

Sudden onset severe preeclampsia during caesarean section, unmasked by the bolus dose of ephedrine

Sir,

A 27-year-old parturient with second gravida presented for elective caesarean section. Throughout the pregnancy, she had regular antenatal follow-up with normal blood pressure (BP) measurements. Her investigations were normal and urine albumin was negative. The day prior to the surgery, four readings of BP measured at 4 hours interval were normal (varying from 100/60 mmHg to 120/80 mmHg) and urine albumin was negative. In the operation theatre, she was monitored with electrocardiograph (ECG), Pulse oximeter and Non-Invasive Blood Pressure (NIBP), every 3 minutes. Her baseline BP in the operation theatre was 120/80 mmHg and heart rate (HR) was about 80/min. Spinal anaesthesia was given with 2.2 ml of hyperbaric bupivacaine 0.5% and surgery commenced after achieving block level of T4. She was preloaded with 500 ml of intravenous normal saline. About 5 minutes into surgery, the patient developed hypotension with BP of 90/56 mmHg and HR of 60/min which was treated with Intravenous (IV) ephedrine 15 mg. Immediately the BP increased to 180/110 mmHg and the HR to 120/min. The tachycardia and hypertension were presumed to be due to increased response to the ephedrine. A live, active baby was delivered at 9th minute after the spinal block. Soon, the patient complained of severe headache, vomiting and visual blurring during the caesarean section which lasted for about 45 minutes. Throughout caesarean, her BP persisted to be high in the range of 150/90-170/106 mmHg and HR was around 110/min. The persistence of high BP beyond the usual duration of ephedrine raised suspicion of intracranial haemorrhage, other neurological diseases or preeclampsia. She was transferred to intensive care unit (ICU) and IV metoprolol 1 mg was given for control of HR and BP. About 10 minutes later, the patient developed tonic clonic convulsion, which was immediately aborted with IV diazepam 5 mg and Phenytoin. The patient was also started on IV MgSO₄ infusion. Patient's complaints of headache and visual blurring persisted for next 6 hours and subsided thereafter. Magnetic resonance imaging (MRI) of brain

performed next day showed minimal cerebral oedema with no evidence of any intracranial haemorrhage. The urine albumin was 1 gm/L confirming the diagnosis of preeclampsia. Her platelet count, renal and liver function tests were normal. Two days later, her BP was stabilised at 140/90 mmHg, patient was started on oral anti hypertensive nifedipine. Mother and baby were discharged from hospital uneventfully.

Preeclampsia is the commonest cause of hypertension and neurological symptoms like visual blurring and convulsion during pregnancy. But in our patient, ephedrine bolus given for the treatment of hypotension led to the hypertensive episode. Ephedrine is known to have slow onset and long duration of action making it difficult to titrate for spinal induced hypotension.^[1] Many studies have documented tendency of blood pressure to overshoot following the use of ephedrine.^[2] The hypertensive response lasted more than usual duration of ephedrine, raising the suspicion of other neurological diseases like: Cerebrovascular haemorrhage, ruptured aneurysm and cerebral venous thrombosis etc., There are previously reported cases of neurological diseases like ruptured aneurysm, subdural haemorrhage precipitated by spinal anaesthesia presenting as headache and photophobia during the surgery.^[3] Persistent high BP and presence of albumin in urine confirmed the diagnosis of preeclampsia.

Previous studies have shown that the vasoconstrictive potential of pressor substances (e.g., angiotensin II) is magnified in preeclampsia.^[4] Hence, ephedrine requirements are reduced during spinal anaesthesia for caesarean section in preeclampsia.^[5] The standard dose of ephedrine for treatment of hypotension in normal parturient is 10-30 mg.^[2] Our patient was not a known preeclamptic, hence higher normal dose of ephedrine was given. It is possible that the patient had tendency for preeclampsia and the bolus dose ephedrine unmasked it and brought it to clinical picture.

To diagnose preeclampsia, hypertension is defined as BP of more than 140/90 mmHg on at least two occasions, with measurements being done at least 4 hours apart.^[6] Our patient's BP was monitored regularly and abrupt increase in the BP with less than 4 hour interval added to the diagnostic dilemma.

Therefore, in parturient developing severe hypertension, headache and visual disturbances, preeclampsia should be thought as a possibility along

with the other intracranial pathology, even in absence of prior history of preeclampsia.

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