

Case report



POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME AND PREECLAMPSIA: CASE REPORT AND BRIEF LITERATURE REVIEW

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ABSTRACT

Purpose: Posterior Reversible Encephalopathy Syndrome (PRES) is a neurotoxic condition characterized by reversible subcortical white matter and cortical edema, commonly affecting the parieto-occipital regions. Pregnancy, particularly in the context of preeclampsia or eclampsia, represents a significant risk factor for PRES.

Material/methods: We report a case of a 26-year-old woman with monochorionic diamniotic twins who developed PRES in the immediate postoperative period following elective cesarean section at 36 weeks of gestation. Despite initial normal blood pressure and laboratory findings, she experienced sudden bilateral vision loss accompanied by confusion.

Results: MRI revealed symmetrical occipital cortical hyperintensities consistent with PRES. Intensive management, including antihypertensive therapy and close monitoring, led to full recovery of vision and normalization of MRI findings within one month.

Conclusion: This case underscores the importance of prompt recognition and early intervention in PRES, even in normotensive postpartum patients. Awareness of PRES in the context of pregnancy is critical, as timely diagnosis and management significantly improve clinical outcomes and reduce the risk of long-term neurological sequelae.

Keywords: posterior reversible encephalopathy syndrome, preeclampsia, complication,

BACKGROUND

Posterior Reversible Encephalopathy Syndrome (PRES) is initially described in 1996 as Reversible Posterior Leukoencephalopathy Syndrome. PRES represents a neurotoxic syndrome occurring due to the susceptibility of the posterior circulation to fluctuations in blood pressure [1]. Neuroimaging typically reveals symmetric parieto-occipital white matter edema [2].

Clinically, PRES has an acute onset and manifests with headache, nausea and vomiting, visual disturbances, seizures and altered mental status [3]. It is associated with a variety of underlying conditions such as hypertension, preeclampsia-eclampsia, renal failure, autoimmune disorders (e.g. systemic lupus erythematosus) and the use of certain immunosuppressive agents, including Cyclosporine, Cyclophosphamide and Methotrexate [4].

Pregnancy represents a particularly important clinical context. PRES is considered a key neurological component in the pathophysiology of preeclampsia [5]. Clinical manifestations may occur as early as the first half of pregnancy (before 20 weeks of gestation) or in later stages. Although most commonly seen in severe preeclampsia, PRES can also occur in normotensive pregnant women or in the postpartum period. In recent years, there has been increasing scientific interest in the close association between preeclampsia/eclampsia and PRES.

The incidence of PRES has increased, largely due to advancements in neuroimaging techniques and their wider availability. Prompt recognition of PRES is essential, as early diagnosis and treatment of the underlying cause often result in complete clinical and radiological recovery [6]. Although most patients experience full resolution of symptoms, delayed diagnosis or inadequate therapeutic intervention may lead to adverse outcomes, including increased morbidity and mortality [7].

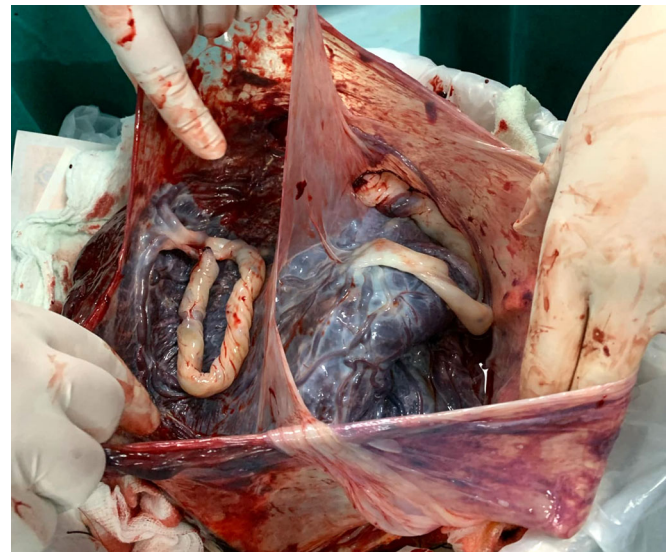
CASE DESCRIPTION

A 26-year old pregnant woman, G3P1, was admitted to the Clinic of Obstetrics and gynecology in UMHAT „Dr Georgi Stranski”, Pleven, for an elective Cesarean section at 36 weeks of gestation. The current pregnancy was spontaneously conceived with monochorionic diamniotic twins.

Her medical history included a right salpingectomy for a previous tubal pregnancy and a cesarean delivery performed one year later due to excessive myopia. Both intrauterine pregnancies were properly monitored and progressed without complications. At the time of hospital admission, the patient was asymptomatic and her laboratory findings were in normal ranges. The fetal monitoring indicated satisfactory fetal well-being. An uncomplicated cesarean section was performed, resulting in the delivery of monochorionic female twins weighing 2400 g and 2100 g, respectively. Monochorionicity was diagnosed by ultrasound detection of the T-sign in the first trimester and confirmed after delivery (Figure 1). The early postoperative period was uneventful, marked by stable hemodynamic parameters. However, approximately six hours after surgery, following initial mobilization, the patient experienced a sudden onset of bilateral vision loss. Hemodynamic status remained stable throughout the event, with normal blood pressure values and no significant abnormalities in laboratory findings. Given the patient's history of myopia, an ophthalmologic evaluation was promptly performed. Ophthalmological examination revealed the absence of light perception, pupils reactive to light, and a normal macula. Thereafter, a neurological assessment identified early alterations in mental status, characterized by confusion and disorientation. Magnetic resonance imaging was subsequently performed. Radiologists described symmetrical cortical hyperintense signals in the occipital lobes of both hemispheres, corresponding to white matter edema. The patient was immediately transferred to the intensive care unit (ICU) for close monitoring and active management. Upon admission, continuous monitoring of vital signs revealed a sudden increase in blood pressure, reaching 170/100 mmHg. A cardiology consultation was obtained, and antihypertensive therapy was initiated with a calcium channel blocker, labetalol, and hydralazine. The therapeutic target was to maintain a mean arterial pressure between 105 and 125 mmHg, which was achieved through continuous intravenous infusions. Anticoagulant prophylaxis was also started. During the first night in the ICU, the patient developed agitation accompanied by confusion, necessitating the administration of sedatives. Approximately ten hours later, partial recovery of vision was observed. Blood pressure values remained labile, ranging between 140/100 mmHg and 120/90 mmHg, requiring ongoing monitoring and adjustment of antihypertensive therapy. Over the following days, strict clinical and laboratory surveillance demonstrated persistent fluctuations in blood pressure, along with hypoproteinemia and elevated LDH levels. These findings prompted further optimization of antihypertensive treatment and administration of bioproducts. The patient's neurological status progressively improved, with complete restoration of visual function. After seven days of hospitalization, the patient was discharged in stable condition with recommendations for outpatient blood pressure monitoring and maintenance of optimal blood pressure control according to the European Society of Cardiology (ESC) Guidelines for treatment of hypertension. Nimodipine was prescribed as maintenance antihypertensive therapy. Follow-up evaluations by both a cardiologist and a neurologist, together with brain

MRI, were scheduled one month after discharge. The follow-up MRI demonstrated no pathological abnormalities, and antihypertensive therapy was subsequently discontinued.

Fig. 1. Inter-twin membrane folding in monochorionic pregnancy



DISCUSSION

PRES is associated with a variety of underlying conditions, most commonly hypertension. Neurological manifestations in patients with preeclampsia and eclampsia are strongly linked to PRES. The initial description of PRES in 1996 involved 15 patients, 3 of whom had eclampsia [1]. Subsequent studies have demonstrated a high prevalence of PRES among patients with severe preeclampsia and eclampsia. A retrospective cohort study of 47 eclampsia patients at a single center revealed radiographic evidence of PRES in 46 cases (97.9%) [8]. Similarly, a case series from other research, including 39 patients with eclampsia or preeclampsia complicated by neurological symptoms, found PRES on MRI in 12 of 13 patients with eclampsia (92.3%) and in 5 of 26 patients with preeclampsia (19.2%) [9]. PRES within several weeks after delivery, defined as delayed eclampsia, has also been reported [10]. Although commonly associated with hypertension, PRES may also occur in normotensive patients, and its wide spectrum of clinical manifestations often poses a diagnostic challenge.

The exact pathophysiologic mechanisms underlying PRES remain incompletely understood. Several theories have been proposed, with hypertension-induced autoregulatory failure and endothelial dysfunction being the most widely accepted. Elevated blood pressure and endothelial injury are commonly observed in these conditions. Vasoconstriction, a compensatory response to hypertension, can result in both vasogenic and cytotoxic edema and is considered a major factor contributing to clinical symptoms and radiological findings [11]. Additionally, hypersecretion of arginine vasopressin (AVP) appears to be closely linked to the development of PRES. Excess AVP, or increased sensitivity of its receptors, may trigger cerebral vasoconstriction via V1a receptor activation, leading to endothelial dysfunction, cer-

erebral ischemia, and the development of cytotoxic and vasogenic brain edema. This suggests that AVP plays a central pathophysiologic role in PRES [12]. Another hypothesis proposes that PRES development may involve immune system activation, leading to the release of cytokines such as tumor necrosis factor (TNF) and interleukin-1 (IL-1), which in turn cause endothelial dysfunction and fluid leakage [13].

PRES most commonly presents with a range of acute or subacute neurological symptoms. Although it is frequently associated with elevated blood pressure, cases occurring in normotensive patients and in association with hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome have also been reported [14]. Patients may exhibit various quantitative or qualitative disorders of consciousness, including cognitive deficits, stupor, somnolence, or coma. Seizures, either focal or generalized, are frequently observed and, in some cases, may progress to status epilepticus [15]. Visual disturbances, including reduced visual acuity, visual field deficits, cortical blindness or visual hallucinations, occur in approximately two-thirds of PRES patients, reflecting frequent occipital lobe involvement as well as in the present case [1]. Less specific symptoms include headache, nausea, vomiting, and altered consciousness. Focal neurological deficits are reported in 5–15% of cases, and rare myelopathic symptoms are also described in patients with spinal cord involvement [16]. West T et al. reported a case of postpartum PRES in a woman presenting with agitation and psychotic features, highlighting that acute psychotic episodes in the postpartum period may signal underlying PRES [17].

Since PRES may occur in both pregnant and non-pregnant patients, Roth C. conducted a retrospective analysis of 21 cases, dividing them into two groups: pregnant and non-pregnant [18]. No significant differences were observed in terms of clinical presentation, cerebral imaging, or outcomes, although the incidence of headache was significantly higher among pregnant patients.

Marrone et al. conducted a literature review comparing PRES in pregnant and non-pregnant patients. Their findings indicated that non-pregnant patients exhibited higher blood pressure values as well as elevated creatinine levels. Based on these results, the authors suggested that in pregnant patients, PRES may develop due to a breakdown of cerebral autoregulation occurring at lower mean arterial blood pressure thresholds [19]. These differences highlight the importance of considering pregnancy-specific pathophysiological mechanisms in diagnosing and managing PRES.

PRES is typically characterized by subcortical white matter and cortical edema, most commonly affecting the bilateral parietal and occipital lobes [14]. On computerized tomography, some patients may demonstrate vasogenic edema presenting as hypodense regions, whereas brain MRI represents a more sensitive imaging modality. In addition to edema, MRI may reveal restricted diffusion consistent with ischemia, as well as evidence of intracranial hemorrhage in certain cases. These features establish MRI as the gold standard for the diagnosis of PRES [4].

The differential diagnosis of PRES encompasses a broad spectrum of neurological conditions, which can be generally classified as vascular, infectious, metabolic, and

demyelinating disorders. These include:

- ischemic or hemorrhagic stroke,
- cerebral venous sinus thrombosis,
- metabolic/toxic encephalopathy,
- encephalitis/meningitis,
- electrolyte imbalances – hyponatremia, hypocalcemia,
- demyelinating disorders (multiple sclerosis),
- primary or secondary brain tumor,
- epilepsy.

In the present case, the differential diagnosis was particularly challenging because the initial manifestation was sudden bilateral visual loss. Ophthalmologic examination revealed preserved pupillary light reflexes and a normal macular appearance, making primary ocular causes such as retinal detachment, retinal artery occlusion, and optic nerve pathology less likely. Acute ischemic or hemorrhagic stroke was considered; however, the absence of focal neurological deficits and the complete recovery of visual function argued against permanent cerebrovascular injury. Cerebral venous sinus thrombosis was also considered given the postpartum status, but neuroimaging findings were not supportive of this diagnosis. Metabolic and infectious causes were considered less likely because laboratory investigations revealed no significant electrolyte disturbances, and there were no clinical signs of central nervous system infection. Demyelinating disorders such as multiple sclerosis were considered unlikely due to the acute onset of symptoms, the absence of previous neurological episodes, and the complete clinical and radiological resolution during follow-up. Epilepsy-related visual disturbances, including postictal cortical blindness, were also considered; however, no seizures were observed prior to the onset of visual loss, and the clinical course was not suggestive of a postictal state. The combination of acute visual symptoms, altered mental status, severe blood pressure fluctuations, and subsequent complete clinical and radiological resolution was most consistent with PRES.

However, the diagnosis of PRES should not rely solely on blood pressure values, as rapid fluctuations in blood pressure are common and normal measurements do not exclude the condition. Therefore, MRI remains the cornerstone of diagnosis, allowing confirmation of PRES and differentiation from other neurological disorders. Although the syndrome is typically reversible, some patients may experience persistent neurological deficits. Prompt recognition and treatment of the underlying cause are essential for achieving a favorable outcome.

CONCLUSION

PRES is a reversible condition when promptly recognized and managed. Women, especially during pregnancy and the postpartum period, are at increased risk. MRI plays a key role in establishing the diagnosis and distinguishing it from other neurological disorders. Our case highlights the importance of early recognition, multidisciplinary collaboration, and close monitoring of blood pressure and neurological status. Increased awareness of clinical warning signs is essential to prevent complications and achieve favorable outcomes.

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