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Published paper's title : Case Report : Posterior Reversible Encephalopathy Syndrome (PRES) in an eclamtic patient after delivery of a baby

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Case Report

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Case Report : Posterior Reversible Encephalopathy Syndrome (PRES) in an eclamtic patient after delivery of a baby

Shaheen Bano1, Shashi Prakash2, and Yashpal Singh3

Declaration

The Declaration of the authors for publication of Research Paper in Asian Journal of Modern and Ayurvedic Medical Science (ISSN 2279-0772) We *Shaheen Bano1, Shashi Prakash2, and Yashpal Singh3* the authors of the research paper entitled *Case Report : Posterior Reversible Encephalopathy Syndrome (PRES) in an eclamtic patient after delivery of a baby.* declare that , We take the responsibility of the content and material of our paper as We ourself have written it and also have read the manuscript of our paper carefully. Also, We hereby give our consent to publish our paper in ajmams , This research paper is our original work and no part of it or it's similar version is published or has been sent for publication anywhere else.We authorise the Editorial Board of the Journal to modify and edit the manuscript. We also give our consent to the publisher of ajmams to own the copyright of our research paper.

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SUMMARY : A 23 year old woman, 2 hours after delivery of a baby admitted to ICU with altered sensorium, elevated blood pressure and two episodes of generalized tonic clonic seizure one hour before admission. She was managed with oxygen inhalation, intravenous (i.v) $MgSO_4$ infusion, i.v Labetolol, i.v Mannitol, Dexamethasone i.v and Tab. Nimodipine added on day 3 as blood pressure remains higher in spite of increasing dose of labetolol. Patient remains unconscious on day 3 and CT scan was planned to rule out any intracranial complication. CT scan reveals diffuse hypo density in the white matter in B/L parieto- occipital lobes S/O ? Posterior Reversible Leucoencephalopathy Syndrome (PRES). Patient regained consciousness on day 6 and shifted to ward on day8.

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KEYWORDS: Posterior reversible encephalopathy syndrome (PRES), generalized tonic clonic seizure, intracranial complication.

KEY MESSAGES: PRES is a rare but a serious maternal complication of eclampsia and should be suspected clinically and confirmed by CT or MRI finding. It is a 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.

INTRODUCTION

The most serious maternal complications of eclampsia include intracerebral hemorrhage, 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 Leucoencephalopathy Syndrome was introduced into clinical practice in 1996. [1] The Posterior Reversible encephalopathy Syndrome (PRES) is a cliniconeuroradiologic entity. Eclampsia is one of the important causes of PRES. [2][3] In majority of cases changes are localize in posterior irrigation area of the brain and in the most severe cases anterior region is also involved. On CT and MRI 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.

CASE REPORT

A 23 year old pregnant female was all right one month back following she complains of swelling in bilateral leg and pain in abdomen. She consulted nearby hospital and managed by some oral medication and got relieved. Day before admission to intensive care unit (ICU) patient complains of pain in abdomen, blurring of vision and headache. She admitted to nearby hospital and delivers a female baby vaginally. Baby cry immediately after birth and Apgar score was 9 at 3 min. One hour after delivery patient had 2-3 episodes of generalize tonic colonic (GTCS) seizure and altered sensorium for which she was referred to ICU. At time of admission to ICU patient was in altered sensorium with GCS of E2V2M5, Heart Rate (HR) 130/min, Noninvasive Blood Pressure (NIBP) 170/110 mmHg, Oxygen saturation (SpO2) 90%, cough reflex was present. Chest and cardiovascular system (CVS) was within normal limit. Patient put on ventimask with FiO2 of 35%, Inj. MgSO4 infusion 2gm/hr, Inj labetolol 20mg i.v. stat then 20mg i.v BD, Inj Mannitol 100 ml slow i.v. , Inj Dexamethasone 6mg i.v. TID was started from day 2, Tab Nimodipine 30mg through Ryle's tube (RT) TID was started from day 3 as BP was not controlled with increasing dose of labetolol. MgSO4 infusion was stopped after 48 hrs but patient remained unconscious. We suspected some intracranial complications of eclampsia and planned CT scan of brain on day 3. CT scan reveals -diffuse hypo density in the white matter in B/L parieto- occipital lobes S/O ? Posterior Reversible Leucoencephalopathy Syndrome. Patient regained consciousness on day 6 on conservative management and shifted to ward on day 8.

DISCUSSION

Reversible Posterior Leucoencephalopathy Syndrome a clinicoradiological diagnosis was introduced into clinical practice in 1996. Usually reported in Hypertensive encephalopathy, Renal failure with hypertension, Eclampsia (pregnancy or puerpurium), Immunosuppressive agents and cytotoxic drugs. Clinical signs and symptoms are non-specific and may be acute or sub acute. Symptom includes Headaches, Altered mental state, lethargy and somnolence, possibly progressing to confusion and coma, Convulsions, Blurred vision, hemianopia, visual defect and hallucinations. Hypertension and metabolic disturbances are common signs.

Three theories was proposed, The earliest theory suggested that overreaction of brain auto regulation results in reversible vasospasm, which in turn results in potentially reversible ischemia to the

brain, especially in vascular border zone territories.

The newer theory suggested that auto regulation maintains a constant blood flow to the brain, despite systemic blood pressure alterations, by means of arteriolar constriction and dilatation. So, the constricted arterioles are forced to dilate because of the increased systemic blood pressure, resulting in brain hyper perfusion. This increased perfusion pressure is sufficient to overcome the blood-brain barrier, allowing extravasion of fluid, macromolecules, and even red blood cells into the brain parenchyma. So, PRES represents vasogenic rather than cytotoxic oedema in the majority of cases. [4]

The last theory suggests that at intravascular pressures just below those that could rupture the capillary wall, permeability through the endothelium increased markedly, which was most likely due to increased pinocytotic activity through the capillary wall. The active passage of fluid through the capillary wall may act to relieve intravascular pressure, forestalling the development of large haemorrhages. So in this case, PRES is not an example of cytotoxic or vasogenic oedema but hydrostatic oedema. However the pathogenesis is attributed to a failure of cerebral auto regulation that is probably facilitated in posterior brain regions due to sparse sympathetic innervations of the vertebrobasilar vascular system. Posterior circulation has less sympathetic adrenergic innervations, and therefore is thought to be more susceptible to effects of rapid rise in blood pressure.

CSF usually normal or may be elevated protein. 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.CT- hypodence lesion. MRI- iso/hypo intense in T1, hyperintence T2.

Management is supportive includes control of blood pressure, seizure and to reduce brain edema.

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.

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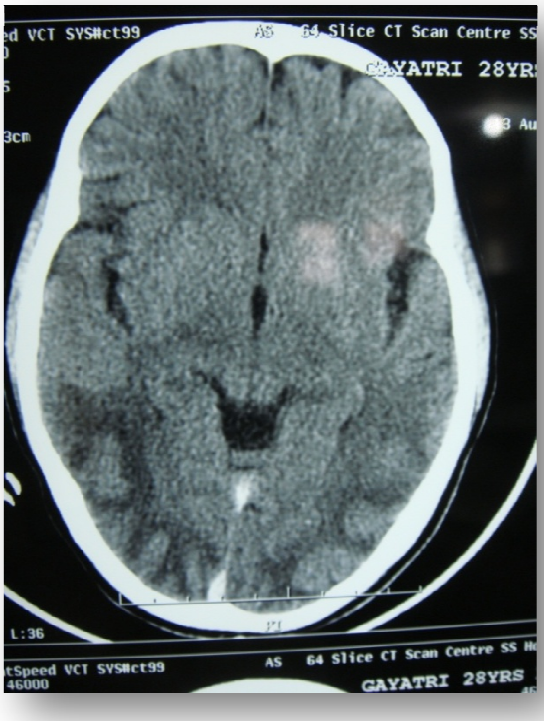
LEGENDS:

FIGURE:1 CT scan of head showing hypo density in the white matter in B/L parieto-occipital lobe.

FIGURE:2 Patient of Posterior Reversible Encephalopathy Syndrome in ICU.



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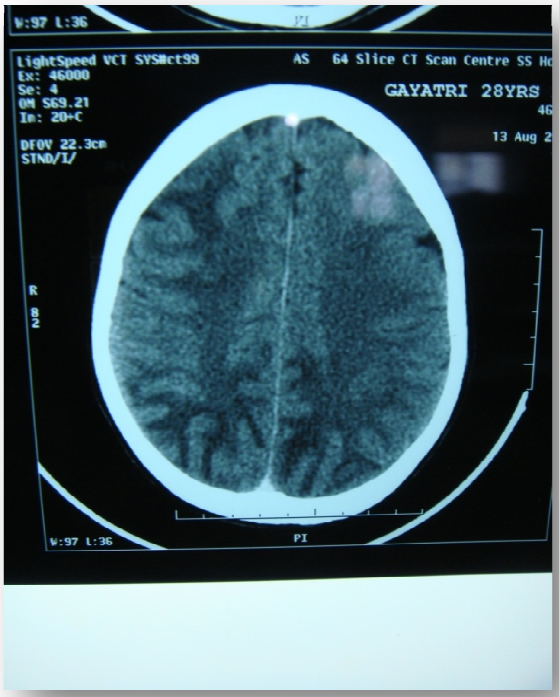

FIGURE:2 Patient of Posterior Reversible Encephalopathy Syndrome in ICU