Posterior Reversible Encephalopathy Syndrome (PRES)

**Definition**
Posterior reversible encephalopathy syndrome (PRES) is a radiological and clinical entity that is characterized by a variety of symptoms that include headache, altered mental status, visual loss, seizures, and loss of consciousness in conjunction with unique neuroimaging findings of vasogenic edema involving the posterior circulation.\(^1,2,3\)

**Epidemiology**
The global incidence of PRES is unknown and no predominant age group has been identified. However, PRES is more common among female patients than male.\(^3,4\)

**Pathophysiology**
In 1996, Hinchey et al., described 15 patients who developed reversible vasogenic cerebral edema related to sudden increased arterial blood pressure. The term PRES was proposed.\(^3\)

Predisposing factors related to PRES include pre-eclampsia, eclampsia, glomerulonephritis, dialysis, organ transplant, autoimmune diseases, infection, and administration of radio-contrast, cyclosporine or immunosuppressant.\(^5,6\)

Acute elevation of blood pressure in a normotensive individual is the typical presentation of PRES. Posterior reversible encephalopathy syndrome is rarely seen in patients with chronic hypertension.\(^7\) Even PRES is most frequently related to patients with hypertension, it can also occur in normotensive patients.\(^8\)

The exact pathogenesis of PRES is not completely clear. There are several theories that explain the mechanism of PRES. They are the autoregulatory disturbance with hyperperfusion theory; cerebral vasospasm resulted from acute hypertension, and cytotoxic theory.\(^9\)

The most favourable theory is the autoregulatory disturbance with hyperperfusion theory. In PRES, the blood pressure exceeds the upper limits of autoregulation that leads to hyperperfusion. As a result of the hyperperfusion, the blood-brain barrier is disrupted and leaded to vasogenic cerebral edema.\(^10,11\)

The second theory indicated that cerebral vasospasm is developed due to “over-reaction” to the increased blood pressure. The cerebral vasospasm results in arterial thrombosis and ischemia, thus leads to cytotoxic edema.\(^11\)

The third theory stated there is direct toxicity effect on the cerebral vascular endothelium that leads to blood brain barrier disruption and results in vasogenic edema.\(^11\)

Researchers suggested the posterior cerebral is more prone to be affected is due to the posterior circulation having less sympathetic innervations, which results in a decreased response to the increased blood pressure.\(^12\)

The original description of vasogenic edema is bilateral symmetrical in the posterior parenchyma, especially the parieto-occipital region. However, PRES could be unilateral and also affect other regions such as the frontal lobes, temporal lobes, cerebellum, brainstem, deep white matter, and basal ganglia. In addition, the gray matter could also be affected.\(^5,8,10,12\)

Although PRES is called “reversible”, sometimes even with early diagnosis and adequate treatment, some complications from PRES such as cerebral ischemia, intracranial hemorrhage, and cerebral infarction may occur and leave patients with permanent neurological deficits.\(^11,12\)

**Diagnostic Tests**
The most effective diagnostic tests for PRES are computed tomography (CT scan) and magnetic resonance imaging (MRI). The characteristic pattern of PRES in CT scan is the presence of edema (hypodensured areas) involving both cerebral hemispheres. Asymmetrical or unilateral patterns are not uncommon. The most common location involved is the occipital region (94%).\(^6,8\)

In MRI especially T2 or fluid attenuated inversion recovery (FLAIR), PRES shows hyperintense areas that consistent with edema.\(^9\)

**Manifestations**
The symptoms of PRES usually develop rapidly over a few hours and peak in 12 – 48 hours.\(^13\) Symptoms of PRES are non-specific, including headaches, nausea, vomiting, aphasia, facial numbness, ataxia, altered mental status, seizures, and progressive visual loss.\(^2,10,12\)

Headache is usually bilateral and dull.\(^13\)

Visual anomalies are the most frequent symptoms due to the involvement of the occipital lobe especially in patients with eclampsia.\(^14\) Visual anomalies include blurred vision, homonymous hemianopia, visual neglect, visual hallucinations, and cortical blindness.\(^15\) These anomalies are usually reversible. However, the visual anomalies may not be reversed if cerebral infarction was resulted from PRES.\(^9\)
Seizures are one of the most common presentations, especially in the pediatric population. It may occur as the first sign of PRES or it can occur later during the onset. Types of seizures vary from focal seizures to generalized seizures. Status epilepticus is a common presentation of PRES.

Treatment Options

Early recognition of PRES and control of the elevated blood pressure is the essential component of treatment for PRES. Treatment goals aimed at lowering the mean arterial blood pressure (MAP) to the premorbid levels. Adequate reduction of blood pressure is able to prevent complications such as intracranial hemorrhages, cerebral ischemia, cerebral infarct, and permanent neurological deficits.

Medications most commonly used for treatment of PRES are antihypertensive medication such as labetalol, hydralazine, and nifedipine, diuretic agents, and glucocorticoids. Nitroglycerin may aggravate PRES due to the cerebral vasodilatation effect, and their use should be avoided. Angiotensin converting enzyme inhibitors should be avoided for pregnant patients due to the risk of harmful effects to fetal kidneys.

Other interventions include eliminating the causative mechanism and preventing or controlling complications such as seizures should be considered. Seizures normally do not progress into chronic epilepsy. Antiepileptic agent should not be given more than three months unless patients developed ongoing or recurrent seizures activities. The average recovery period is approximate 5 days.

Nursing Implication

Prompt recognition and timely treatment for PRES can reverse the symptoms and avoid permanent neurological deficits. It is essential to monitor patients' blood pressure very closely. Maintaining a low blood pressure is important to prevent an increase in vasogenic edema. Laboratory tests should include a serum magnesium level. Magnesium is a competitive antagonist to calcium. It can reverse cerebral vasoconstriction. Hypomagnesemia may augment PRES and should be avoided.

Because of the possible visual disturbance, it is important to protect patients from risk of falls such as provide adequate lighting, unclustering patients' room, reinforcing the need to call for assistance, ensuring the floor is dry and has stable furniture.

Reference


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