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CLINICAL REPORT

Postoperative blindness associated with posterior reversible encephalopathy syndrome: a case report

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Abstract We report the case of a 46-year-old woman presenting with postoperative bilateral cerebral visual loss that was initially misinterpreted as an irreversible ischemic event. Magnetic resonance imaging of the brain showed high signal intensity on T2-weighted and fluid-attenuated inversion recovery images and normal signal intensity on diffusion-weighted images of the posterior lobe, which mostly disappeared with the improvement of clinical symptoms. Subsequent diagnosis revealed posterior reversible encephalopathy syndrome (PRES). Recognition of PRES as the correct diagnosis led to the appropriate management strategy and the recovery of normal vision. Differentiation from acute cerebral ischemia is important in order to prevent permanent vision loss due to delay in initiating prompt and vigorous treatment of exacerbating factors, such as intermittent hypertension. We believe that it is important for anesthesiologists and critical care physicians to accurately diagnose PRES in view of the key differences in the management of similarly presenting conditions.

Keywords Blindness · Posterior reversible encephalopathy syndrome · FLAIR · DWI

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Introduction

Posterior reversible encephalopathy syndrome (PRES) was first described in 1996 [1]. Patients with PRES show various kinds of neurologic symptoms that include the acute or subacute onset of confusion, lethargy, and visual disturbances. The diagnosis of PRES is often confirmed on the basis of the clinical situation and findings on neuroimaging consistent with subcortical vasogenic edema, particularly those involving the posterior lobes without hemorrhage or infarction, as opposed to cytotoxic edema [2]. The etiological cause of PRES is thought to be hypertensive encephalopathy, with dysregulation of the cerebral vasculature resulting in acute cerebral edema [3]. PRES presents with a variety of neurological features that are potentially reversible on prompt recognition and appropriate treatment, but clinicians sometimes fail to diagnose it immediately [4]. Delayed diagnosis can lead to severe and long-term neurological disability.

Although several cases of PRES have been reported in the neurology and neuroradiology literature, exposure in the anesthesiology literature has been rather limited. We describe a patient who developed sudden blindness associated with PRES in the setting of intraoperative hypertension and also discuss the pathogenesis and the role of neuroimaging findings in determining the diagnosis of PRES and in distinguishing it from other diseases.

Case report

A 44-year-old woman without any specific medical history was admitted for hysterectomy due to uterine myoma. On hospital admission, her blood pressure (BP) was 120/80 mmHg, and she had no neurologic deficits and no edema.

Blood cell counts, electrolyte levels, and urinalysis results were normal. Glycopyrrolate 0.2 mg and midazolam 2 mg were given intramuscularly 30 min before anesthesia induction. Electrocardiogram, pulse oximetry and noninvasive BP were monitored. Following pre-oxygenation with O_2 5 L/min, anesthesia was induced with a target-controlled infusion (TCI) of 3 ng/ml remifentanil and propofol 100 mg. Rocuronium 50 mg was injected to the peripheral vein for facilitating intubation. Intubation was done easily with a cuffed tracheal tube. Anesthesia was maintained with oxygen (FiO_2 0.5), sevoflurane (1 MAC), and TCI of remifentanil titrated to maintain hemodynamic parameters. Muscle relaxation was continued with a bolus dose of rocuronium 10 mg. Ventilation was adjusted to maintain $ETCO_2$ at 30–35 mmHg. Intraoperative BP was 170/90 mmHg after intubation and 160/90 mmHg at the end of the surgery; otherwise, the intraoperative BP was maintained at 120–140/60–80 mmHg.

Postoperatively, the patient was transferred to the postanesthetic care unit (PACU). She was disorientated to time throughout her PACU stay. During PACU stay, the BPs were 140–160/60–70 mmHg, with one instance of 180/80 mmHg.

Three hours postoperatively, the PACU nurse noted that the patient complained of severe headache on both temporal regions and was unable to see anything with both eyes. She was transferred to the intensive care unit (ICU), and emergent ophthalmology and neurology consultation was requested. Regular neurologic examinations, including determining Glasgow coma scores, were conducted, and the patient was neurologically intact, with the exception of mild confusion, especially related to time. Near visual acuity (VA) without correction was light perception in all quadrants bilaterally. Pupils were 3.5 mm in each eye and were reactive to light, and intraocular pressures (normal range <20 mmHg) were 13 and 11 mmHg, respectively. Portable slit-lamp and dilated fundus examinations were unremarkable, indicating that the function of the eye balls were normal.

An emergent head computed tomography (CT) scan was normal. Magnetic resonance imaging (MRI)/magnetic resonance angiography studies of the head were obtained. Cerebral edema involving the posterior parieto-occipital white matter was seen as increased T2 and a fluid-attenuated inversion recovery (FLAIR) signal (Fig. 1) without any reduction in the apparent diffusion co-efficient signal (ADC) on diffusion-weighted imaging (DWI) (Fig. 2). There was no evidence of cerebral hemorrhage or infarction. When the clinical features were also taken into account, these imaging findings suggested a diagnosis of PRES.

We initiated labetalol infusion at 2 mg/min, titrated up to a maximum dose 8 mg/min to maintain a systolic BP <140–150 mmHg. This strategy was successful in maintaining the BP in the desired range. One day later, the patient was alert and oriented to person, place, and time. She was no longer confused, and near VA was 20/25 bilaterally. She was discharged from the hospital 7 days after her operation, and a brain MRI, repeated 1 month later, showed mostly amelioration.

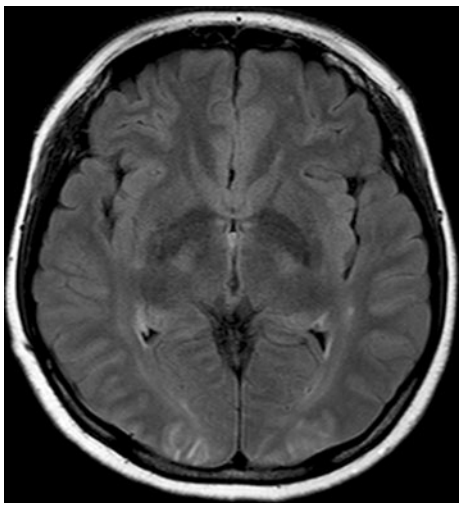

Fig. 1 Axial fluid-attenuated inversion recovery magnetic resonance image showing increased signal in subcortical lesions of the both posterior lobes

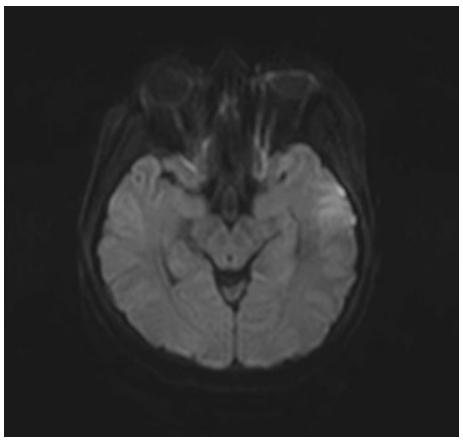

Fig. 2 The diffusion-weighted image showing no abnormal findings

Discussion

Hinchey et al. were the first to report a reversible posterior leukoencephalopathy syndrome, and the name was displaced by 'PRES' in 2000, which is now the most widely accepted terminology [5].

The clinical features that should alert the clinician to PRES are headache and behavior ranging from drowsiness

to stupor, seizures, mental abnormalities, including confusion, and abnormalities of visual perception [6]. Most cases of PRES are associated with hypertensive disorders, particularly those of pregnancy-induced hypertension [2]. The presenting features of PRES, including blindness, seizures, impairment of consciousness, and motor signs, usually develop suddenly. Sudden blindness without a contributory history in this patient is unusual. A mini-mental neurologic examination can assist the clinician in determining the extent of confusion and can facilitate the diagnosis of PRES.

The early diagnosis of PRES is very important in terms of initiating therapy with antihypertensives or anticonvulsants, eliminating possible offending medication, treating associated disorders and, possibly, preventing irreversible brain damage. CT scanning is less sensitive, so MRI is the favored modality of investigation [4]. Cerebral edema involving the posterior parieto-occipital white matter is seen as an increased T2 and FLAIR signal without any reduction in ADC on DWI and distinguishes PRES from the cytotoxic edema of acute cerebral infarction [6]. Indeed, a decreased ADC signal portends areas of permanent injury in PRES.

The pathophysiology of PRES has been somewhat controversial. The currently favored hypothesis is that PRES is associated with vasogenic edema rather than cytotoxic edema [7]. Acute hypertension that overcomes cerebral autoregulation leads to breakdown of the blood–brain barrier, cerebral vasodilatation, and transudation of fluid resulting in brain edema. Because the anterior cerebral circulation is much better supplied with sympathetic innervation than the posterior circulation, the posterior cerebral circulation may be predisposed to loss of protective vasoconstriction, breakthrough vasodilation and vasogenic edema in the face of acute hypertension [8].

Early accurate diagnosis and prompt treatment of PRES may prevent permanent brain damage [6]. In hypertensive encephalopathy, the mean arterial pressure should be reduced by 20–25% within the first 1–2 h or the diastolic blood pressure (BP) reduced to 100 mmHg over minutes to hours. A rapid reduction in BP should be avoided because end organ dysfunction and cerebral infarction can occur [9]. Intravenous administration is generally preferred, and the current drugs of choice include sodium nitroprusside, labetalol, and calcium channel blockers [10]. Angiotensin-converting enzyme inhibitors are contraindicated in patients with hypovolemia and underlying renal artery stenosis [6]. Because of their known association with PRES, any immunosuppressive drugs should be withdrawn or reduced [1]. Seizures should be treated actively in all cases, as status epilepticus sometimes occurs [2]. General supportive care is also important in PRES.

PRES is a clinical entity that may be unfamiliar to many anesthesiologists, but the anesthesiologist is often the first clinician to be confronted with a patient presenting with acute visual loss after intraoperative hypertension. As such, he/she should be familiar with this diagnosis. Differentiation from acute cerebral ischemia and prompt and vigorous treatment of exacerbating factors, such as intermittent hypertension, are important if permanent visual loss is to be avoided.

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References

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, Pessin MS, Lamy C, Mas JL, Caplan LR. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494–500.
2. Striano P, Striano S, Tortora F, De Robertis E, Palumbo D, Elefante A, Servillo G. Clinical spectrum and critical care management of posterior reversible encephalopathy syndrome (PRES). Med Sci Monit. 2005;11:CR549–53.
3. Schwartz RB, Feske SK, Polak JF, DeGirolami U, Iaia A, Beckner KM, Bravo SM, Klufas RA, Chai RY, Repke JT. Pre-eclampsia-eclampsia: clinical and neuroradiographic correlates and insights into the pathogenesis of hypertensive encephalopathy. Radiology. 2000;217:371–6.
4. Mukherjee P, McKinstry RC. Reversible posterior leukoencephalopathy syndrome: evaluation with diffusion-tensor MR imaging. Radiology. 2001;219:756–65.
5. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. AJNR Am J Neuroradiol. 2000;21:1199–206.
6. Stott VL, Hurrell MA, Anderson TJ. Reversible posterior leukoencephalopathy syndrome: a misnomer reviewed. Intern Med J. 2005;35:83–90.
7. Port JD, Beauchamp NJ Jr. Reversible intracerebral pathologic entities mediated by vascular autoregulatory dysfunction. Radiographics. 1998;18:353–67.
8. Morita Y, Hardebo JE, Bouskela E. Influence of cerebrovascular sympathetic, parasympathetic, and sensory nerves on autoregulation and spontaneous vasomotion. Acta Physiol Scand. 1995; 154:121–30.
9. Calhoun DA, Oparil S. Treatment of hypertensive crisis. N Engl J Med. 1990;323:1177–83.
10. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet. 2000;356:411–7.