

Late postpartum eclampsia at five weeks post-delivery

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ABSTRACT

Postpartum eclampsia is a serious and unexpected complication. A relative increase in the incidence of postpartum eclampsia has been noted in the last few years. Late postpartum eclampsia, though initially controversial, is now recognised up to four weeks after delivery. We present such a case occurring in a 26-year-old woman. This patient, who had undergone lower segment caesarean section due to pregnancy-induced hypertension, presented with typical features of postpartum eclampsia 34 days after an asymptomatic interval.

Keywords: eclampsia, hypertensive pregnancy disorders, postpartum complications, postpartum eclampsia, pregnancy-induced hypertension

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INTRODUCTION

Eclampsia is a life-threatening complication of hypertensive pregnancy disorders. Late postpartum eclampsia occurring between 48 hours and four weeks is becoming increasingly recognised.⁽¹⁻⁴⁾ Late postpartum eclampsia occurring up to 23 days have previously been reported.^(1,2) We report a patient presenting, 34 days after delivery, with features of eclampsia. This case illustrates that postpartum eclampsia may occur beyond four weeks, and needs to be considered in the differential diagnosis.

CASE REPORT

A 26-year-old woman, 34 days postpartum, presented to our emergency department with complaints of headache and vomiting for the past four days. The headache was described as sharp and throbbing, and was localised at the back of her head. There was no history of photophobia or any visual changes. She had two episodes of generalised tonic-clonic seizures after the onset of headache. She had undergone an elective lower segment caesarean section (LSCS) 34 days earlier for pregnancy-induced hypertension and breech presentation. She was a booked primigravida with regular antenatal checkups.

She was diagnosed to have hypertension one week prior to her estimated date of delivery. After LSCS, her blood pressure normalised, without the need for antihypertensives. She was discharged in a satisfactory condition on the third postpartum day. She remained asymptomatic till her present admission.

On evaluation at the emergency department (ED), she was conscious and oriented with a Glasgow coma scale of 15/15. Her vital signs were stable with a blood pressure of 160/110 mmHg, pulse rate of 92/min, temperature of 37.1°C and respiratory rate of 16/min. The pupils were bilateral, equal and reactive to light. Extraocular movements were normal and fundus examination did not show any evidence of papilloedema or haemorrhage. There was no neurological deficit, and cranial nerve, motor and sensory examinations were normal. All deep tendon reflexes were mildly exaggerated and bilateral plantars were flexor. Cardiovascular, respiratory, and abdomen examinations were normal. Laboratory studies showed haemoglobin level of 10 g/dL, total leucocyte count of 7,500 cells/mm³ and platelet count of 83,000 cells/mm³. Urine examination showed a protein of 3+ and no active sediments. Renal function tests, arterial blood gas analysis, coagulation profile, chest radiograph and ECG were normal. Liver function tests showed a total bilirubin level of 1.7 mg/dL (normal 0.8–1.3 mg/dL), direct bilirubin 0.5 mg/dL (normal 0.6–1.0 mg/dL), alanine transaminase 28 IU (normal 10–35 IU) and aspartate transaminase 18 IU (normal 10–35 IU). A repeat liver function test after three days showed normal values. Computed tomography (CT) of the head was normal. Initially, the patient was evaluated for all other possible causes of seizures. As the clinical manifestations were typical of eclampsia and other investigations were unremarkable, the diagnosis of late postpartum eclampsia was considered. The patient was given a loading dose of phenytoin intravenously and was put on oral phenytoin subsequently. She was also started on amlodipine tablets for hypertension. She had no recurrence of seizures during her hospital stay and blood pressure was adequately controlled. She was discharged after four days of hospital stay in a satisfactory condition.

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DISCUSSION

Eclampsia is the most severe manifestation of pregnancy-related hypertensive disorders characterised by hypertension, proteinuria and seizures. This group of disorders is due to the pathophysiological changes occurring during pregnancy and should abate after delivery. The present theory for eclampsia proposes that vasospasm of cerebral arteries, in response to a paradoxical increase in thromboxane, results in cerebral ischaemia and cytotoxic oedema. Generally, eclampsia occurs either before or within 48 hours of delivery. Eclampsia occurring more than 48 hours but less than four weeks after delivery is known as late postpartum eclampsia.⁽¹⁻⁴⁾ Late postpartum eclampsia was initially thought to be very uncommon.⁽¹⁾ However, recent data suggests that the timing of eclampsia is changing in the developed countries and late postpartum eclampsia may account for 12%–16% of all eclampsia cases.⁽¹⁻⁴⁾ Lubarsky et al, in his study of 334 cases of eclampsia from 1977 to 1992, reported 97 (29%) cases of postpartum eclampsia, of which 56% occurred in the late postpartum period.⁽¹⁾ In a more recent study of eclampsia by Chames et al between 1996 and 2001, 33% of the cases occurred in the postpartum period, of which 79% was in the late postpartum period.⁽²⁾

Convulsions attributed to eclampsia have been noted up to 23 days postpartum.⁽³⁾ Early discharge of postpartum patients, infrequent follow-up in the postpartum period and an atypical presentation may be contributing factors for this changing trend.⁽⁵⁾ This may be all the more relevant in developing countries where eclampsia contributes to one-third of maternal mortality and where access to medical facilities is limited. Late postpartum eclamptic patients are more likely to present in the ED and investigated for all other probable causes.⁽⁶⁾ Seizures may occur in the postpartum period due to other causes, such as cortical venous thrombosis, hypertensive encephalopathy, intracerebral haemorrhage or metabolic disorders, including hypoglycaemia and hyponatraemia. These conditions will have to be ruled out by blood chemistry and CT/MR imaging before a diagnosis of late postpartum eclampsia is made.

The most common signs and symptoms heralding the onset of eclampsia include headache,

hyperreflexia, absolute or relative hypertension (defined as 30/15 mmHg increase over baseline), proteinuria, oedema, and visual disturbances. Late postpartum eclampsia may not present with all the classical symptoms of intrapartum eclampsia. Laboratory abnormalities, such as haemolysis, elevated liver transaminases, and low platelet count (HELLP syndrome) are seen in a minority of cases of late postpartum eclampsia.⁽⁴⁾ This makes the diagnosis of late postpartum preeclampsia-eclampsia more difficult. However, a study conducted to determine the predictive factors for postpartum eclampsia showed that normotensive women with an increase in mean arterial pressure (MAP > 10 mmHg) were found to have more than a threefold risk of readmission in the postpartum period with severe preeclampsia or eclampsia.⁽⁷⁾ The management for postpartum eclampsia is similar to antepartum or intrapartum eclampsia. The case presented here suggests that eclampsia may occur beyond the defined time duration of four weeks. This case once again reinforces that it is essential to be vigilant even in the postpartum period. A high index of suspicion will help in the early detection of this condition and avoid unnecessary investigations. Patients with antepartum preeclampsia-eclampsia and those with high risk of developing this condition may benefit from regular postnatal follow-up. Knowledge of the unusual presentations of eclampsia will facilitate prompt treatment with good recovery.

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