Case Report

Late-onset postpartum eclampsia: Do not ignore the headache!

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ABSTRACT

Pre-eclampsia and eclampsia continue to be a poorly understood neurological entity of pregnancy that substantially contributes to maternal morbidity and mortality. The onset of postpartum eclampsia occurring more than 48 h after the onset of the postpartum period is defined as late-onset postpartum eclampsia (LPE). We report the case of a 27-year-old woman who had eclampsia on the 6th postpartum day after cesarean section with a throbbing headache as the only prodromal symptom and normal blood pressure (BP) recordings at the time of convulsion. She was managed successfully with magnesium sulfate therapy. This case emphasizes that headache and/or visual changes developing during the postpartum period can be the only warning symptom of impending eclampsia and that LPE can occur even when the BP has normalized post-delivery. Early diagnosis and subsequent initiation of appropriate therapy prevent severe complications.

Key words: Cerebrovascular disorders, Eclampsia, Postpartum period, Pregnancy

Pre-eclampsia and eclampsia continue to be a poorly understood multisystem complication of pregnancy that substantially contributes to maternal morbidity and mortality [1]. Approximately one-half of all cases of eclampsia occur postpartum [2]. Convulsions with an initial presentation more than 48 h but <4 weeks after delivery are referred to as late-onset postpartum eclampsia (LPE) [3]. The latest onset of LPE that has been reported in the literature so far has occurred 89 days postpartum [4]. Unusual timing, however, may not be the only feature of LPE deviating from the classic diagnostic criteria of eclampsia. The combination of delayed manifestation after delivery and an atypical clinical presentation can pose a diagnostic challenge.

We report an unusual presentation of LPE in a patient in whom LPE manifested after a very short preceding pre-eclamptic phase, with normal blood pressure (BP) after delivery and only headache as the prodromal symptom.

CASE REPORT

A previously healthy 27-year-old woman (gravida 3 and para 2) had an unremarkable pregnancy till the 32nd week with normal BP and no evidence of proteinuria. Her previous obstetric and family histories were also non-significant. During the 32nd week, at the antenatal visit, pitting edema was noticed in both the feet. On examination, her BP was 150/90 mm Hg, heart rate (HR) was 104 beats/min (bpm), respiratory rate was 16/min, and patellar deep tendon reflexes were normal.

A urine dipstick test revealed a protein level of about 0.3 g/L. Her blood investigations were within normal limits. She was admitted and put on the tab. labetalol 100 mg 3 times daily and monitored closely. However, her BP kept rising over the next 2 days (170/110) and she then started complaining of dyspnea. The oxygen saturation (SpO_2) was initially 98%, but it gradually started decreasing and reached 94%. Chest auscultation revealed crept in bilateral bases. She was started on hydralazine infusion @ 50 µg/min, oxygen by face mask, and shifted to the operation theatre for an emergency cesarean section (CS) in view of impending pulmonary edema.

General anesthesia was administered for CS. She delivered a 1.8 kg healthy female baby with Apgar scores of 8 and 9 at 1 min and 5 min, respectively. The patient was shifted to the intensive care unit (ICU) for post-operative elective mechanical ventilation with a BP was 148/98 mm of Hg, HR of 112 bpm, and SpO_2 of 98%. She was gradually weaned off from the mechanical ventilation after 32 post-operative h and her trachea was extubated. Hydralazine was gradually tapered and tab. labetalol 100 mg 3 times daily was restarted. Her BP recordings were in the range of 120/70 mm Hg, and HR was 80–90 bpm. All the other vital parameters, including urine output, were within acceptable limits.

On the 6th post-operative day, when the patient was about to be shifted out of the ICU, she complained of a sudden-onset throbbing

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occipital headache that was not associated with photophobia or epigastric pain. Her BP was 126/76 mm Hg. Her other vital signs were unremarkable. Her neck was supple and non-tender, and her pupils were equal and reactive to light. The rest of her neurologic examination was unremarkable except for the increased patellar deep tendon reflexes. While she was being examined, she had a generalized tonic-clonic seizure with loss of consciousness that lasted for 2 min. The seizure was terminated by midazolam 2 mg IV. After the seizure, she was drowsy with a BP that was 130/80 mm Hg and HR of 110 bpm. She was administered oxygen by face mask and an intravenous magnesium sulfate infusion was started (4 g loading dose followed by 1 g/h) after making a provisional diagnosis of eclampsia in spite of a normal BP. She was also given a bolus of phenytoin 1000 mg intravenously, as we were as yet not sure of the cause of her convulsion.

Her blood analysis done after the seizure, including full blood count, liver function tests, serum electrolytes, and glucose was normal, as was her thyroid function tests, urinary catecholamines, and cerebrospinal fluid cytology and culture. Only her uric acid level was 8.9 mg%. Computerized tomography (CT) scan done a few hours later revealed no gross abnormality. She did not have any other episode of seizure and was shifted to the ward after 2 days, where the rest of her stay was uneventful. She was discharged home without any neurological sequelae.

DISCUSSION

By definition, our patient had LPE as she developed the seizure on the 6th postpartum day. Our diagnosis was based on her clinical course (pregnancy-induced hypertension with high BP and proteinuria and albeit of very short duration) and exclusion of underlying disorders. The differential diagnoses of postpartum headache followed by convulsions include encephalitis, meningitis, epilepsy, electrolyte or metabolic disturbances, space-occupying lesions, vasculitis, venous thrombosis and stroke, and side-effects of epidural analgesia [2,3]. Her response to magnesium sulfate, the exclusion of metabolic and infectious causes, and a normal CT scan strongly supported the diagnosis of eclampsia.

Lubarsky *et al*. [5] and Chames *et al*. [6] have reported that 44% and 79% of their patients, respectively, with LPE, had not been identified as having preeclampsia before seizure onset. 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. Santos *et al*. [3] and Redman *et al*. [7] have also reported headache as the most common symptom of impending eclampsia in their patients.

The majority of reported cases of LPE; however, late they occur, have been associated with high BP [3,8]. Munjuluri *et al*. [9] have reported that occasionally LPE can occur in normotensive women, our patient also had normal BP readings and was apparently well when she developed a convulsion. However, she did complain of headache (but no visual disturbances) which was promptly attended. This may not be the case when the patient is at home or in the ward of a busy maternity center. Moreover, headache is often a symptom that is dismissed lightly particularly in a mother of a newborn who is just coming to terms with her situation or simply suffering from lack of sleep.

Complications in eclampsia are common and can be fatal; more importantly, they are amenable to treatment [2,3]. Diagnosing this clinical condition correctly not only prevents complications but also avoids unnecessary investigations which may be both invasive and costly and result in treatment getting delayed. Because LPE is considered a subtype of eclampsia, the same therapeutic approaches can be used and early use of magnesium sulfate remains the mainstay of treatment [9]. Early initiation of magnesium sulfate therapy reduced morbidity in our patient.

The atypical presentation of LPE with normal BP and no proteinuria emphasizes the need to be aware of the possibility of this diagnosis. And that due importance should be given to headaches and/or visual changes that develop during the postpartum period as an important prodromal symptom by itself, which might otherwise be dismissed lightly. This will help address the issue of the lack of caretaker attention which constitutes another serious but potentially reversible obstacle to diagnosis and management in our kind of setups.

CONCLUSION

This case emphasizes the importance of considering headache and/or visual changes only developing during the postpartum period as a prodrome for LPE after delivery, even in those patients where BP has returned to normal. Early diagnosis and subsequent initiation of anticonvulsant therapy and antihypertensive if required and can prevent severe complications.

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