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Case Report

Headache and seizure on postpartum day 7: late postpartum eclampsia - a case report

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ABSTRACT

Historically, convulsions beginning more than 48 hours, but less than 4 weeks, after delivery, known as late postpartum eclampsia, was thought to be uncommon; however, recent evidence suggests that its incidence is increasing. In addition, the presentation of late postpartum preeclampsia-eclampsia may differ from that occurring during the pregnancy. About 40% of late eclampsia has no premonitory symptoms. This contributes to difficulty in diagnosing late postpartum preeclampsia-eclampsia in an emergency department setting. Greater awareness and knowledge of this disorder by ED physicians should improve outcomes in these potentially life-threatening cases. The authors present a case of new-onset seizures occurring 07 days postpartum. The patient presented with headache, hypertension, and generalized tonic-clonic seizures to the emergency department. Postpartum eclampsia was diagnosed and IV magnesium sulphate was administered. The patient had no further seizures and did not require long-term anticonvulsants.

Keywords: Late postpartum eclampsia, Gestational hypertension, Pre-eclampsia, Eclampsia, Seizures, Hypertension, Pregnancy, Magnesium sulphate, Maternal death, Cerebral venous thrombosis, Cerebral venogram

INTRODUCTION

Hypertensive disorders of pregnancy cause 14% of all maternal deaths globally, approximately 42,000 each year.[1,2] Nearly all of these deaths occur in low-resource settings (99%), with death in high-income settings being very rare.[3] Hypertensive disorders of pregnancy encompass chronic hypertension, gestational hypertension (new hypertension without proteinuria), pre-eclampsia (new hypertension with proteinuria or end-organ damage after 20 weeks of gestation), and eclampsia. The majority of morbidity and mortality is associated with pre-eclampsia and eclampsia.[4]

It is estimated that the prevalence of pre-eclampsia globally is 4.6% (95% CI 2.7%–8.2%).[5] The prevalence of eclampsia globally is reported to be 0.3%.[6] This is based on secondary analysis of a World Health Organization (WHO) multi-country survey that included 875 cases of eclampsia, collected over a short duration from only secondary or tertiary hospitals.[6] Women under 20 years of age, women with low levels of education, and women in their first pregnancy are all reported to be at higher risk.[6] Reliable data reporting the prevalence of maternal deaths related to eclampsia globally are scarce. Estimates from 16 datasets reported the case fatality rate to be 8.3%, whereas the WHO survey reported 32 maternal deaths, 3.7% of women with eclampsia.[5,6] Data from individual countries suggest that prevalence and mortality risk vary depending on region and socio-economic status.[7]

Administration of magnesium sulphate more than halves the risk of eclampsia in women with pre-eclampsia.[8] It is considered an essential drug by WHO, but data on its availability in relation to prevalence of eclampsia are scarce.[5,9] Planned delivery after 36 weeks of gestation is

effective at preventing maternal morbidity in women with pre-eclampsia.[10] Evidence for other interventions effective at reducing morbidity and mortality of pre-eclampsia is mixed, and research is generally undertaken in high-income settings, where the burden of illness is small.[11] There is a lack of understanding around modifiable risk factors and availability of life-saving interventions, both vital in reducing the high number of deaths from this treatable cause.

Late postpartum eclampsia constituted 56% of total postpartum eclampsia and 16% of all cases of eclampsia. Convulsions began from postpartum days 3-23 (mean 6). Thirty women (56%) had been identified as preeclamptic before their convulsions. A history of either severe headache or visual disturbances before convulsion was elicited in 83% of the patients. Severe headache or visual disturbance frequently antedates late postpartum eclampsia.[12]

CASE REPORT

A previously healthy 33-year-old woman P_2 L_2 A_1 with previous two LSCS was brought in A&D department of a Military Hospital at POD 07 with history of generalised tonic-clonic seizure at her home and again at the A&D department on arrival. She had an unremarkable antenatal period in all her pregnancies. She underwent LSCS with B/L tubectomy on 21 August 2021 and was discharged on 25 August 2021 having an uneventful immediate post op pd. She had no history of pregnancy associated hypertension or preeclampsia during her antenatal period or in the post-op period during her stay at the hospital. Her liver function test (LFT), S. urea, S. creatinine, platelet count (145000) and Hb- (10.8 gm%) were with in normal limit during her antenatal period.

After discharge the patient went home where she developed a headache on 26 August 2021 which continued for 2 days and culminated in generalised tonic-clonic seizure on 28 August 2021. She again had a second episode of generalised tonic-clonic seizure at the A&E department.

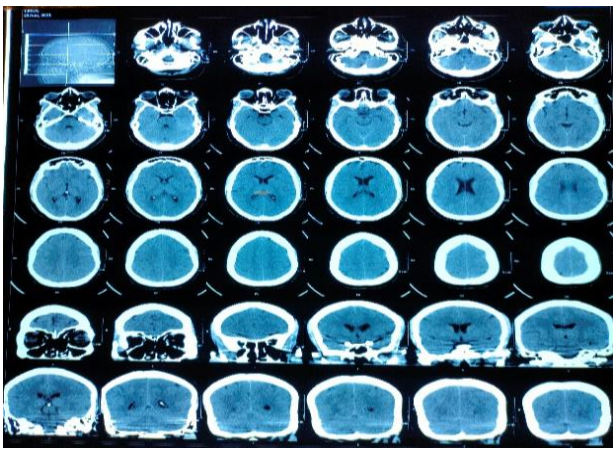

Figure 1: NCCT head.

Her BP recordings at A&E on arrival was 160/98 mmHg. She was drowsy, confused but afebrile. Her pulse and respiratory rate were 90 BPM regular and 20/min respectively and she was maintaining SPO_2 at 99% on room Air. Local evidence of tongue bite and frothing of the mouth was present. At A&D department she was managed by inj. levetiracetam 1 gm i/v, lorazepam and injection labetalol 20 mg iv stat and was admitted to ICU and was started with 2 gms per hour $MgSO_4$ infusion for the next 24 hours.

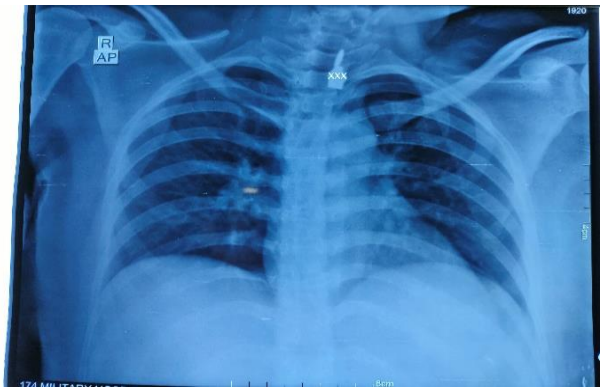

Figure 2: Chest X-ray.

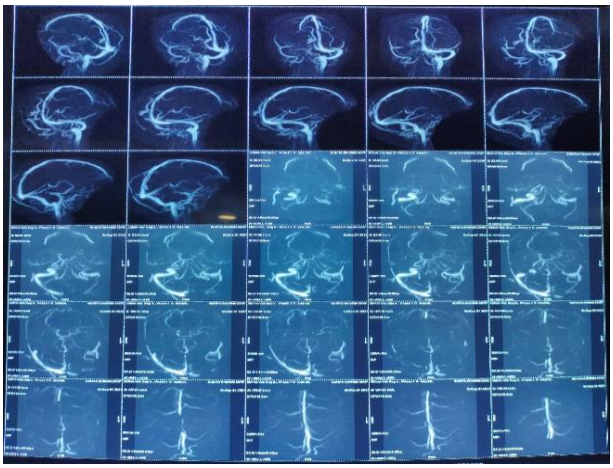

Figure 3: Cerebral venogram.

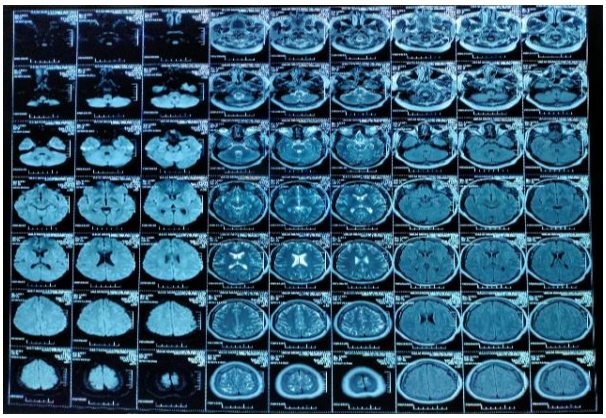

Figure 4: MRI brain.

She was extensively evaluated with NCCT head which showed normal findings. Chest X-ray PA view within normal limits. Her B/L renal doppler studies were also within normal limits. The MRI brain and cerebral venogram showed left hypoplastic/atretic transverse sinus with no flow related contrast enhancement. Otherwise, there was no significant abnormality seen in the brain parenchyma and cerebral venogram.

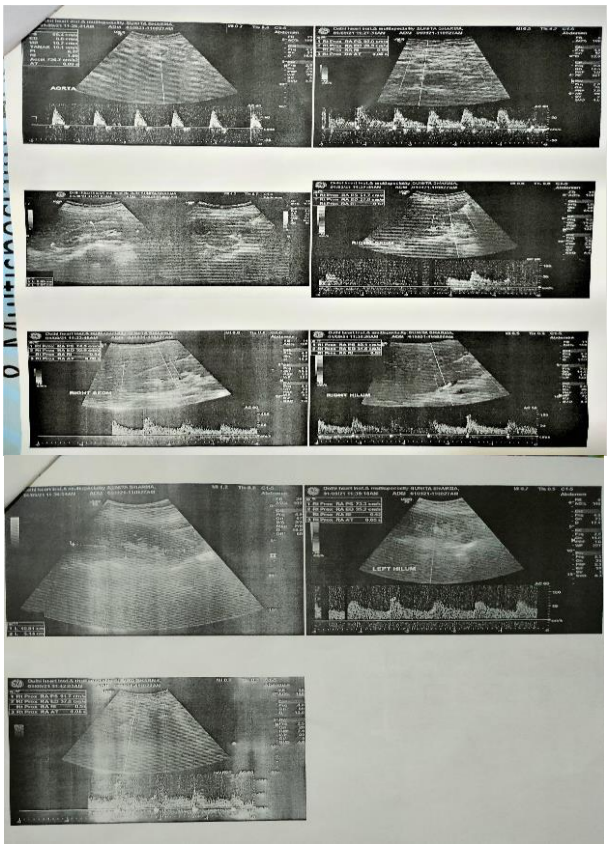

Figure 5: B/L renal artery doppler studies.

DISCUSSION

The previously controversial existence of a delayed postpartum variant of eclampsia is now acknowledged by most experts.[13-19] Convulsions with initial presentation more than 48 hours but less than four weeks after delivery are commonly referred to as late postpartum eclampsia.[17]

Our patient had convulsions on the seventh postpartum day. Medical and neurologic evaluations failed to reveal any other etiology for the seizures and the exclusion of metabolic and infectious causes strongly support the diagnosis of eclampsia.

Postpartum eclampsia can present with a variety of clinical and neurological symptoms and signs. Lubrasky and Chames reported that 44% and 79% of their respective patients with late onset postpartum eclampsia had not been identified as having preeclampsia before seizure onset.[16,17] They reported that severe and persistent headache, visual symptoms, epigastric or right upper quadrant pain, and hypertension can present as prodromal symptoms before the onset of eclampsia.[15-17] Our patient had these symptoms.

Eclampsia should be considered in any postpartum woman who develops any of these prodromal symptoms. Further indicators include convulsions up to four weeks after delivery, hypertension, or proteinuria. This is important as eclampsia is amenable to treatment with magnesium sulphate.

The differential diagnosis includes epilepsy, cerebral venous thrombosis, intracerebral haemorrhage, phaeochromocytoma, space occupying lesions, and metabolic disorders.[20] Neuroimaging, especially magnetic resonance imaging, shows micro-ischaemic injury patterns in parieto-occipital lobes.[21] This form of posterior leukoencephalopathy syndrome can cause cortical blindness, which may be reversible with control of hypertension and magnesium sulphate therapy. Lesions on a magnetic resonance scan cannot predict whether damage leading to cortical blindness is permanent or likely to be reversible.

Magnesium sulphate remains the drug of choice for preventing and treating eclamptic seizures. If such seizures are not treated appropriately, grave complications such as intracerebral haemorrhage and death can occur. Several studies have examined therapeutic protocols for the management of eclampsia. However, these protocols have not been evaluated in late postpartum eclampsia.[22] Because late postpartum eclampsia is considered a subtype of eclampsia, the same therapeutic approaches can be used. Intravenous magnesium sulfate therapy (bolus of 2–4 g delivered over 10–30 minutes, followed by an infusion of 1–2 g per hour for 24–48 hours) has been found to be superior to other anticonvulsive therapies.[22-24] However, excessive levels of magnesium sulfate can lead to hypotension, loss of reflexes and respiratory arrest.

Late onset postpartum eclampsia can occur in normotensive uncomplicated postpartum women as well as in women with pre-eclampsia. Although delivery of the newborn usually corrects the signs and symptoms of pre-eclampsia and eclampsia, this does not occur in the case of late postpartum eclampsia. When convulsions occur later postpartum, diagnosis is difficult and treatment disputed. The presence of prodromal symptoms should be thoroughly investigated, even in the absence of antecedent pre-eclampsia. Our patient had late postpartum seizure and hypertension that occurred without true pre-existing pre-eclampsia. We suggest that such patients seen in accident and emergency units within 14 days of delivery should be assessed by an experienced obstetrician.

Several agents can be used to treat hypertension in patients with pre-eclampsia, eclampsia and postpartum hypertension. Diuretics should be avoided, especially in combination with other antihypertensive agents, except in cases of life-threatening fluid overload.[23,24] This is

because, although one might expect patients with eclampsia to have increased total body fluids, they actually have a depletion of intravascular volume, and the use of diuretics can precipitate severe hypotension. The guidelines of the Society of Obstetricians and Gynaecologists of Canada state that, on average, longer durations of antihypertensive therapy are needed for women who have pre-eclampsia (about 2 weeks postpartum) compared with those who have gestational hypertension without proteinuria (1-week post-partum). The blood pressure treatment target is 130–155 mm Hg systolic and 80–105 mm Hg diastolic for those without comorbid conditions and 130–139 mm Hg systolic and 80–89 mm Hg diastolic for those with comorbid conditions. Nonsteroidal anti-inflammatory drugs should not be given after delivery if the patient's hypertension is difficult to control. Gestational hypertension usually resolves within 6 weeks after delivery; however, women with severe pre-eclampsia may have hypertension for several months after delivery.

CONCLUSION

With the recent trend of discharging new mothers soon after delivery (often within 1 day), patients with eclampsia may present to primary care physicians and emergency departments with early signs and symptoms of eclampsia. The early recognition of the signs and symptoms of postpartum eclampsia may lead to early treatment and fewer complications.

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