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Case Report

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN POSTPARTUM WOMAN: A CASE REPORT

Makarim AHM[a], Karunarathna SMG[b]

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INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) also known as reversible posterior leukoencephalopathy syndrome (RPLS) is a neurotoxic state that occurs secondary to the inability of posterior circulation to auto-regulate in response to acute changes in blood pressure. PRES has been accompanied by a number of medical conditions such as hypertensive encephalopathy, preeclampsia, eclampsia, acute or chronic renal diseases, hemolytic uremic syndrome, use of cytotoxic and immunosuppressant drugs, blood transfusion, and electrolyte disturbances[1]. Clinical and radiological features of PRES include headache, encephalopathy, seizures, cortical visual changes, and parieto-occipital white matter edema visualized on neuroimaging modalities. An association between eclampsia and PRES was first described by Hinchey et al in 1996[2]. Low magnesium levels can augment PRES(3). The diagnosis is typically made clinically, with supportive findings on magnetic resonance imaging of the brain. The treatment of PRES depends on the underlying cause. Prompt recognition and treatment are crucial to avoid the permanent damage leading to sequelae and even mortality. Many cases resolve within 1–2 weeks of controlling the blood pressure and eliminating the inciting factor. PRES may recur in about 5-10% of cases[3]. In this case report, we present a pre-eclamptic pregnant woman presented with acute loss of vision and elevated blood pleasure due to PRES without seizures after delivery (which is an uncommon complication of preeclampsia.)

[a] *Registrar in Obstetrics and Gynaecology, Sri Jayewardanapura General Hospital Sri Lanka*

[b] *Consultant Obstetrician & Gynaecologist, Sri Jayewardanapura General Hospital, Sri Lanka*

Correspondence: Dr. A.H.M Makarim MBBS

E-mail: makarimlk@gmail.com

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CASE REPORT

Mrs. THD, 33 years old G4P2C2 mother weighing 68kg, presented with acute complete loss of vision on both eyes with frontal headache on 8th day after an elective lower segment caesarian section. She had a normal vaginal delivery 2 years ago following 2 first trimester miscarriages. This pregnancy was complicated with gestational diabetes & pregnancy induced hypertension at 37 weeks. Both were managed without drugs. Elective Caesarean Section was done due to large baby (4.5Kg) at local hospital, which was uncomplicated & she was discharged on day 2 with plan of monitoring blood pressure. On 4th day of delivery she has had high blood pressure (150/90mmHg) and on 7th day it was 200/110mmHg. She was given oral Nifedipine 20mg by a GP and four hours later she experienced acute loss of vision. She was admitted & managed at local hospital. She was transferred to tertiary care hospital for specialized management on same day. On admission she had frontal headache with total loss of bilateral vision, had no fever or per vaginal bleeding. On examination she was otherwise well-looking, afebrile, mild ankle edema, with BP 150/100mmHg & PR of 64 bpm. Had no neck stiffness, calf tenderness or clonus. Funduscopy finding was normal & pupils were equally reactive to light. Her urine albumin was 1+. Rest of the neurological examination was unremarkable. She was managed as pre-eclampsia with captopril. MRI scan of Brain (with MR Arteriogram & MR Venoragm) was done & found to have bilateral symmetrical cerebral ischemia/ vasogenic edema of cortical & sub-cortical regions of parieto-occipital areas and water shed areas of frontal lobes. (MRA & MRV study were normal). Clinical and radiological findings were assumed to be consistent with PRES. Her Preeclampsia investigations, D-dimer (200ng/dl), Serum Calcium (7.9mg/dl), ANA (Negative), Serum Magnesium (2.2mg.dl), cardiolipin antibodies (Negative) and clotting profile were normal. She was added IV Dexamethasone &Prozocine with captopril. On the 3rd day of admission, she had gradual improvement in vision & had complete regaining of vision on 8th day. During this period she had fluctuating BP recording & managed with drugs. She was discharged on 11th day with captopril 25mg tds. She was followed up at neurology & ophthalmology clinic. She recovered completely without residual effect.

DISCUSSION:

Acute loss of bilateral vision is alarming to both patient & doctors. A post-partum woman presenting with hypertension and blindness following delivery constitutes a diagnostic dilemma[4]. The possibilities that must be kept in mind include cerebrovascular haemorrhage, eclampsia, and clinical syndromes like PRES. Hypertensive retinopathy, exudative retinal detachment, and cortical blindness are three common visual complications of preeclampsia and eclampsia. Currently, blindness in severe preeclampsia is more likely to be associated with cortical aetiology[5]. Preeclampsia and its variants affect approximately 5% of pregnancies and remain leading causes of both maternal and fetal morbidity and mortality world-wide[6].

Our patient had normal funduscopy with normal CT scan of Brain.MRI revealed the diagnosis of PRES. She had a short history of hypertension & her symptoms of visual loss improved after Nifedipine. Immunosuppressive drugs, cocaine & NSAIDs are associated with PRES syndrome. A study done by Steven J Wagner in 2011 suggest a

strong association between eclampsia and characteristic neuro-radiologic PRES findings[7]. Visual abnormalities that accompany PRES include hemianopia, visual neglect, auras, visual hallucinations, and cortical blindness[(8)].

Fluctuating BP in our patient was managed by drugs with intensive monitoring & supportive treatment. So the recovery was rapid& complete. The visual loss is usually regained in PRES within a period of 4 hours to 8 days after treatment[(8)]

The pathophysiology of PRES is under debate, but it is related to disordered cerebral autoregulation. There are three theories responsible for the development of PRES[9.] which include over reaction of brain autoregulation resulting in reversible vasospasm, increase perfusion pressure due to hyper-perfusion, allowing extravasation of fluid into the brain parenchyma and intravascular pressures resulting in rupture of the capillary wall with haemorrhage[9].

The most characteristic imaging pattern in PRES is the presence of edema involving the white matter of the posterior portions of both cerebral hemispheres, especially the parieto-occipital regions, in a relatively symmetric pattern that spares the calcarine and paramedian parts of the occipital lobes[10,11,12].

The differential diagnosis of PRES is broad, and history may be limited. Venous sinus thrombosis or subdural, intracerebral, or subarachnoid hemorrhage, infective encephalitis or meningitis, particularly herpes simplex encephalitis should be considered. It is important to consider the diagnosis of posterior circulation stroke, because both treatment and prognosis is differ from those in PRES[(13)].

CONCLUSION

Since PRES is often unsuspected by clinicians, it should be considered in patients who present with seizures, altered consciousness, visual disturbance, or headache, particularly in the context of acute hypertension. Typical MRI findings include reversible, symmetrical, posterior subcortical vasogenic edema. Control of blood pressure is vital to avoid irreversible damage to central nervous system.If recognized and treated promptly, the rapid-onset symptoms and radiologic features usually fully resolve within days to weeks. ■

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