Hg. The examination of the injured site revealed an incised wound of approximately 05×02 cm of peritoneal depth through which the omentum was herniating. The patient was immediately taken to the operating room and explored through a midline incision.

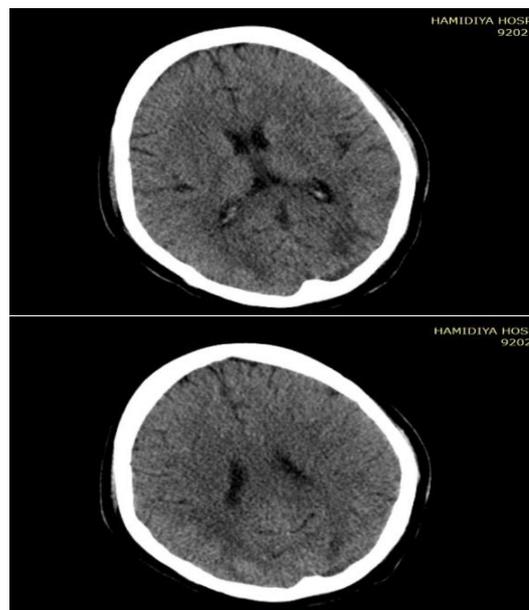
Intraoperatively there was a massive hemoperitoneum of around 2.5 litres and grade 4 splenic injury. Emergency splenectomy was carried out. Along with fluid resuscitation, 2 pints of packed red cells and 4 pints of fresh frozen plasma were transfused intraoperatively for haemodialytic stability. The entire procedure was carried out in 3 hours and the patient shifted to surgical intensive care unit (ICU) on ventilatory support with vitals of 120 beats per minute of pulse rate, blood pressure of 128/58 mm of Hg on inotropic support, and 100% saturation.

On postoperative day 1 (POD-1) patient continued to be on ventilatory support with post-operative haemoglobin of 6 g/dl, leucocyte count of 5200, platelets of 2 lakhs/cumm. With serum electrolytes within normal laboratory limits. Further transfusion of 2 pint-packed red cells and 3-pint fresh frozen plasma was done. The patient was given intravenous calcium gluconate to neutralize the post-transfusion chelating effect.

On the next day (POD-2) patient was vitally stable and was planned to wean off the ventilatory support. Taken on synchronized intermittent mandatory ventilatory (SIMV) mode and patient was maintaining saturation of 100% with stable vitals. There was a sudden shoot-up of the patient blood pressure on next morning at 4 am up to 240/120 mm of Hg followed by an episode of convulsion for 4 minutes, which was managed by giving an intravenous midazolam injection. Later the patient was sedated and shifted to the assist control mode of ventilation. The patient's vitals settled and came within normal physiological limits within 30 minutes. The patient's non-contrast computed tomography of the head was done which showed parietooccipital white matter oedema (Figure 1).

On day 3 of post-surgery when the patient was weaned off from the mechanical ventilation and taken on T-piece ventilation, he tolerated well initially for 15 minutes, and then the blood pressure went up to 180/100 mm of Hg and he threw another episode of convulsion. The patient was given a loading dose of injection phenytoin and shifted to assist control mode of ventilation. Anti-convulsion medications started and medications to reduce intracranial pressure were started. After stabilization of the vitals, the patient was weaned off and extubated the next day. Post extubation he complained of nausea, giddiness, and headache. The patient was closely monitored for any further episodes of convulsions. Anticonvulsants were continued. All the haematological parameters were found to be within normal limits. We got a computed tomography

of the head to evaluate the cause of convulsions. No intracranial abnormalities could be identified other than minimal resolving parieto-occipital white matter oedema.



**Figure 1: Computed tomography of the head showing parietooccipital white matter oedema.**

## DISCUSSION

PRES is a condition that needs high clinical suspicion and vigilance to diagnose for prompt treatment. It's a self-limiting condition with symptomatic treatment alone if treated early which can otherwise lead to serious neurological damage. There are two theories; which can explain the pathophysiology of PRES. The cytotoxic and vasogenic theories. The cytotoxic theory implies vasoconstriction and hypoperfusion as the cause of brain ischemia and subsequent vasogenic oedema. Other theories state that weak cerebral autoregulation and endothelial dysfunction cause the breakdown of blood brain barrier (BBB) and vasogenic oedema.<sup>8</sup>

PRES has been observed in hypertensive patients but has been documented in normotensive patients as well. The following risk factors have been identified with PRES, the abrupt elevation of blood pressure, impaired renal function, eclampsia/pre-eclampsia, autoimmune diseases, infections, and chemotherapeutic agents. Our patient was normotensive in all the pre-operative evaluations and showed abrupt elevation in blood pressure before each episodes of seizure and other symptoms.

Patients with PRES syndrome will present with varied presentations like seizures, visual disturbances, headache, and focal neurological deficits.<sup>10,11</sup> But often the symptoms are nonspecific and manifest acutely or subacutely over several hours or days.<sup>7</sup> However prolonged progression of symptoms over several weeks is uncommon proving its transient nature. Seizures being one

of the most common symptoms<sup>10</sup> can occur in various types. It includes generalized tonic-clonic type, focal type, and status epilepticus. The majority of the patient's seizures are terminated spontaneously or from the use of antiepileptic medications. It has been found that it is common to have serial episodes of seizures in the acute phase.<sup>12</sup> It is common to have provoked seizures from recurrent PRES or other provoking factors around the acute phase. Despite that the long-term risk of unprovoked seizures is infrequent and epilepsy is rare.<sup>13</sup> In our patient, there were two episodes of seizures serially on two consecutive days which were followed by an abrupt rise in blood pressure and were controlled by antiepileptic medications. Our patient had no further episodes of seizures even during one month of follow-up.

It has been well documented that early neuroimaging is vital for the diagnosis and proper line of management of the condition. Despite being termed posterior reversible encephalopathy syndrome, it can be found in the non-posterior distributions, watershed areas, frontal, inferior temporal, and brain stem areas. Although computed tomography can pick up vasogenic oedema in some patients, brain MRI, especially T2 weighted and fluid-attenuated inversion recovery (FLAIR) sequences are much more sensitive. At present, there is no gold standard diagnostic tests.<sup>6</sup> Cerebral oedema in these patients has been classified into different grades as mild, moderate, and severe.<sup>14</sup> Mild PRES was defined as cortical or subcortical white matter oedema without haemorrhage, mass effect, herniation, and minimal involvement of one of the groups – cerebellum, brain stem, or basal ganglia. In our patient Computed tomography was done after 24 hours of the last episode of seizure which revealed minimal brain oedema. The MRI would have been much more information that could not be done as the patient was on a ventilator in the post-operative period and after seizures.

Overall, the spectrum of clinical features, vasogenic cerebral oedema, and various risk factors are crucial in making the diagnosis of PRES syndrome.<sup>11</sup> It has a fairly rapid onset and spontaneous resolution. More than 90% of the patients show typical radiological and clinical findings.<sup>15</sup> Recently the PRES early warning scoring (PEWS) scale which consists of: risk factors, clinical features, and EEG features have improved the prediction and early diagnosis of PRES in suspected patients with a high index of suspicion in patients with a score of 10 points or higher.<sup>16-18</sup> Multidisciplinary hospital care involving a team of intensivists, anesthesiologists, pain physicians, neurologists, and treating doctors team is crucial for optimum management. Delay in management could lead to serious sequelae.<sup>19</sup> Early diagnosis with preferably MRI and prompt control of blood pressure is the key to managing these patients.<sup>20</sup>

## CONCLUSION

At present, the available data on PRES and its outcomes are from single institutional studies with a paucity of data

from long-term epidemiological studies. Its heterogeneous nature limits its ability to generalize the results. In general, many studies have concluded the favourable outcome of the condition. A systematic study that incorporates the clinical, etiological, serological markers, imaging features, with various comorbidities will be essential to look for future studies. There is still a need for randomized studies to better understand the disease course and follow-up for the adequate duration for concluding on the long-term post-PRES sequelae. Lack of keen suspicion of this syndrome in post-operative patients, delay in imaging studies due to various postoperative constraints can be the major setbacks in diagnosing PRES in postoperative surgical wards.

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