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CASE REPORT

Post Partum Eclampsia Complicated with Posterior Reversible Encephalopathy Syndrome: A Case Study

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ABSTRACT

Background: Posterior reversible encephalopathy syndrome (PRES) is a rare but serious clinical neuroradiological entity characterized by headache, visual disturbances, vomiting, seizures, altered mental status and unconsciousness with characteristic imaging findings including sub-cortical vasogenic edema at the bilateral parietal and occipital lobes.

Objective

1. To identify potential risk factors, signs and symptoms that can lead to PRES syndrome.
2. To promptly diagnose this condition with the help of imaging modalities so that timely treatment can be started.
3. This condition is to be kept in mind as a differential diagnosis in comatose patient of pregnancy with uncontrolled hypertension or history of convulsions (severe pre-eclampsia and eclampsia).
4. Explain importance of knowledge and communication among interprofessional team to enhance patient care and management with patient of PRES.

Method: In this case study, we describe a case of 21 year old PRES patient with post partum eclampsia. The diagnosis of PRES was reached after report of CT brain.

Conclusion: Inspite of treatment, the patient could not survive.Early diagnosis and aggressive treatment should be kept in mind for such patients.

KEYWORDS

PRES (posterior reversible encephalopathy syndrome) • eclampsia • Mental status • Unconsciousness

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INTRODUCTION

PRES (Posterior reversible encephalopathy syndrome) is a neurological disorder characterized by a range of neurological signs & symptoms and distinctive neuroimaging finding reflecting vasogenic edema.[1]

The most common clinical presentation includes elevated arterial blood pressure. PRES frequently develops in the context of cytotoxic medication, (pre) Eclampsia, Sepsis, renal disease or autoimmune disorder.[2]

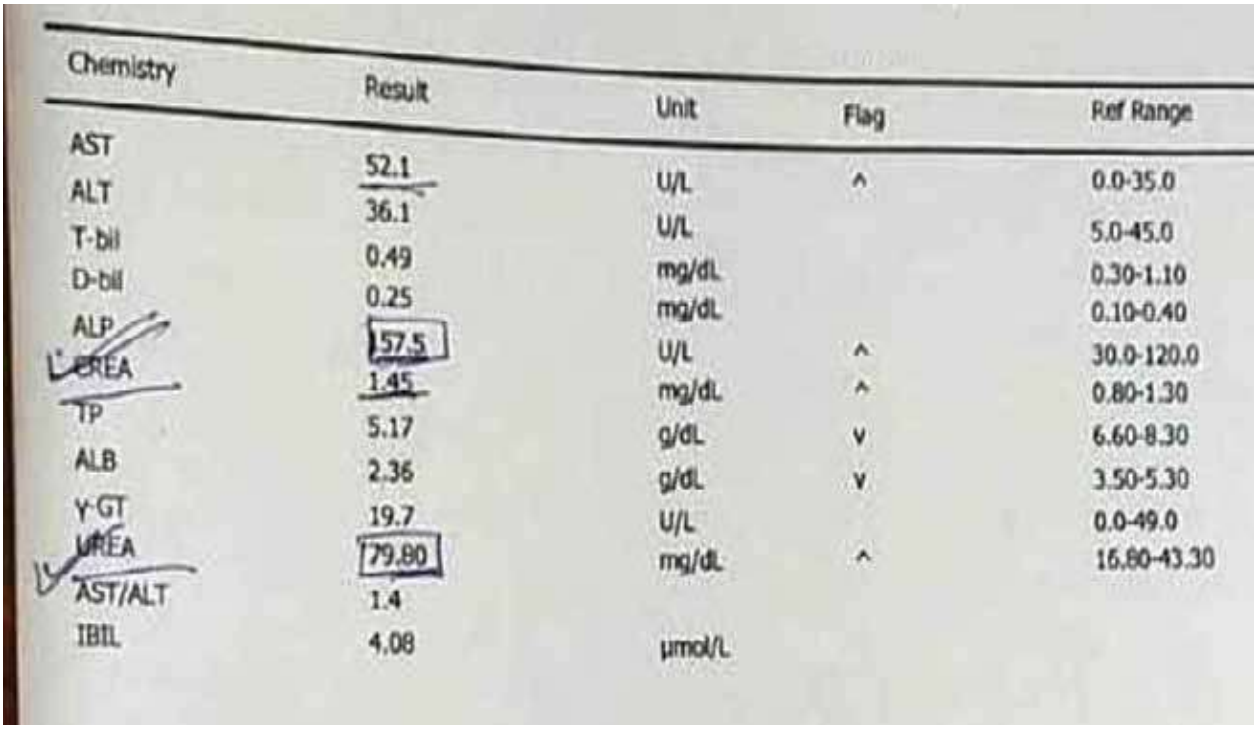
It can present with a variety of symptoms such as altered mentation or stupor, drowsiness, visual disturbances (e.g Visual hallucination, cortical blindness, hemianopia, quadrantanopia and diplopia), seizures (focal or general tonic-clonic) and headaches.[3]

In a retrospective study which involved the review of demographics, risk factors and clinical presentation of 113 patients with PRES, Fugate and colleagues found that 51 individuals (45%) had an autoimmune disease with most of the individuals presenting with seizures (74%) followed by encephalopathy (28%)headache (26%) and visual disturbance (20%).[4]

CASE PRESENTATION

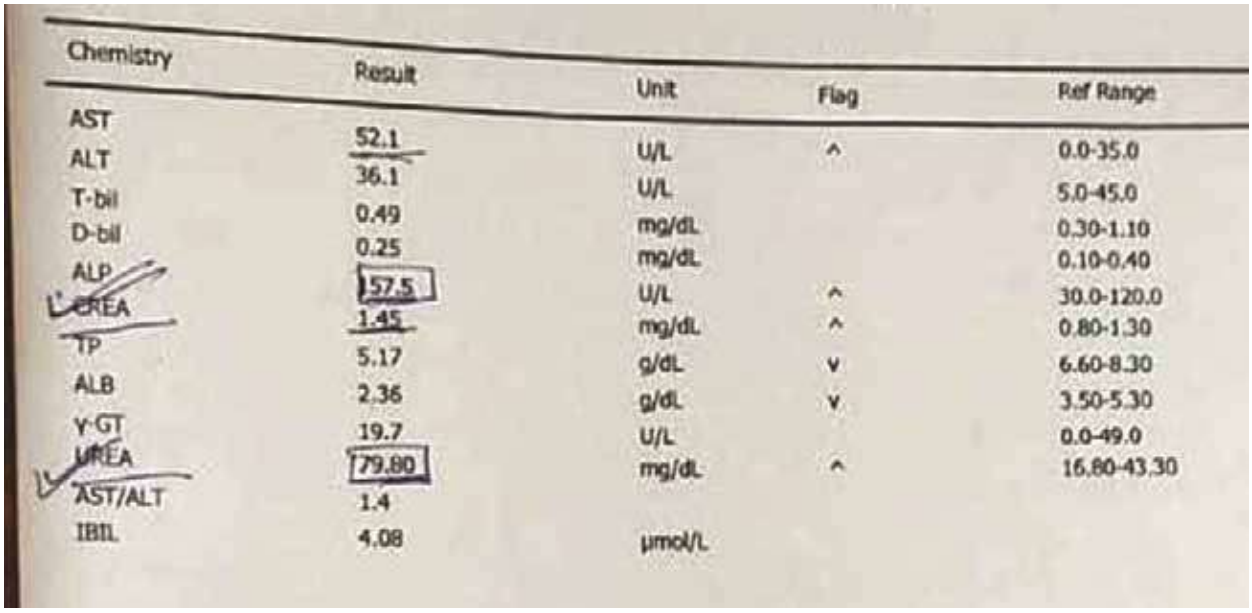
A patient was referred to Shaheed Nirmal Mahato Medical College and hospital, Dhanbad from a district hospital (Sadar, Deoghar). She was 21 yrs old with provisional diagnosis of post partum eclampsia after lower segment caesarean section (LSCS) done on 16.12.2024. She was primigravida with preterm pregnancy of gestational age 34 weeks 6 days. Indication of LSCS was severe oligohydraminos. She had multiple episodes of convulsion on 2nd day of LSCS. She was referred to higher centre on 3rd day of LSCS.

On examination, patient was semiconscious, disoriented. The Glasgow Coma Scale (GCS) was 10. Her BP was 128/72mmHg, PR-110/min, SPO2- 92% in room air, 97% on 6 liters of oxygen. Conservative management done, antibiotics started, Pritchard regimen started. Investigations sent were CBC, LFT, RFT, RBS, BT-CT, HIV, URINE R/E and C/S, ECG. On next day, patient was semiconscious, responding to deep stimuli. Physician call was sent according to which Levipril, Infusion Mannitol and inj. Meropenem was started. Her laboratory investigations were Hemoglobin 10.2gm/dl, TLC-16000 cells/cmm, Platelets count- 1.3 lakh /cmm, S. creatinine 1.49mg/dl, S. Urea – 79.8mg/dl, D. Bil-0.25mg/dl, Indirect Bil. – 4.08mg/dl, TP- 52.1g/dl, AST – 52.1 U/L, ALT- 36.1 U/L, AST/ALT- 1.4, Albumin 36.1u/l, Gamma-Globulin- 19.7 U/L.



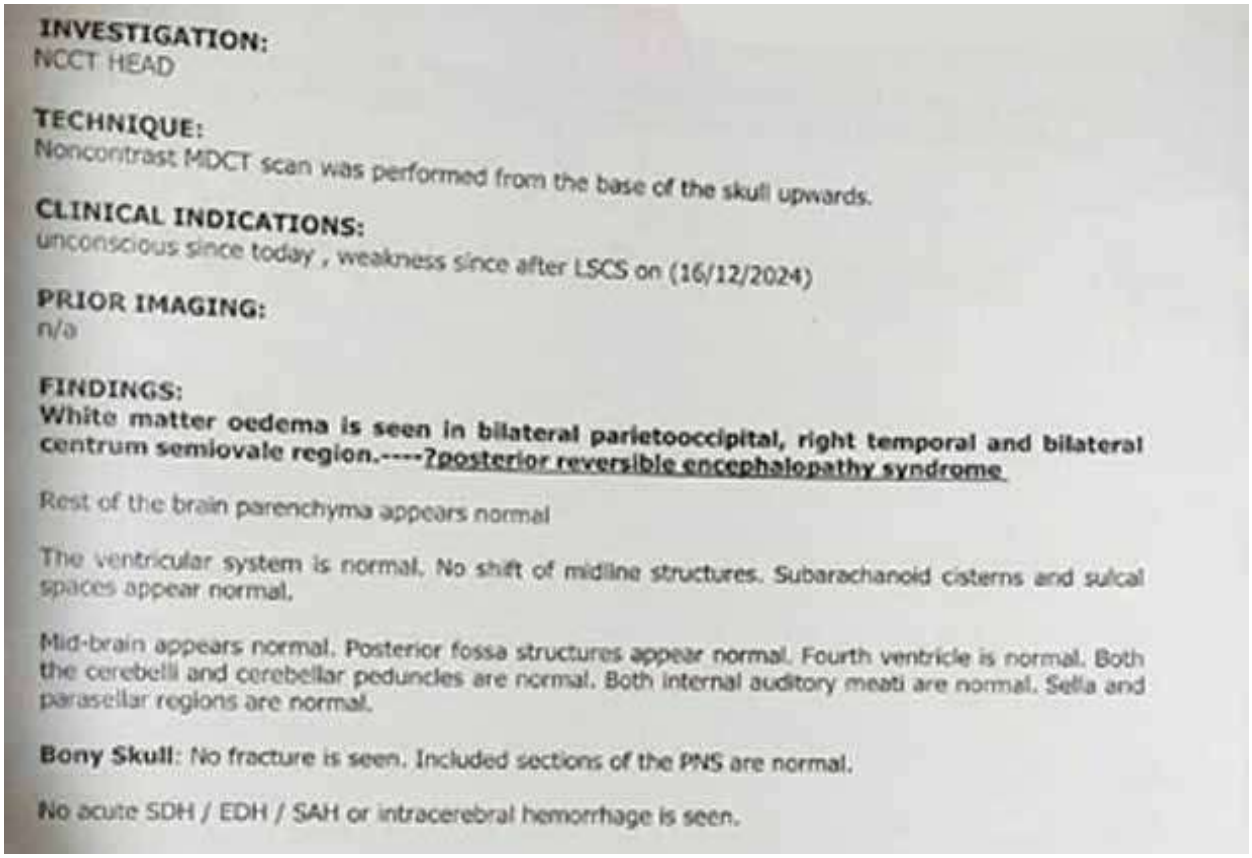
Chemistry	Result	Unit	Flag	Ref Range
AST	52.1	U/L	^	0.0-35.0
ALT	36.1	U/L		5.0-45.0
T-bil	0.49	mg/dL		0.30-1.10
D-bil	0.25	mg/dL		0.10-0.40
ALP	57.5	U/L	^	30.0-120.0
UREA	1.45	mg/dL	^	0.80-1.30
TP	5.17	g/dL	v	6.60-8.30
ALB	2.36	g/dL	v	3.50-5.30
Y-GT	19.7	U/L		0.0-49.0
UREA	79.80	mg/dL	^	16.80-43.30
AST/ALT	1.4			
IBIL	4.08	µmol/L		

Fig. 1: NCCT brain report showing PRES syndrome



Chemistry	Result	Unit	Flag	Ref Range
AST	52.1	U/L	^	0.0-35.0
ALT	36.1	U/L		5.0-45.0
T-bil	0.49	mg/dL		0.30-1.10
D-bil	0.25	mg/dL		0.10-0.40
ALP	157.5	U/L	^	30.0-120.0
UREA	1.45	mg/dL	^	0.80-1.30
TP	5.17	g/dL	v	6.60-8.30
ALB	2.36	g/dL	v	3.50-5.30
Y-GT	19.7	U/L		0.0-49.0
UREA	79.80	mg/dL	^	16.80-43.30
AST/ALT	1.4			
IBIL	4.08	µmol/L		

Fig. 2: NCCT brain report showing PRES syndrome



INVESTIGATION:
NCCT HEAD

TECHNIQUE:
Noncontrast MDCT scan was performed from the base of the skull upwards.

CLINICAL INDICATIONS:
unconscious since today , weakness since after LSCS on (16/12/2024)

PRIOR IMAGING:
n/a

FINDINGS:
White matter oedema is seen in bilateral parietooccipital, right temporal and bilateral centrum semiovale region.----?posterior reversible encephalopathy syndrome

Rest of the brain parenchyma appears normal

The ventricular system is normal. No shift of midline structures. Subarachanoid cisterns and sulcal spaces appear normal.

Mid-brain appears normal. Posterior fossa structures appear normal. Fourth ventricle is normal. Both the cerebelli and cerebellar peduncles are normal. Both internal auditory meati are normal. Sella and parasellar regions are normal.

Bony Skull: No fracture is seen. Included sections of the PNS are normal.

No acute SDH / EDH / SAH or intracerebral hemorrhage is seen.

Fig. 3: Lab Report

On next day, 20th December 2024, patient become deeply comatose, febrile. Her GCS was less than 8. Her BP114/72 mmHg, PR- 160/min, SOP2-96% with 6 liters of oxygen, PR-40/ min, Temperature – 106.4°F. Her NCCT Scan showed: white matter oedema is seen in bilateral parieto occipital, right temporal and bilateral antrum semi ovale region ? posterior reversible encephalopathy syndrome. Patient required intensive critical care and was referred to RIMS, Ranchi for better treatment. Patient eventually died the same day inspite of all life saving efforts.

DISCUSSION

The term malignant PRES has been defined based on clinical criteria (Glasgow Coma Score <8 and clinical decline despite standard medical management for elevated intracranial pressure) and radiological criteria (edema with mass effect, intracerebral hemorrhage exerting mass effect, effacement of basal cisterns, transtentorial, tonsillar, or uncal herniation).[5]

Management of malignant PRES requires aggressive supportive care. In such cases, besides routine care like mechanical ventilation, transfusion of blood products for reversal of coagulopathy, steroids for autoimmune disorders, intracranial pressure monitoring is required in patients with GCS of ≤8 (5). Our patient also deteriorated rapidly inspite of general favorable prognosis of PRES leading to probable diagnosis of malignant PRES.

Eclampsia is an important and frequent etiology of PRES.(6,7)There is a scarcity of literature about pregnancy- and eclampsia-related risk factors for developing PRES in eclampsia patients.[6,7]

Pre-eclampsia has the incidence of between about 3–8.7%, the incidence of eclampsia is less than 10/10,000 birth in most countries.[8] In addition to the typical pre-eclampsia/eclampsia, individual atypical cases may occur before 20 weeks of pregnancy or 48 hours after giving birth. There are a few atypical cases without the appearance of hypertension or proteinuria.[9]

PRES presents as a series of non-specific signs and symptoms, which mainly include headache, vomiting,altered mental status, visual disturbances, seizures, and unconsciousness, with the most common ones being seizure and headache.[10]

Typical imaging findings of PRES include reversible vasogenic subcortical edema at bilateral parietal lobes and occipital lobes. Frontal lobes, temporal lobes and cerebellar hemisphere are often involved. Some atypical sites may include the basal ganglia, brainstem and deep white matter. In addition, edema may also occur unilaterally and show asymmetry.[11]

Differential diagnosis of PRES

PRES needs to be differentiated from other diseases, mainly cerebral venous sinus thrombosis (CVST) and ischemic stroke but also from acute or sub-acute cerebrovascular diseases, central nervous system infections and autoimmune diseases.[12]

Treatment of PRES

With correct diagnosis and timely and effective early treatment, the patient prognosis is usually good. Neurological symptoms, signs and radiographic changes disappear completely in 1–2 weeks, while delayed diagnosis and/or incorrect treatment likely cause irreversible neurological damage or even death. Treatment includes treatment of the primary disease, control of neurological symptoms and hypertension.[13]

For Pre-Eclampsia/Eclampsia induced PRES, the main treatment is to control hypertension and prevention or treatment of seizure and termination of pregnancy. The anticonvulsant of choices magnesium sulphate. Other drugs include antihypertensive like labetalol, Nifedipine, Corticosteroids such as Dexamethasone.

Our patient was a case of post partum eclampsia complicated with PRES. This patient had multiple episodes of convulsion following LSCS due to which her GCS had deteriorated and her CT scan showed PRES. Inspite of all evident therapy given to patient, the patient could not survive.

CONCLUSION

In Eclampsia patient with poor GCS score, inspite of protocol treatment patient does not respond. We may suspect more brain damage, So CT brain should be done. PRES is a rare finding in eclampsia patient but obstetrician should be aware of this rare complication with such patient. The key to full recovery of PRES is early diagnosis and treatment. Misdiagnosis and delayed treatment may cause neurological permanent damage.

Conflict of Intestate: The authors have declared no competing interests exist.

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Ethics Declaration: Consent was obtained by the participant's attendant in this study.

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