



Posterior reversible encephalopathy syndrome: a rare cause of seizures following non-transplant cardiac surgery

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is rarely reported as a cause for seizures following cardiac surgery. PRES in non-transplant cardiac surgery may reflect under-diagnosis and under-reporting. While the condition is reversible, a delay in diagnosis can lead to irreversible brain injury. We describe a case of PRES that occurred after aortic valve replacement with concomitant coronary artery bypass grafting.

Keywords Posterior reversible encephalopathy syndrome · CABG · Seizures

Introduction

Tonic–clonic seizures, or convulsive seizures, after cardiac surgery are uncommon and can result from electrolyte imbalances, hypoxia, medications like tranexamic acid, and infections [1–3]. They can also be an initial manifestation of a stroke. One of the most uncommon causes of seizures is the posterior reversible encephalopathy syndrome (PRES), which has been very rarely reported after cardiac surgery.

Case report

A 59-year-old hypertensive and diabetic female underwent coronary artery bypass grafting (CABG) and aortic valve replacement with a 17-mm Corcym-Bicarbon Slimline mechanical valve. Surgery was uneventful, but on the first postoperative morning, she developed refractory generalized tonic-clonic seizures and hypotonia. The systolic blood pressure at the time was around 200 mm Hg. Electroencephalogram (EEG) showed multiple non-specific spikes. A computed tomography angiography (CTA) showed no abnormalities, but magnetic resonance imaging (MRI) revealed

vasogenic edema in the brain (Fig. 1). The MRI finding was pathognomonic for the PRES. After establishing the diagnosis, aggressive control of the blood pressure was aimed for while continuing the antiepileptic medications. Once the blood pressure was controlled, and the systolic pressure maintained below 120 mm of Hg, the seizures stopped, the EEG returned to normal, and she was fully awake on the fifth post-operative day. She was extubated the next day and was found to have slurred speech and paraparesis and required intensive physiotherapy and speech therapy. She fully recovered over the next 2 weeks and was discharged on the 22nd postoperative day. Repeat MRI prior to discharge revealed clearance of previous lesions with few lingering gyriform hyperintensities in the superior and middle frontal gyri (Fig. 2).

Discussion

PRES is a type of hypertensive encephalopathy that exhibits specific imaging characteristics, with cerebral edema typically localized to the parieto-occipital regions. PRES is also an extremely rare cause of post-operative seizures. The term “reversible” refers to the fact that the symptoms of PRES resolve once the underlying cause is treated. It is typically associated with high blood pressure but can also be caused by medications like lidocaine, immunosuppression, and autoimmune disorders. While several reports exist of PRES after solid organ transplants [4], due to the inevitable use of immunosuppressive agents

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Fig. 1 Magnetic resonance imaging T2/FLAIR sequence showing gyriform areas of hyperintensity in the bilateral fronto-parietal and bilateral occipital cortex

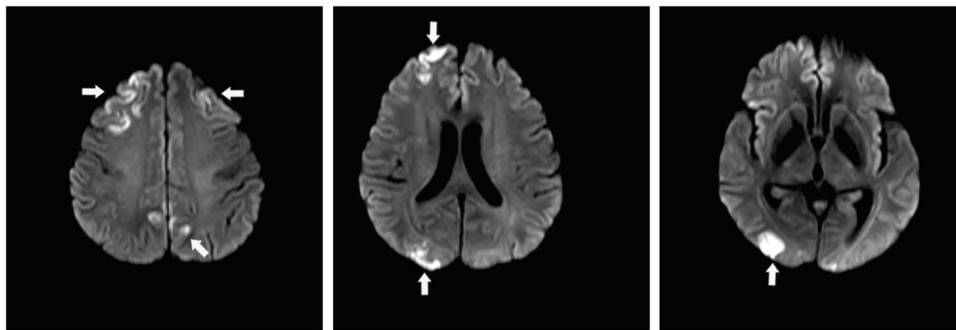
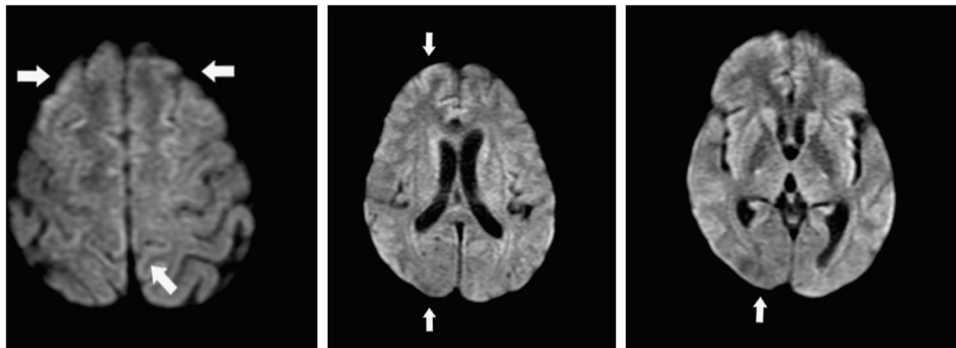


Fig. 2 Magnetic resonance imaging T2/FLAIR sequence showing resolution of previous changes



like tacrolimus, there has been only a solitary report of PRES in non-transplant cardiac surgery prior to this [5]. A PubMed search using the following search strategy “(Posterior reversible encephalopathy syndrome) AND (cardiac surgery) NOT (transplant) NOT (thoracic)” was used to search for cases. This resulted in nine reports, a careful inspection of which confirmed only one case of PRES in non-transplant cardiac surgery.

PRES can manifest across all age groups, encompassing infants to the elderly. However, its prevalence is notably higher among young and middle-aged adults, with an average age of 45 years. There is an observed tendency for a higher incidence of PRES in females, a trend that persists even when individuals with eclampsia are excluded from the analysis. Among adults, PRES is frequently diagnosed in cases related to eclampsia. In other medical conditions, it occurs in patients after bone marrow transplantation (2.7–25%), following solid organ transplantation (in 0.4–6%), and in individuals with end-stage renal disease or systemic lupus erythematosus (0.4 to 0.8%) [1].

While seizures are the presenting feature of PRES in the majority of cases (75–87%), the presentation can be more subtle and non-specific like headaches (26–53%) and visual disturbances (20–39%) that often lead to the condition being unrecognized [3]. Seizures manifest as generalized tonic-clonic seizures in most cases (54–64%) with status epilepticus reported in up to 17% of cases and partial seizures in up to 28% of cases.

Seizures after cardiac surgery are reported in nearly 1 in 200 cases. Following cardiac surgery, fluid administration, insulin therapy, diuretic use, and blood transfusion can result in several electrolyte and glucose imbalances [6]. Hyponatremia, hypophosphatemia, hypocalcemia, hypomagnesemia, and hypoglycemia can all precipitate seizures. Tranexamic acid administration is reported to be associated with a risk of seizures in 4.6% of cases, and certain β -lactam antibiotics can also cause seizures in the post-operative period. Besides, “post-pump encephalopathy,” which results due to multi-focal injury secondary to thrombo-embolism, is another important cause. Significant hypotension during cardiopulmonary bypass can lead to ischemic injury to the watershed areas of the brain, especially in those with carotid artery disease, and is another important cause of postoperative seizures [3]. Visual disturbance is another important manifestation of PRES. Common causes of visual disturbance after cardiac surgery include retinal artery occlusion and ischemic optic neuropathy. Less common causes include choroidal and vitreous hemorrhage, acute glaucoma, and cortical blindness. Most cases of PRES have normal appearing optic disc on fundoscopy or have features of co-existing hypertensive or diabetic retinopathy. Thus, in the absence of any obvious abnormality on clinical and fundoscopic examination in a patient with uncontrolled hypertension, an MRI would aid in diagnosis [7].

Although the pathophysiological changes underlying PRES are not clearly elucidated, failed cerebral

autoregulation, hyperperfusion, blood-brain barrier disruption, and breakdown vasogenic edema are the most commonly accepted explanations. The pathophysiology for developing the vasogenic edema in PRES is poorly understood, and several mechanisms have been proposed. T cell-mediated endothelial cell injury, failure of autoregulation, vasoconstriction with resultant hypoperfusion or hypoxemia, vascular endothelial growth factor expression, and increased endothelial permeability have all been proposed in the etiology of vasogenic edema [3].

Diagnosis of the condition requires a very high index of suspicion due to its rarity. Electroencephalograms are not diagnostic and can be completely normal in appearance [3]. As observed in our case, in nearly one out of five cases computed tomography scans can be essentially normal in PRES [8]. MRI is more sensitive and can reliably diagnose vasogenic edema that involves the subcortical white matter in PRES. There are three distinct patterns of PRES that have been described on imaging; however, knowledge of subtle variations is required for an accurate diagnosis. The main differential diagnoses for PRES include ischemic causes, venous infarction, trauma, and inflammatory conditions like vasculitis, encephalitis, and demyelinating disorders. The lack of history of trauma and features of traumatic brain injury on neuro-imaging can rule out trauma as a predisposing factor. PRES rarely involves just posterior circulation, whereas bilateral posterior cerebral artery distribution infarcts are rare in the absence of top of basilar thrombosis, which also affects other areas like thalami, midbrain, and superior cerebellum. Carotid duplex studies or CTA of the head and neck vessels are also useful in excluding ischemic causes of seizures following cardiac surgery. While normal venous structures on computed tomography (CT) or MRI rule out venous infarction, the distribution of lesions in vasculitis is less systemic and does not demonstrate the parieto-occipital dominance as seen in PRES [2].

The most important step in managing PRES is early diagnosis. While the condition is reversible, the delay in diagnosis can lead to PRES progressing to infarction or hemorrhage and may result in irreversible brain injury [3]. Extensive involvement, presence of intracranial hemorrhage, and involvement of the brainstem are poor prognostic markers [2].

Treatment after diagnosis is entirely supportive. Correction of any underlying metabolic abnormality and withdrawal of the noxious drug is essential. However, in the absence of any obvious metabolic disorder, as seen in our case, aggressive blood pressure control and concurrent anticonvulsant therapy are key. It is advisable not to lower the mean arterial blood in excess of 20% in the initial hour. Rapid reduction in blood pressure should be avoided especially in the elderly hypertensive patients. Intravenous antihypertensive drugs are preferable as they have a rapid onset

of action and are easily titratable. In the cardiac setting, intravenous labetalol and sodium nitroprusside are ideal. Oral antihypertensives should be initiated prior to discontinuing intravenous antihypertensives. Concurrent administration of anticonvulsants, though not evidence-based, is recommended by experts. However, these can be discontinued once the symptoms subside with a resolution of changes on radiological imaging [3]. The usage of intravenous mannitol, to reduce cerebral edema, has also been reported [9].

Conclusion

This case report highlights the rarity of PRES as a cause of post-operative seizures, particularly following cardiac surgery, where it often goes unrecognized. Early diagnosis is critical, and MRI is instrumental in diagnosing PRES. A normal CT scan in the presence of persistent neurological signs and symptoms would warrant an MRI scan. The condition is typically reversible, and timely intervention is paramount to prevent progression to infarction or hemorrhage. Treatment primarily involves addressing underlying metabolic abnormalities, withdrawal of causative drugs, and aggressive blood pressure control. Anticonvulsant therapy is often recommended during the acute phase. Despite its rarity, awareness of PRES is crucial in post-operative care, and a high index of suspicion is required for timely diagnosis and management, particularly in the absence of apparent metabolic disorders.

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Declarations

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