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Intracerebral hemorrhage (ICH)-a rare complication of Pre-eclampsia: a case report

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ABSTRACT

Introduction: Intracerebral hemorrhage (ICH) is a rare complication in pre-eclampsia. It is potentially devastating event to occur in pregnancy and associated with significant maternal morbidity and mortality. **Case study:** A 28-year-old primigravida with pre-eclampsia at 30 weeks 5 days period of gestation, presented with altered sensorium and emergency caesarean revealed massive concealed placental abruption with intrauterine fetal death. She later presented with left sided hemiparesis. Non-contrast computed tomography confirmed intracerebral frontal hemorrhage. Other radiodiagnosis tests done ruled out rupture arterial aneurysms, arteriovenous malformations and cerebral venous sinus thrombosis. She underwent decompressive craniectomy and evacuation of blood clot for intracerebral hemorrhage. Her Modified Rankin score was 2 at the time of her discharge. **Conclusion:** The complication of intracerebral hemorrhage with preeclampsia is rare. An early diagnosis, timely referral and interventions are associated with decreased maternal and perinatal morbidity and mortality.

Keywords: Cerebral hemorrhage; Decompressive craniectomy; Pre-eclampsia.

INTRODUCTION

Intracerebral hemorrhage (ICH) is a type of cerebrovascular accidents, which occur within the brain tissue[1]. ICH is a rare complication in preeclampsia and the incidence is about 3.5 to 26 per 100000 deliveries[1–3]. It is associated with high maternal mortality and estimated to be around 9-38%[1,4,5]. Some among survivors had permanent disability[1,6,7]. Early and prompt diagnosis of intracerebral hemorrhage in pregnancy and timely interventions are associated with decreased maternal as well as perinatal morbidity and mortality.

Intracerebral hemorrhage is known to occur with gestational hypertension, preeclampsia,eclampsia,rupture of arteriovenous malformations and cerebral aneurysms, or from cerebral venous sinus thrombosis[8,9].

This case report is regarding a rare case of intracerebral hemorrhage in a pregnant woman with preeclampsia and placental abruption. The main aim of this report is to create awareness among clinicians managing pregnant women with gestational hypertension or preeclampsia about the occurrence of this complication. Prompt diagnosis and early management will help to prevent severe maternal morbidity and mortality.

Case study

A 28-year-old primigravida of 30 weeks 5 days of gestation was brought to emergency department (ED) in the state of altered sensorium. She had history of severe abdominal pain with few episodes of vomiting, headache, and absent fetal movement a day prior to arrival at ED. There was history of becoming drowsy over the period of 4 to 5 hours. There was no history any seizures, trauma, fall, or development of weakness of upper and lower limbs prior to arrival to ED. There was no history of bleeding per vagina and no significant past medical history. She had regular antenatal clinic visits and was not diagnosed with preeclampsia during her last visit, which was done one month prior to the day of arrival in ED.

On examination, patient was clinically pale with tachycardia, Blood pressure (BP): 179/105mmg Hg, respiratory rate (RR): 22/min, Oxygen saturation (SpO2): 98% at room air. Both pupils were equally reactive with a Glasgow coma scale (GCS) score of E3V2M4 (9/15). There was no hypotonia of both lower and upper limbs.

Obstetric examination revealed the fundal height of 32 weeks size corresponding with the period of gestation but the

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uterus was tense with broad-like rigidity. On assessing the fetal well being by use of fetal Doppler hand set, the fetal heart sound could not be detected and immediately bed side ultrasound was done, where a large retroplacental clots was detected suspicious of concealed type of placental abruption with absent fetal heart beat. The laboratory investigations done were as follows: urine albumin dipstick test was 3 +, hemoglobin was 8.7 g/dl, platelet counts were 102x103/ul, prothrombin time (PT) 12.6 sec, INR was 1.12. Serum urea was 57mg/dl, serum creatinine was 2.5 mg/dl suggestive of acute kidney injury due to preeclampsia. Her liver function test was unremarkable. Patient was diagnosed to be a case of severe preeclampsia with concealed abruption and intrauterine fetal death. She was suspected to have active retroplacental bleeding.

The patient was given 20 mg of bolus intravenous labetalol with repeat bolus dose after 15 minutes till her blood pressure was 140/90 mm of Hg. She had concealed type of placental abruptionwith suspected active retroplacental bleed and her modified bishopscore unfavorable for vaginal delivery, she was taken up for emergency lower segment cesarean section. During the lower segment caesarean section, a large retroplacental clots about 1.5 liters of clotted blood with complete separation of placenta and a coulvelaire uterus was noted (Figure 1). The baby was unfortunately a fresh still birth.

The mother during the postoperative period was managed in the intensive care unit (ICU). She developed acute kidney failure with increasing serum creatinine level (4.2mg/dl), she underwent haemodialysis. The other laboratory investigation done on that day were as follows; Hemoglobin was 7.1 g/dl, Platelet counts was 71x103/ul, Prothrombin time (PT) 16.2 sec, INR was 1.47, Total Bilirubin was 1.9 mg/dl, AST was 50 IU/L, and ALT was 15IU/L. Packed red blood cells and platelets transfusion were given. On 3rd post operative day, it was noticed that she had developed right sided lower and upper limb weakness which was progressive and this finding was missed during the initial assessment as well as during the first few days in the ICU by the clinicians. An urgent non contrast computed tomography (NCCT) was done, which showed a left frontal intracerebral hemorrhage with a mass effect and cerebral edema (Figure 2).

Magnetic resonance imaging also showed a large left frontal hemorrhage with midline shift (Figure 3).

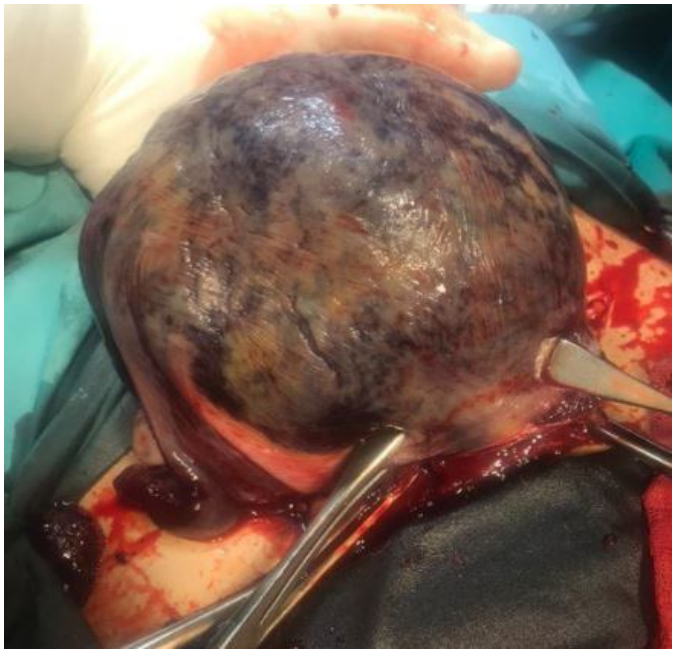

Figure 1. Coulevaire uterus following massive concealed placental abruption

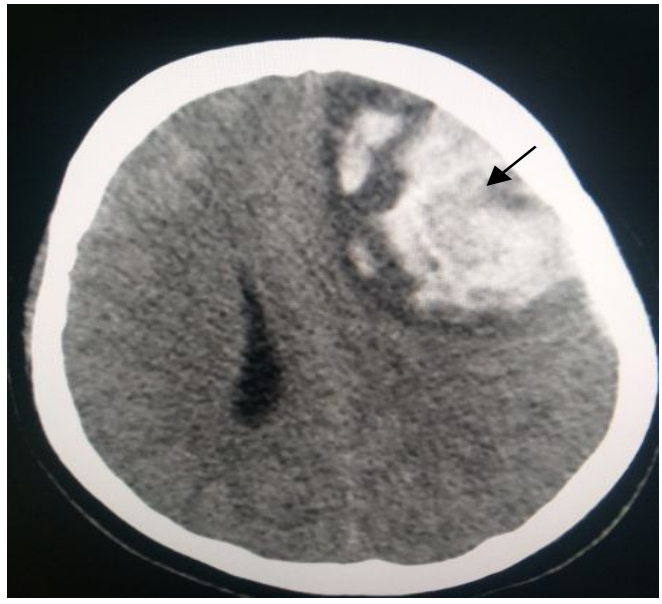

Figure 2. Non contrast computed tomography (NCCT) in axial view showing left frontal hemorrhage (arrow pointed) with mild extension into the frontal horn of left lateral ventricle with midline shift

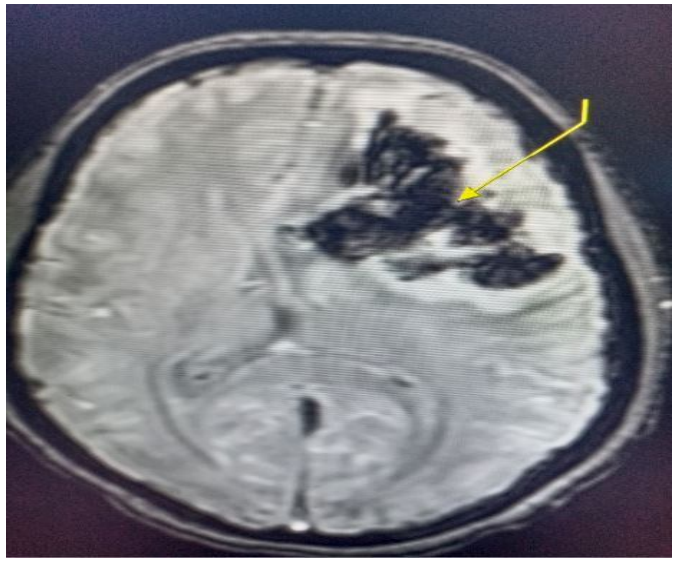

Figure 3. Magnetic resonance imaging (MRI) in axial view showing a left frontal bleed with mass effect and midline shift (arrow pointed)

To rule out other causes of intracerebral hemorrhage due to rupture of arteriovenous malformation, or cerebral aneurysms, or hemorrhage due to cerebral venous sinus thrombosis, magnetic resonance angiography (MRA) and magnetic resonance venography (MRV) were done. MRV and MRA showed no venous sinus thrombosis or occlusion (Figure 4) and no arterial aneurysms or rupture from arterial aneurysms (Figure 5).

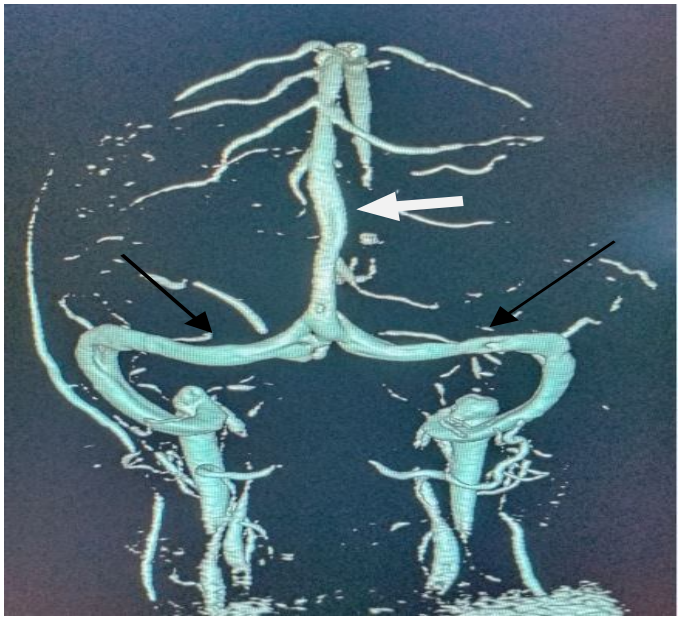

Figure 4. Magnetic Resonance Venography (MRV) revealed normal right and left sigmoid sinuses (black arrow) and normal sagittal sinus (white arrow) with no evidence of thrombus or occlusion

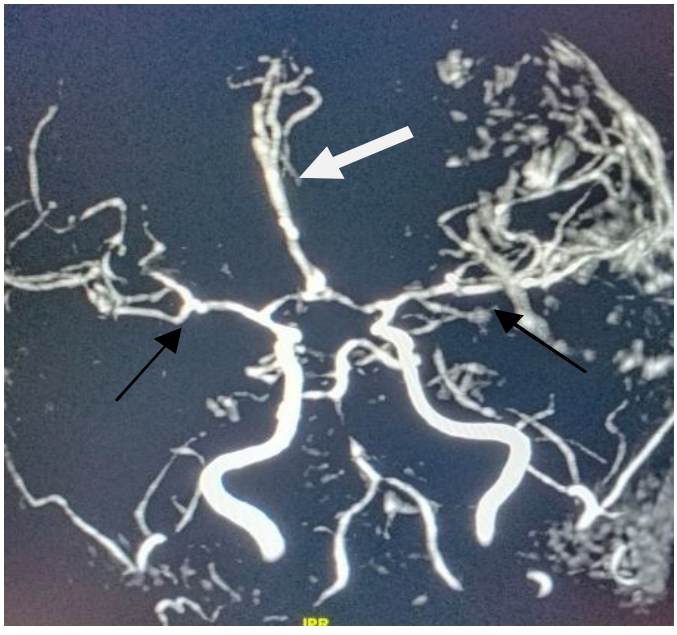

Figure 5. Magnetic resonance arteriogram (MRA) showing normal anterior cerebral artery (white arrow) and both normal middle cerebral arteries (black arrow)

Immediate therapeutic transfusion was conducted and was followed by urgent decompressive craniectomy with clot removal under general anesthesia by the neurosurgical team. In the immediate post operative period, the patient was electively ventilated for 48 hours and extubated. For blood pressure control, labetalol continuous infusion rate at 0.5 to 1.5mg/min was continued until oral feeds were resumed, and after that she was put on tablet losartan and amlodipine. Physiotherapy was done during her hospital stay. The Patient was discharged on 18th post operative day on tablet losartan 25 mg twice daily and tablet levetiracetam 500mg twice daily to prevent seizures. Her Modified Rankin scale score was 2 at the time of discharged. Review at clinic after 2 weeks of discharge, a repeat CT scan was done which showed improvement in the midline shift (Figure 6(a), 6(b))

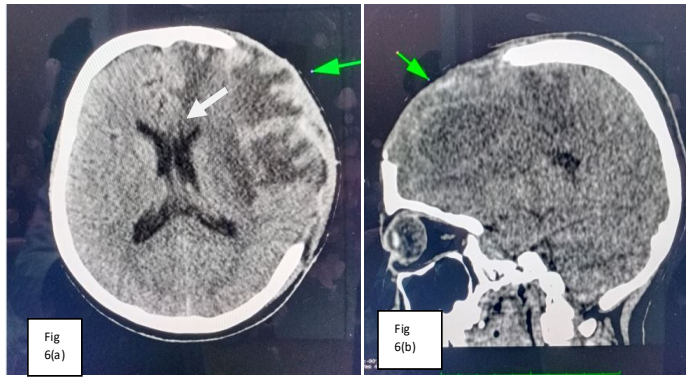

Figure 6(a). Axial view shows an NCCT post-left craniectomy with a skull bone defect (green arrow) and decreased in Midline shift(white arrow). Figure 6(b). Coronal view showing the post craniectomy skull bone defect (green arrow)

Her GCS (E4 V5M6) was 15/15, Motor function; grade IV (right upper and lower limbs), grade V (left upper and lower limbs) at 2 weeks and underwent physiotherapy. She was also counseled about family planning and her next pregnancy. She underwent cranioplasty after 4 months.

DISCUSSION

Fortunately intracerebral hemorrhage (ICH) is a rare complication to occur during pregnancy and delivery. It causes high maternal mortality and disability among survivors[1–3,5,6]. Many factors are responsible and associated with intracerebral hemorrhage. In a study by Bateman et al, gestational hypertension, preeclampsia/ eclampsia and low platelet count were significant independent risk factors for ICH during pregnancy, and accounted for 30.5% of ICH[10]. In other studies, Gestational hypertension and preeclampsia were also important risk factors for ICH in pregnancy, and reported to be present in 14–50% of the cases[4,7,11]. The risk is more if preeclampsia have complications like eclampsia, HELLP syndrome, or abruption with coagulopathy[4,12]. In our case, she had preeclampsia with acute renal failure, with massive abruption and low platelet count, which could

have cause intracerebral hemorrhage. Other causes of ICH in pregnant women are from rupture of vascular malformations like arterio-venous malformations(AVMs), or aneurysms, or from hemorrhage from cerebral venous sinusthrombosis[12]. These were ruled out in our patient by magnetic resonance angiography (MRA) and magnetic resonance venography (MRV).

The pathophysiology of intracerebral hemorrhage associated with preeclampsia and it complications are due to increase in blood pressure which leads to disturbed cerebral auto regulation, cerebral hyper perfusion, blood brain barrier(BBB) disruption leading to formation of cerebral edema and damage to the vascular endothelium (microangiopathy)[4,7]. There is an increased risk of intracerebral hemorrhage with preeclampsia during pregnancy, mostly in third trimester or post-partum period among patients who have the risks[4]. A study done by Bateman et al found that the risk was high in the postpartum period, where other study found that risk was greatest during parturition and in the puerperium[2,10]. Yoshimatsu et al found that ICH developed in twice as many in primiparous women than multiparous women and mostly occurred in the antepartum period, especially in the third trimester[1]. Our case also was a primigravida, with complication of preeclampsia and at third trimester. The lack of specificity of clinical signs and symptoms associated with ICH other than the focal neurologic deficits often fail to alert the physician to this condition, leading to delay diagnosis[1,2,13]. In our case the delayed in the diagnosis as the patient didn't have neurological deficits in the beginning and could diagnosis only later, when clinician notice the weakness of right lower and upper limbs.

The definitive diagnosis is by computed tomography (CT), and by Magnetic resonance imaging (MRI). CT without contrast is the most commonly used initial study to diagnose ICH due to its wide availability and high sensitivity and specificity[13]. Decompression craniectomy or craniotomy with surgical evacuation of the clot is indicated if there is a declining conscious state or worsening neurological deficit[2,4,13]. Our patient had worsening mass effect with midline shift and neurological deficit. Non-operative approach is appropriate in cases of devastating hemorrhage that is non recoverable, although surgery might be considered in an effort to prolong the life of the mother in the interests of the fetus[2]. However, due to the low incidence rate of ICH, neurosurgeons and obstetricians often lack sufficient experience in diagnosing and treating such patients and often fail to make prompt judgments[5].

Preeclampsia, abruption, coagulopathy and moderately or severely disturbed consciousness at disease onset were significantly associated with a maternal mortality and those who survive have disability modified Rankin Scale ≥3[1,4,5,7,12]. Onset to diagnosis time(O–D time)>3 hour were significantly associated with maternal mortality. Our patient despite having preeclampsia with complications of acute kidney failure, massive abruption, low platelet count, and the onset to diagnosis time (O-D) more 3 hours fortunately made a full recovery with GCS 15/15 and with the modified Rank in score of 2 at 3 week review at clinic and she had undergone cranioplasty at 4 months.

CONCLUSION

An intracerebral hemorrhage during pregnancy is associated with gestational hypertension, preeclampsia, eclampsia and coagulopathies. It occurs more commonly towards later part of pregnancy. Early referral of the suspected cases to center with Computed tomography facilities and prompt diagnosis of intracerebral hemorrhage with timely interventions are associated with decreased maternal mortality and prevents development of severe disability among survivors.

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