CASE REPORT

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Devastating Outcome of Late Onset Preeclampsia in a Multiparous Woman: A Case Report

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Abstract

Late onset preeclampsia may occur nascently in a multiparous woman. Although, early onset preeclampsia is associated with more adverse maternal and perinatal outcomes, late onset type in a multiparous woman can occur unexpectedly, and may progress rapidly to a life threatening situation. We report a case of late onset preeclampsia with HELLP syndrome and intracranial haemorrhage in a multiparous woman, who succumbed to death during the course of her treatment. We conclude that preeclampsia is a potential threat for dreadful outcomes, irrespective of its type, parity and duration of onset.

Keywords: HELLP syndrome; Intracranial haemorrhage; Late onset preeclampsia; Multiparity.

Declarations

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Preeclampsia can occur nascently in a multigravida, and therefore is not the disease of only primiparity. It is classified as early and late onset type, based on its occurrence before or after 34 weeks of gestation [1]. The early onset type is associated with more adverse maternal and fetal outcomes as compared to the late onset type [2, 3]. Prolonged exposure of the mother and fetus to ischemic consequences of abnormal placentation could be the possible reason. Nevertheless, late onset preeclampsia in a multiparous woman can be equally alarming as it develops unexpectedly, and can progress rapidly to life threatening conditions.

CASE

A 30-year-old female para two was brought to the emergency department of our tertiary care hospital on her second postpartum day with a history of tightening of limbs, frothing from mouth, up rolling of eyes, and loss of consciousness for one day. Two days back, she had delivered a healthy baby at 37 weeks + 5 days of gestation, with emergency caesarean section under spinal anesthesia at a district hospital. She was taken to the district hospital a day prior to the delivery after she developed burning epigastric pain, throbbing headache, and vomiting. Her blood pressure was 180/110 mm Hg on admission, blood investigations including liver function tests were within normal range. There was no history of hypertension in the past. Her first baby was born 12 years back, by normal vaginal delivery. She had regular antenatal care contacts (ANC) during this pregnancy, and her blood pressure remained within normal range at each visit.

A diagnosis of preeclampsia with severe features was made. She was managed with intramuscular injection of magnesium sulphate and oral nifedipine. After two days of admission, her blood pressure was maintained at around 140/ 100 mm of Hg. She was put on a trial for normal vaginal delivery, but ultimately caesarean section was conducted for non-progression of labor.

During intraoperative period, she became drowsy. After surgery, there was a subsequent fall in the level of her consciousness, and she developed abnormal body movements. The next day she was transferred to the tertiary care hospital, with an oropharyngeal airway inserted for maintaining airway patency. At the time of presentation, her Glasgow Coma Scale (GCS) was 8 with E4V1M3, was gasping, with a respiratory rate of 32 breaths per min, arterial oxygen saturation of 98% with oxygen via face mask. Her blood pressure was 200/ 100 mmHg, and pulse was 110 beats per min, regular. On chest auscultation, bilateral crepitations were present. Abdomen was soft, the uterus was well retracted to 22-24 weeks size, cervix was healthy. Active bleeding was absent. Pupils were dilated and non-reactive to light. Abnormal blood investigation included platelet: 35,000 cell/ cumm, total/ conjugated bilirubin: 6.4/ 1.7 mg/ dl, SGPT/ SGOT/ ALP/ LDH: 425/1039/50/1676 U/L, Urine RME: Protein ++, RBC plenty. Non contrast computerized tomography (CT) head showed left fronto-temporo-parietal intraparenchymal hematoma with intraventricular extension with mass effect and subarachnoid haemorrhage (**Fig. 1**).

A diagnosis of severe preeclampsia with HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome with intracranial hemorrhage was made. She was transferred to the intensive care unit (ICU), intubated, and kept on ventilatory support. Her condition further deteriorated with a fall in her GCS to 2T/ 10, absent gag reflex, and a drop in blood pressure. Intravenous noradrenaline infusion was started; adrenaline and vasopressin were added subsequently. Repeat CT head showed diffuse cerebral edema. Surgical intervention was not done due to poor prognosis. Over the next few days, she developed renal failure with severe metabolic acidosis and ultimately died of cardiac arrest due

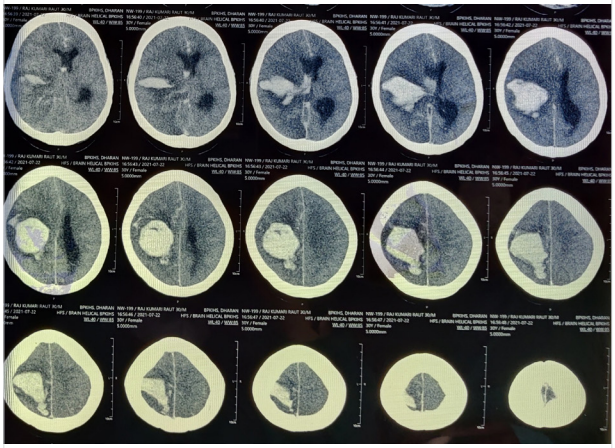

Figure 1: CT scan of head showing left fronto-temporo-parietal intraparenchymal hematoma with intraventricular extension with mass effect and subarachnoid haemorrhage.

to resistant bradycardia and hypotension.

DISCUSSION

Preeclampsia occurs in 3-12 % of pregnancies, and is one of the leading causes of maternal and perinatal morbidity as well as mortality [3, 4]. The risk of preeclampsia in first pregnancy is around 4%, and in a multiparous woman without a prior history of preeclampsia as in our case is 1% [5]. The pathophysiology of preeclampsia centres around maternal immune response to paternal antigen expressed in the placenta, resulting in defective trophoblast invasion and placental dysfunction. Oxidative stress of placenta then releases proinflammatory cytokines, exosomes, anti-angiogenic agents, and foetal DNA into the maternal circulation, initiating a global systemic inflammatory response [1].

The late onset preeclampsia is more common compared to the early onset type. However, it is the early onset type that is associated with more adverse maternal and fetal outcomes [2, 3]. A 10-fold risk of perinatal and maternal death with early onset type, and 2-fold risk of perinatal and 3-fold risk of maternal death with late onset type is reported when compared to normal pregnancy [2]. Prolonged exposure of the mother and foetus to the oxidative stress of defective placentation and greater unfolded protein response with the early onset type could be the reasons [1]. Late onset preeclampsia could be due to discrepancy between normal maternal perfusion and increased metabolic demand of the placenta and foetus [1]. This explains why preeclampsia can occur for the first time in a multigravida.

Unlike more adversities linked with early onset preeclampsia, late onset type can have an equally alarming outcomes, as it develops abruptly and progress rapidly to a life threatening situation, as in our case. Moreover, the early onset type mostly presents as a mild disorder that progress slowly over time, providing adequate time for monitoring women for any possible complications, in their existing and subsequent pregnancies. However, monitoring may not be adequate in the late onset type as it develops unexpectedly, especially if occurs for the first time in a multigravida. Therefore, it is crucial to consider women with preeclampsia to be at high risk for adverse pregnancy outcomes, irrespective of its type and must be monitored cautiously and continuously throughout their pregnancy.

Complications associated with preeclampsia like HELLP syndrome and intracranial hemorrhage (ICH) increase during the third trimester of pregnancy. Incidence of HELLP syndrome in a women with preeclampsia ranges from 4-12% [6], with maternal and perinatal mortality reaching up to 24% and 60% respectively [7]. Hypertension associated with pregnancy significantly increases the risk of ICH and stroke suggesting management of hypertension to be a simple, yet crucial step in preventing many untoward events [8, 9]. Other contributors of preeclampsia for ICH include endothelial dysfunction, increased cerebral perfusion pressure, and disturbances of cerebral blood flow autoregulation [8]. Concomitant presence of HELLP syndrome with preeclampsia further increases haemorrhagic risk due to thrombocytopenia and coagulopathy. Preeclampsia and its complications may even develop in the postpartum period, necessitating monitoring and follow up of women even after the delivery of baby.

The frequency and the quality of ANC between pregnant woman and health care provider plays a crucial role in the detection of both active and impending cases of preeclampsia. Overall, inadequate number of ANC shows a 12-fold risk of poor maternal outcome, 53-fold risk of poor foetal outcome and significantly higher risk of neonatal mortality compared to adequate number of visits [10]. World Health Organization recommends at least 8 ANC between pregnant woman and their care provider, first within 12 weeks of gestation, with subsequent at 20, 26, 30, 34, 36, 38 and 40 weeks of gestation [11]. Increase in the frequency of ANC during third trimester would help in early identification of women with late onset preeclampsia and its adversities. Our patient had a regular ANC, and her blood pressure had remained within the normal range at each visit, done before the 37 week.

Identification of women at risk of preeclampsia may help in reducing the associated morbidity and mortality. Women with chronic hypertension, pre-pregnancy BMI > 30, and previous preeclampsia are some of the known risks for development of preeclampsia [1]. Proper attention to women with these risks during ANC may help in predicting and detecting preeclampsia early. The two risk factors in our case were high BMI and a longer interpregnancy interval. The interpregnancy interval of 10 years in a multigravida is reported to increase the risk of preeclampsia to that of a nulliparous woman [12]. The interpregnancy interval was 12 years in our case. Predictive efficacy of preeclampsia can be further increased by combining the risk factors with serum markers like inhibin A, activin A, placental growth

factor and the uterine artery doppler pulsatility index [13].

In addition to identifying women with preeclampsia, their proper follow up and management is equally important for a better pregnancy outcome. Women with severe preeclampsia must be managed in a facility with multidisciplinary expertise, so that all associated adversities can be managed timely and effectively. Our case had some features of severe preeclampsia like high blood pressure, severe headache, vomiting, and abdominal pain. However, her blood investigations including liver function tests were normal. This may be the reason why she was not referred early to a tertiary care centre. The patient might have developed ICH during intraoperative period, as level of her consciousness dropped during caesarean section and progressively decreased in the postoperative period. Prompt imaging with rapid diagnosis and management immediately after delivery, would have possibly improved the outcome in this case.

Overall, development of preeclampsia irrespective of its type, is a major risk for maternal and perinatal morbidity and mortality. Early identification of women at risk of developing preeclampsia, their regular and quality follow up at each ANC and early referral of preeclamptic women with features of severity to a multidisciplinary facility may improve their pregnancy outcomes.

CONCLUSION

Preeclampsia in a multigravida occurs unexpectedly and may progress rapidly to life threatening situations. Identification of women at risk of preeclampsia, and their meticulous follow up during the third trimester is crucial for detecting the late onset type. Preeclamptic women with features of severity, must be referred early to a facility with multidisciplinary expertise to improve their pregnancy outcome.

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