

PRES (Posterior Reversible Encephalopathy Syndrome) and Eclampsia:-Review

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Abstract

The Posterior Reversible encephalopathy Syndrome (PRES) is a cliniconeuroradiologic entity. Eclampsia is one of the important causes of PRES. Most patients have severe hypertension, some have only mildly elevated or even normal blood pressure. Symptoms include headache, nausea, vomiting, altered mental status, seizures, stupor, and visual disturbances. On CT and MR studies, edema is relatively symmetrical pattern, typically in the sub cortical white matter and occasionally in the cortex of the occipital and parietal lobes. PRES is reversible when treatment is instituted early, delayed diagnosis and treatment can result in chronic neurological sequelae. Early recognition and controlled of blood pressure and seizure is the main stay of treatment. Anesthesiologist, intensivists and other physicians involved in the evaluation of patients with markedly elevated blood pressure, eclampsia, renal failure etc should presumed PRES and must be aware of the clinical spectrum of the associated conditions, its diagnostic modalities, and treatment.

INTRODUCTION

Preeclampsia is one of the most common medical disorders affecting pregnancy, with significant maternal and fetal morbidity and mortality. The most serious maternal complications of preeclampsia include intracerebral hemorrhage, eclampsia, and renal failure, as well as hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome and the recent entity of posterior reversible encephalopathy syndrome (PRES). Reversible Posterior Leukoencephalopathy Syndrome was introduced¹ into clinical practice in 1996 in order to describe unique syndrome, clinically expressed during hypertensive and uremic encephalopathy, eclampsia and during immunosuppressive therapy. First clinical investigations showed that leucoencephalopathy is major characteristic of the syndrome, but further investigations showed no significant destruction in white cerebral tissue. In majority of cases changes are localise in posterior irrigation area of the brain and in the most severe cases anterior region is also involved. Taking into consideration all above mentioned facts, the suggested term described was Posterior Reversible Encephalopathy Syndrome (PRES) for the syndrome clinically expressed by neurological manifestations derived from cortical and subcortical changes localised in posterior regions of cerebral hemispheres, cerebral trunk and cerebellum.

CAUSES OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

Hypertensive encephalopathy

Eclampsia²

Renal failure with hypertension

Immunosuppressive agents³ and cytotoxic drugs:- Cyclosporin A, Interferon alfa, Intravenous immunoglobulins, Cisplatin, , Cytarabine Erythropoietin, Tacrolimus

Collagen vascular disorders-e.g. Systemic lupus erythematosus⁴ etc

Others :- hypercalcemia⁵, Postdural puncture and spinal anaesthesia^{6,7,8}

Aetiopathogenesis

The exact pathogenesis of the condition is not known. A rapid rise in blood pressure leads to altered autoregulation of cerebral blood flow producing dilatation of cerebral arterioles with opening up of endothelial tight junctions and leakage of plasma and red cells into the extracellular space, producing cerebral oedema. The cerebral white matter is composed of myelinated fibre tracts in a cellular matrix of glial cells, arterioles, and capillaries that makes this structure

more susceptible to accumulation of fluid in the extracellular spaces (vasogenic oedema). The posterior circulation is thought to be more susceptible to this type of damage, because there is less sympathetic innervation of the vertebrobasilar vasculature to protect the parenchyma from rapid increases in arterial blood pressure >100 . According to another hypothesis, patients with PRES develop vasospasm secondary to sudden and severe rises in blood pressure and ischaemia of brain tissue. Ischaemic damage to brain tissue first produces cytotoxic oedema and then extracellular oedema. However, the reversibility of imaging abnormalities with immediate treatment is not consistent with the hypothesis of vasospasm and cerebral ischaemia.

CLINICAL FEATURES

Most common symptoms are headache, nausea, vomiting, confusion, behavioural changes, changes of consciousness (from somnolence to stupor), vision disturbances (blurred vision, haemianopsia, cortical blindness) and epileptic manifestations (mostly focal attacks with secondary generalisation). Mental functions are characterised with decreased activity and reactivity, confusion, loss of concentration and mild type of amnesia. Stupor and coma rarely occurred. Multiple seizures are more common than single events. Recently few cases reported in postpartum woman who, developed posterior reversible encephalopathy syndrome after spinal anesthesia and/or complicated by postdural puncture headache⁶⁷⁸

DIAGNOSIS

Brain MRI better detects smaller focal abnormalities than brain CT. The most often neuroradiological finding is relatively symmetrical oedema of white cerebral tissue in parieto-occipital regions of both cerebral hemispheres. Gray cerebral tissue is sometimes involved, usually in mild form of disease. Diagnosis of this "cortical" form of PRES is possible by MR FLAIR (Fluid-Attenuated Inversion Recovery) technique. ¹¹

DIFERENTIAL DIAGNOSIS

One of the important differential diagnoses of PRES in pregnancy and post-partum period is cerebral venous thrombosis¹² (CVT). Onset of CVT is acute and evolved in days. Usually presented as headaches, seizures, focal neurological deficits (hemiparesis or monoparesis), papilloedema, stupor or coma, evidence of venous thrombosis elsewhere and infrequently hypertensive

TREATMENT

The posterior encephalopathy syndrome needs to be recognized promptly. The syndrome is usually reversible within 7 days, after controlling the blood pressure. Offending immunosuppressive agents should either be discontinued or the dose should be reduced. Delay in initiating the appropriate treatment may result in permanent damage to the brain. Patients experiencing seizures become seizure free after resolution of imaging abnormalities and do not require chronic antiepileptic treatment. Therapeutic strategy depends on the cause of PRES and clinical picture. Most important are blood pressure regulation (labetalol, nitropruside, diuretics), control of epileptic attacks (phenytoin), anti-oedema therapy (Manitol). Induction of vaginal delivery in eclampsia and discontinuation of cyclosporin therapy. In most cases there are no neurological manifestations after the 7th day, but some studies showed normalisation of clinical finding after one year and more.

SUMMARY

Eclampsia is one of the important causes of obstetric related morbidity and mortality. Early recognition of PRES in eclampsia is of paramount importance because prompt control of blood pressure will cause reversal of the syndrome. Antihypertensive controlled of seizure and anti-edema measures are the main stay of treatment. Delay in the diagnosis and treatment can result in permanent damage to affected brain tissues.

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