Case Report

Antepartum cerebral angiopathy presenting as middle cerebral artery aneurysm rupture – A rare case report from India

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ABSTRACT

The incidence of middle cerebral artery aneurysm rupture in pregnancy is rare. Rupture of an intracranial aneurysm is the most common cause of subarachnoid hemorrhage in pregnancy and during puerperium. Intracranial aneurysm rupture during pregnancy is disastrous for the mother as well as the fetus. We report a case of a dissecting left middle cerebral artery aneurysm in a 25-year-old woman, G2P1L1, at 18 weeks of gestation, who presented with a 1-day history of giddiness, slurring of speech, and facial deviation to the left.

Key words: Endovascular coiling, Intracranial aneurysm, Middle cerebral artery aneurysm, Subarachnoid haemorrhage

The incidence of middle cerebral artery aneurysm rupture in pregnancy is rare. On the contrary, intracranial aneurysm rupture still remains the most common cause of subarachnoid hemorrhage (SAH) during pregnancy and puerperium [1]. It occurs more frequently in primiparous women and in the last trimester of pregnancy. Maternal mortality due to aneurysmal rupture is as high as 5–12% [1]. Intracranial aneurysm rupture during pregnancy is disastrous for the mother as well as the fetus.

Herein, we report a case of a dissecting left middle cerebral artery aneurysm in a 25-year-old woman, G2P1L1, at 18 weeks of gestation, who presented with a 1-day history of giddiness, slurring of speech, and facial deviation to the left. Appropriate diagnosis and treatment of SAH are important in pregnancy as the probability of recurrent hemorrhage is 33–50% and maternal mortality rates are about 50–68% in untreated cases.

CASE REPORT

A 25-year-old female, G2P1L1, at 18 weeks of gestation presented with a 1-day history of giddiness, slurring of speech, and facial deviation to the left. When she woke up in the morning, she developed giddiness and headache of 1-day duration followed by two episodes of vomiting. She was not able to talk since then. The patient's medical history was unremarkable and there was no significant family history. Initially, she was taken to a local hospital, where she received intravenous fluids, thiamine, and analgesics.

On examination, her vital signs were within normal limits. She was conscious and oriented. Pupils were equal and reactive to light. There was no motor weakness. Plantar showed flexor response. She had slurring of speech and right facial palsy.

Laboratory studies (reference range in parentheses) revealed platelet count: 316,000/μL (150,000–450,000/μL), white blood cell count: 11200/μL (4000–11000 K/μL), hemoglobin: 10.1 g/dL (12–15 g/dL), mean corpuscular volume: 83 fL (83–101 fL), red blood cell distribution width – standard deviation: 13.6% (11.6–14%), erythrocyte sedimentation rate: 16 mm/h. (<20 mm/h), and C-reactive protein: 12.9 mg/L (<5 mg/L). Urinalysis revealed pale yellow urine, no occult blood, 1–2 white blood cells per high power field, and no bacteria. Peripheral blood smear showed a microcytic hypochromic blood picture.

Magnetic resonance imaging (MRI) of the brain showed acute intraparenchymal hematoma in the left frontoparietal operculum and insular cortex, acute SAH in the left Sylvian cistern, suprasellar cistern, and along with the sulcal spaces of the left cerebral hemisphere (Fig. 1). MR non-contrast angiogram revealed suspicious small out-pouching from the M3 segment of the left middle cerebral artery suspicious of an aneurysm (Fig. 2).

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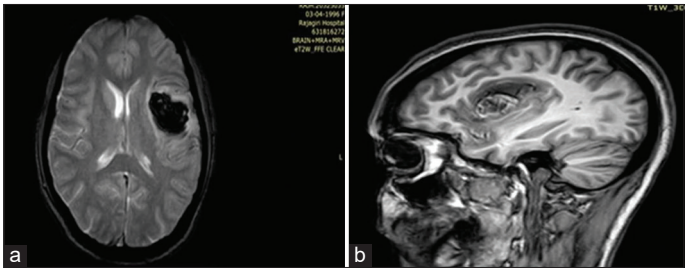

Figure 1: Magnetic resonance imaging of the brain. (a) T2-weighted Fast Field Echo showing acute intraparenchymal hematoma showing blooming in the left frontoparietal operculum and insular cortex; (b) T1-3D showing bleed – iso to hyperintense

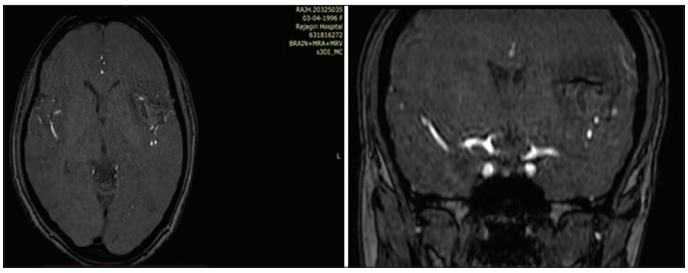

Figure 2: Magnetic resonance non-contrast angiogram showing small aneurysmal outpouching from the M3 segment of the left middle cerebral artery projecting anteriorly - partially thrombosed/ dissection, surrounded by the bleed

Diagnostic cerebral angiography confirmed a dissecting/mycotic left M3 middle cerebral artery aneurysm with two tiny blebs from the right cavernous and clinoidal internal carotid artery (Fig. 3a). Endovascular coiling with parent artery occlusion was done for the left M3 middle cerebral artery aneurysm (Fig. 3b). The procedure was uneventful.

The patient had multiple aneurysms and the chance of worsening of angiopathy is high in pregnancy. Gynecology opinion was taken and medical termination of pregnancy was done.

Screening MRI of the brain [post-coiling] did not show any evidence of a significant interval increase in the size of the previously documented acute intraparenchymal hematoma in the left frontoparietal region (Fig. 4). Acute SAH involving the left Sylvian cistern, suprasellar cistern, and along with the sulcal spaces of the left cerebral hemisphere was persisting with no evidence of interval increase. Subsequent non-contrast computed tomography of the brain showed acute to subacute left frontotemporal intraparenchymal hematoma with mild perihematoma edema (Fig. 5). Metal artifacts from the coil are noted in the superior aspect. There was mild effacement of the left lateral ventricle. There was no significant midline shift. Follow-up MRI showed hemosiderin deposition in place of the left frontal hematoma [T2-weighted Fast Field Echo] and Gliosis/ Encephalomalacia in place of the left frontal hematoma [T2 FLAIR] (Fig. 6).

She was started on anti-seizure medications. Nimodipine was initiated to prevent vasospasm. Connective tissue disease and vasculitis workup were negative. The patient improved clinically and was discharged.

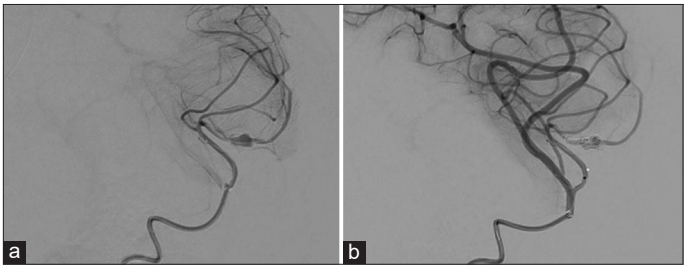

Figure 3: (a) Digital subtraction angiography (DSA) – Lateral Projection showing aneurysm; (b) DSA – Lateral Projection – Post coiling

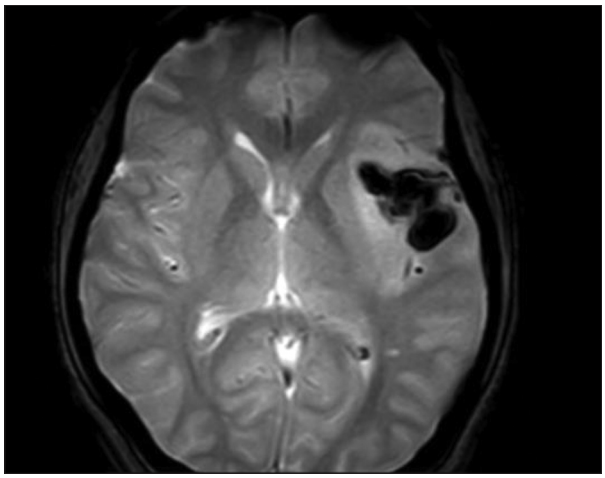

Figure 4: Magnetic resonance imaging of brain T2-weighted Fast Field Echo showing a reduction in the size of the area of blooming (size of the bleed) and a mild interval increase in adjacent neuroparenchymal edema

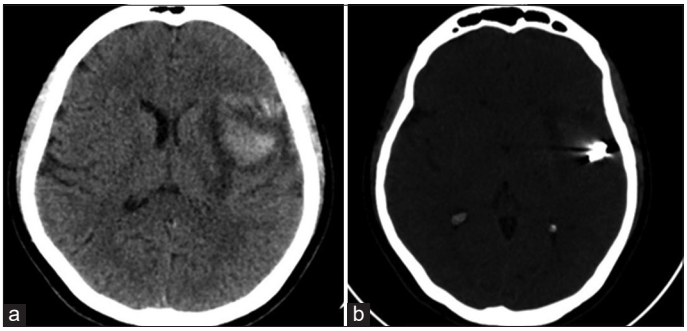

Figure 5: Non-contrast computed tomography of brain showing hyperdense bleed in the left frontal lobe (a), insular cortex, (b) showing a coil in place, surrounding streak artifacts

DISCUSSION

In pregnancy, rupture of intracranial aneurysms is common in the last trimester [2]. The distribution rate of intracranial aneurysm diagnosed in the first, second, or third trimester of pregnancy is 6%, 31%, and 55%, respectively. In our case, the patient was aged 25 years and in her second trimester of pregnancy. The risk of aneurysm development and rupture during pregnancy is related to hormonal changes and hemodynamic stress [1].

Eclampsia, pituitary apoplexy, intra-arterial occlusion, dural sinus thrombosis, intracranial space-occupying lesions, meningitis, encephalitis, and demyelinating diseases should be considered in

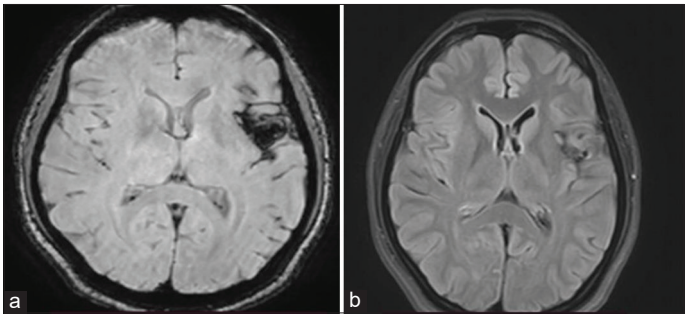

Figure 6: Follow-up magnetic resonance imaging showing hemosiderin deposition in place of the left frontal hematoma (a-T2-weighted Fast Field Echo) and Gliosis/Encephalomalacia in place of the left frontal hematoma (b-T2 fluid attenuated inversion recovery)

the differential diagnosis in cases of neurologic deficit including sudden acute headache and decreased consciousness [3].

The diagnostic tools include lumbar puncture, CT, and MRI [4]. The clinical symptomatology and clinical signs in our patient pointed toward the presence of cranial pathology. MRI showed acute intra-parenchymal hematoma and acute SAH. Magnetic resonance angiography (MRA) showed a distal left middle cerebral artery aneurysm. Cerebral angiography confirmed dissecting left M3 middle cerebral artery aneurysm. Appropriate diagnosis and treatment of SAH are important in pregnancy as the probability of recurrent hemorrhage is 33–50% and maternal mortality rates are about 50–68% in untreated cases [5].

SAH management is the same as with non-pregnant patients in the early pregnancy period. In the later period of pregnancy, an emergency cesarean section should be performed before SAH treatment [6]. Aneurysmal SAH clipping; intravascular embolization; and endovascular coiling are various modalities in the treatment of cerebral aneurysms [7].

CONCLUSION

The distribution rate of intracranial aneurysms diagnosed in the first, second, or third trimester of pregnancy was 6%, 31%, and 55%, respectively. In our case, the patient was aged 25 years and in her second trimester of pregnancy. The clinical symptomatology and clinical signs in our patient pointed toward the presence of cranial pathology. MRI showed acute intraparenchymal hematoma and acute SAH. MRA showed a distal left middle cerebral artery aneurysm. Cerebral angiography confirmed dissecting the left M3 middle cerebral artery aneurysm. Appropriate diagnosis and treatment of SAH are important in pregnancy as the probability of recurrent hemorrhage is high and maternal mortality rates are more than 50% in untreated cases.

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