Case Report

Spontaneous subdural hematoma in a multigravida with chronic kidney disease and superadded pre-eclampsia detected by MRI: a case report

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ABSTRACT

Chronic kidney disease and pre-eclampsia are both known individual culprits for significant morbidity and mortality of mother and child worldwide. Non traumatic spontaneous subdural hematoma in pregnancy is a rare scenario, however stage 5 chronic kidney disease with superadded pre-eclampsia can be contributory factor for spontaneous extra axial bleed. Our patient was 25-year-old with history of end stage renal disease and chronic hypertension admitted with superadded pre-eclampsia. She had 1 living issue and 2 stillbirths. There was sudden deterioration of mental status as well as motor performance, the patient turned stuporous and was not following verbal commands. Patient was sent for MRI evaluation of brain and diagnosed as right sided subdural hematoma with significant midline shift towards the contralateral side. No intraparenchymal haemorrhage was noted.

Keywords: Chronic kidney disease, Magnetic resonance imaging, Pre-eclampsia, Subdural hematoma

INTRODUCTION

Worldwide, 10% of all pregnancies are complicated by hypertension, with pre-eclampsia and eclampsia being the major causes of maternal as well as prenatal morbidity and mortality.1 It is estimated that pregnancy induced hypertension (PIH), one of the hypertensive disorders of pregnancy, affects about 5-8% of all pregnant women worldwide.2 Pregnancy induced hypertension (PIH) is defined as BP ≥ 140/90mmHg, taken after a period of rest on two occasions or ≥160/110mmHg on one occasion after 20 weeks of gestation in a previously normotensive woman.

Pre-eclampsia affects 5-7% of all pregnancies. It is broadly defined by hypertension and proteinuria.3 Eclampsia includes pre-eclampsia with the presence of convulsions not attributable to other neurologic disease. Women with chronic kidney disease are less capable of making renal adaptations needed for a healthy pregnancy. Their inability to boost renal hormones often leads to normochromic normocytic anemia, reduced expansion of plasma volume, and vitamin D deficiency.4 Women with moderate to severe disease (stages 3-5) are at highest risk of complications during pregnancy and of an accelerated decline in renal function.4

A subdural hematoma (SDH) is a collection of blood below the inner layer of the dura but external to the brain and arachnoid membrane.5 The MR characteristics of acute and subacute subdural hematoma are similar to those of intraparenchymal haemorrhage, whereas chronic subdural hematoma differ from parenchymal hematoma in several ways.6 As opposed to the typical parenchymal hematoma, which is markedly hyperintense on short TR/TE images, a chronic subdural hematoma will usually be slightly hypointense to isointense relative to gray
matter on short TR/TE images. This loss of the T1 shortening effect appears to result from a decrease in the concentration of free methemoglobin by either dilution, absorption, and/or degradation. Re-hemorrhage is a frequent phenomenon with subdural hemorrhage and MR offers excellent visualization of this process. Subdural hematoma are prone to re-hemorrhage even without clinically evident trauma and there is visualization of layering on MRI scan.

CASE REPORT

A 25-year pregnant female patient (G4P3L1A0) with period of gestation 29 weeks and 3 days (by ultrasonography) presented with decreased fetal movement and multiple bouts of vomiting for the last 2 days. She is a known case of stage 5 chronic kidney disease receiving dialysis. There was history of two stillbirths and one live birth by vaginal delivery. She neither paid any antenatal visit nor took any prophylactic medications. She was chronic hypertensive with a baseline blood pressure of 156/100 mmHg at the time of admission. Her blood parameters were, Hb -10 gm%, TLC - 14000/cumm, platelet - 3.5 lakh/cumm, Renal function test: urea 134 mg/dl, creatinine 4.2 mg/dl, 24 hour urinary protein 0.5 gm. Liver function test: serum total bilirubin - 0.6 mg/dl, SGPT - 238 U/L, SGOT - 542 U/L, PT - 13.5 sec, INR - 1.3, serum albumin 5.3 gm/dl, globulin 2.6 gm/dl. Triglyceride - 333 gm/dl, serum amylase - 553 U/L, serum lipase - 677 U/L, Sodium - 133 mmol/L, potassium 3.9 mmol/L. Serum LDH - 594 U/L. IgG rubella antibody positive, TORCH antibody negative. She was nonreactive for HIV, HCV, HBsAg; Anti cardiolipin antibody negative. Clinicians diagnosed superadded pre-eclampsia in this patient, however they could not conclude the cause of recurrent pregnancy loss.

As the patient presented with decreased fetal movements, she was sent for ultrasound to assess fetal wellbeing. The biophysical score was 6/8 and chorioamnionitis was diagnosed. There was sudden deterioration of neurological status and the patient became stuporous having a GCS of 5/15 with left sided hemiparesis. The clinician sent her for urgent MRI evaluation of brain. MRI revealed mixed intensity subdural hematoma along right cerebral convexity with significant midline shift towards contralateral side (Figure 1).

CT was not advised with the concern of avoiding hazardous effect of radiation to the fetus. The patient was taken to neurosurgery OT on an emergency basis and drainage of subdural hematoma was performed. After that the patient was shifted to intensive care unit and her neurological status improved gradually.

DISCUSSION

Chronic kidney disease and pre-eclampsia are known independent risk factors for poor outcome in pregnancy affecting both mother and fetus. Spontaneous epidural hemorrhage has been described in cases of chronic renal failure (CRF). During hemodialysis, patients with CRF rarely develop spinal or cranial epidural hematomas. Such hemorrhages have been attributed to intracranial pressure fluctuations during hemodialysis, heparin administration, uremic-platelet syndrome, or hypertension. Although the exact pathophysiology is not understood, this rare consequence of hemodialysis has been well documented in the literature.

Pregnancy induced hypertension or pre-eclampsia is a well-known causative factor for intracerebral hemorrhage. However not many spontaneous extra-
Axial bleeds in pre-eclampsia have been reported in literature.

Present case was a multigravida young lady with stage 5 chronic kidney disease, with grossly deranged renal function test. The presence of significant proteinuria, vomiting, raised liver enzymes, borderline INR levels and overshoot of blood pressure up to 220/110mmHg denoted superadded pre-eclampsia. The platelet level was absolutely normal, so thrombocytopenia induced bleed was ruled out. No episode of seizure was documented. The patient did not undergo dialysis on the day she went into stupor. Sudden neurological deterioration of the patient drew the attention for urgent cross-sectional imaging of brain. MRI revealed no evidence of any intraparenchymal hemorrhage. There was a large subdural hematoma along right cerebral convexity which was causing significant midline to shift towards the contralateral side. The cause of spontaneous subdural hematoma is not understood, but it is evident that the cause of neurological deterioration is subdural hematoma and not intraparenchymal hemorrhage or metabolic encephalopathy.

CONCLUSION

Chronic kidney disease with superadded pre-eclampsia are contributory factors for significant morbidity in pregnant patient. Though intracerebral hemorrhage is known causative factor for sudden neurological catastrophe, spontaneous subdural hematoma is a very rare manifestation however treatable.

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