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Case Series

A study of posterior reversible encephalopathy syndrome as a cause of sudden transient vision loss during pregnancy

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ABSTRACT

Aim: To emphasize the importance of Posterior Reversible Encephalopathy syndrome (PRES) as the cause of sudden vision loss during pregnancy in patients with acute haemodynamic instability like preeclampsia, eclampsia, severe PIH.

Materials and Methods: In this prospective observational study pregnant women within age group of 25-40 years presenting with acute onset of visual, neurological symptoms to the emergency department were examined. Blood pressure recorded. Patients vision was recorded using snellens chart at the time of presentation and after the recovery. Slit lamp examination, fundus examination, pupillary reactions were noted to rule out other causes of vision impairment. MRI was done to detect PRES. Patients were stabilised and their blood pressure was monitored and treated with intravenous anti hypertensives.

Results: 8 out of 10(80%) of the pregnant women presented with sudden vision impairment were preeclamptic and 2 out of 10(20%) were with features of eclampsia. Majority were in third trimester. All the patients were diagnosed early with PRES on MRI and regained vision within 2 days with immediate treatment with intravenous antihypertensives.

Conclusion: Posterior Reversible Encephalopathy syndrome has to be kept in mind as one of the differential diagnosis while examining the pregnant women with eclampsia, preeclampsia, PIH coming with sudden visual impairment.

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1. Introduction

Posterior Reversible Encephalopathy syndrome[1] is a clinicoradiological condition[2] characterized by visual disturbances, headache, nausea, vomiting, seizures, altered sensorium due to acute haemodynamic disturbances. MRI shows vasogenic edema of brain involving subcortical regions of bilateral parietal and occipital lobes predominantly.[1] Pregnancy is a high risk condition with hemodynamic abnormalities that includes preeclampsia, eclampsia, severe PIH. These women presenting with sudden loss of vision, neurological symptoms has to be suspected for PRES and managed accordingly. PRES can be associated with other conditions other than pregnancy like acute renal failure, acute hypertensive crisis, DIC, TTP, septic shock. This study was intended to bring into limelight the importance of detection of PRES among pregnant women with acute hemodynamic disturbances presenting with sudden vision loss, headache, nausea, vomiting, seizures etc with normal fundus findings, so that timely management can save the lives.

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2. Materials and Methods

A prospective observational study was done on pregnant women of age group 20-40 years presenting with complaints of sudden onset of diminution of vision associated with acute pre eclampsia, eclampsia, severe PIH were screened in a tertiary care hospital from March 2021 - April 2022. Of these 10 patients were selected based on normal anterior segment, no pupillary abnormality, no fundus abnormality, Covid RTPCR negative. Patients with other causes of sudden vision loss having retinal changes, focal neurological deficits were excluded from the study.

2.1. Procedure methodology

After written informed consent was obtained, patients were asked about the detailed history regarding vision loss and associated symptoms, relevant obstetric history and past medical history. Blood pressure was recorded by manual sphygmomanometer. Patient's vision was assessed using snellen's chart. Detailed anterior segment and fundus examination was done to rule out other causes of vision loss during pregnancy. After confirming anterior segment, fundus was normal, Patients were immediately sent for non-contrast MRI to diagnose PRES. Patients were reassured regarding the condition.

Patients were stabilized and blood pressure was controlled gradually with intravenous nicardipine (5-15mg/h) or labetalol (2-3mg/min) in a well-equipped ICU setup by a team of physician, obstetrician and neurologist. Symptoms like seizures, vomitings were managed accordingly. Electrolyte disturbances were corrected if any and supportive care given with hydration. Timely caesarean section was done.

All patients regained vision approximately within 3 days and other neurological symptoms were relieved. Patients vision was again reassessed with snellen's chart. Fundus examination was done daily which was normal in all patients. MRI picture was normal after the treatment.

3. Results

All 10 patients presented with cortical visual impairment suddenly. Out of these 10 cases, 8 women had pre eclampsia (80%), 2 women showed eclampsia features (Table 1). Out of 10, 6 patients are from rural area and other 4 patients from urban area. Socio economic status, occupation, educational status of the patients were not relevant to be associated with the syndrome. These parameters can be assessed with more sample size. Most cases appeared during third trimester. PRES can also occur during postpartum period. All patients regained normal vision with immediate intervention. Immediate management for all patients helps in the recovery of the condition. Most patients regained vision approximately within 2 days of treatment.

Table 1: Pregnant women presenting with preeclampsia and eclampsia

Acute hemodynamic instability conditions	Total no of cases	Percentage
Pre eclampsia	8	80%
Eclampsia	2	20%

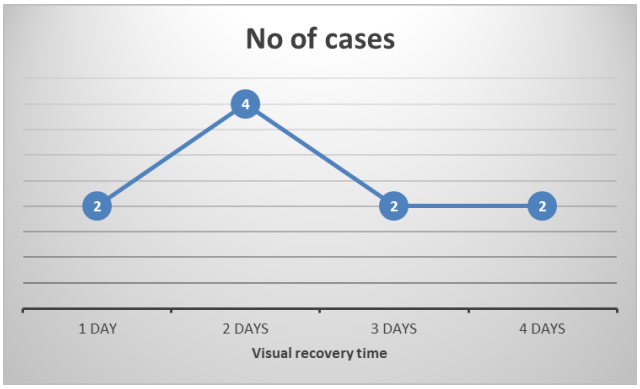

Fig. 1: Time taken for visual recovery

4. Discussion

PRES is a rare neurotoxic state that presents with visual disturbances, altered mental status, neurological signs like headache, nausea, vomiting, seizures in patients with acute hemodynamic instability, most commonly among pregnant women with eclampsia, pre eclampsia, PIH.[3] Visual disturbances like cortical blindness, blurred vision are more common in eclampsia related PRES.[4] MRI shows vasogenic edema of brain involving sub cortical regions of bilateral parietal and occipital lobes predominantly.[3]

Exact pathogenesis was not clear. Well known accepted theory was vasogenic theory. Cerebral blood flow remains steady and constant despite of alterations in cerebral perfusion pressure because of auto regulatory mechanism. When elevation in systemic blood pressure exceeds the normal cerebrovascular auto-regulation capacity, there will be failure of cerebral autoregulatory mechanism. Regions of vasoconstriction and vasodilation develop. Breakdown of blood brain barrier occurs.[5] At the capillary level disruption of end capillary pressure leads to hyperperfusion, extravasation of plasma and macro molecules causing secondary vasogenic edema. The upper limit of cerebrovascular autoregulation capacity is approximately 150-160mmHg. This range can extend up to 30mmHg higher in acute sympathetic states because of the rich sympathetic innervation of the majority of the cerebral vasculature. However, because of lack of sympathetic innervation in the posterior fossa, the parieto-occipital regions of the brain are more susceptible to hyperperfusion.[6] Another theory states that sudden rise in blood pressure causes vasospasm of cerebral vessels

Table 2: Best corrected visual acuity recorded at the time of presentation and after treatment

Case		Age	BCVA at the time of presentation	Recovery time	BCVA after treatment
1	Pre eclampsia	23	CF 1 meters	2 days	6/6
2	Pre eclampsia	29	3/60	2 days	6/6
3	Pre eclampsia	26	4/60	3 days	6/6
4	Pre eclampsia	30	CF 2 meters	8 hours	6/6
5	Pre eclampsia	25	3/60	12 hours	6/6
6	Pre eclampsia	32	CF CF	4 days	6/6
7	Pre eclampsia	23	PL + ,PR accurate	2days	6/6
8	Pre eclampsia	33	PL +, PR accurate	4 days	6/6
9	Eclampsia	35	CF 1 meters	3 days	6/9
10	Eclampsia	23	CF 2 meters	2 days	6/6

CF - Counting fingers, CF CF - Counting fingers close to face, PL- Perception of light, PR - Projection of rays

Table 3: Blood pressures at presentation and after treatment

Case		Age	Blood pressure at the time of presentation in mm of Hg	Blood pressure after the treatment with anti hypertensives in mm of Hg
1	Pre eclampsia	23	180/100	120/90
2	Pre eclampsia	29	200/100	120/80
3	Pre eclampsia	26	150/100	110/80
4	Pre eclampsia	30	160/100	110/90
5	Pre eclampsia	25	150/90	120/80
6	Pre eclampsia	32	190/110	110/80
7	Pre eclampsia	23	180/100	120/90
8	Pre eclampsia	33	190/100	120/70
9	Eclampsia	35	180/110	110/80
10	Eclampsia	23	150/100	120/90

causing cerebral ischemia leading to cytotoxic edema and infarction.[5,6]

MRI is particularly useful in the diagnosis of PRES. There is increased signal intensities on T2 and fluid-attenuated inversion recovery (FLAIR)[7]imaging of subcortical white matter with vasogenic edema. Cortical grey matter can be involved, depending upon the severity of the disease, predominantly involving the parieto-occipital and posterior temporal lobes of both hemispheres of the brain. Diffusion-weighted (DWI) MRI reliably distinguishes vasogenic oedema in PRES from cytotoxic oedema in the setting of cerebral ischemia.[8] Based on the extent of involvement, severity and prognosis can be assessed and treated aggressively.

Pregnant women presenting with PRES features with elevated blood pressure should be treated as hypertensive emergencies. The blood pressure should be reduced gradually not more than 25 percent within the first few hours of treatment to avoid risk of cerebral, coronary, renal ischemia. First-line agents for PRES related hypertensive emergency include intravenous nicardipine, labetalol, nimodipine.[9] Prompt delivery has to be considered.[10] Seizures if present, has to be managed with antiepileptics.

The reversibility of the imaging findings, may take days to weeks following initiation of treatment. When unrecognized or if treatment is not promptly initiated, PRES

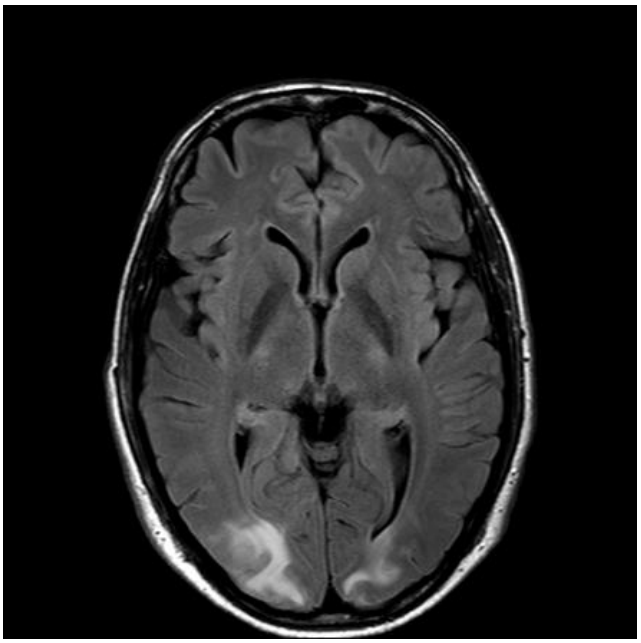

Fig. 2: Axial section of MRI brain in flairsequence showing high signal intensity in bilateral occipital cortex

may progress to cerebral infarction or hemorrhage and death.

5. Conclusion

Posterior Reversible Encephalopathy syndrome has to be diagnosed among pregnant women with elevated blood pressure and hemodynamic disturbances presenting with sudden visual deterioration. Early diagnosis of the condition by MRI and immediate treatment reverses the condition.

6. Conflict of Interest

None.

7. Source of Funding

None.

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