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Case Report

Obstetrics & Gynecology

Pregnancy with Peripheral Arteriovenous Malformation

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Abstract

22 years old primigravida with 37 weeks singleton gestation was admitted with profuse bleeding from swelling over left anterior superior iliac spine region. Ultrasound findings suggested a high flow vascular malformation with feeder from left common femoral artery. Bleeding was managed conservatively with pressure bandage. Delivery was uneventful. Live female baby of 2.045 kg was delivered by spontaneous vaginal delivery at 38 weeks. After 4 weeks of delivery vascular malformation was confirmed with angiography and treated successfully with endovascular embolization of the major arterial feeder. In periodic follow up until 6 months, no further complication was reported by the patient. **Keywords:** Arteriovenous malformation, Peripheral AVM, Angiography, Endovascular embolization.

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INTRODUCTION

AVM is a blood vessel condition in which the arteries and veins communicate abnormally. Some of the capillaries are replaced by larger abnormal channels, known as Nidus, in AVM. The veins are directly connected to the arteries via these aberrant channels. There is no limit to where AVMs may appear in the human body. Peripheral AVMs are those that occur outside of the head, neck, and spine. This results in decreased blood flow to the surrounding tissue in a peripheral AVM. When shunting via an AVM, the heart has to work to pump the blood throughout the body. Heart failure may occur if an AVM is very large. Pain, skin breakdown, and bleeding can result from a lack of blood supply to the surrounding tissue. At the time of puberty or during pregnancy, hormonal changes seem to enhance the growth of AVM [1].

In this case report, we discuss the diagnosis and treatment of a primigravida at term with a peripheral AVM. AVM rupture is also discussed here, irrespective of where it occurs or how much of a risk it varies during the pregnancy period.

CASE REPORT

A 22-year-old primigravida arrived to the Gynecology and Obstetrics department at the Medical College in Kolkata with profuse bleeding from swelling over the left anterior superior iliac spine region (Figure 1).



Figure 1: AV Malformation

She had had trauma ten years before, during which she acquired a tiny lump measuring about 2cm x 3cm immediately above the left anterior superior iliac spine within three to four months of the accident. She was treated with steroids, antibiotics, and electrocautery at the time, but showed no substantial improvement. There was, however, no blood or discomfort at the moment.

Throughout pregnancy, the swelling expanded in size and at 37 weeks, the lesion was about $7\text{cm} \times 4\text{cm}$ in size and burst spontaneously with heavy bleeding, necessitating her admission (Figure 2).



Figure 2: Pre-embolization

A bruit was detected during the test. Pressure bandage dressings were used to control bleeding. Ultrasonography with Doppler examination of the edema revealed a high-flow vascular malformation with a convoluted feeder channel originating from the left common femoral artery. Echocardiography revealed a grade II MR with a grade II TR and an appropriate left ventricular ejection fraction (LVEF). Cardiovascular surgeons proposed that AVM may be managed postpartum. Patient was handled conservatively and spontaneously went into labor. Female baby was delivered vaginally without major vaginal hemorrhage. During the peripartum phase, no more complications or bleeding from the edema were seen.

She was readmitted to the Department of Interventional Radiology four weeks following release for angiography. Angiography of the left lower limb revealed a conglomerated tuft of capillaries in the left iliac area, fed by the left common femoral artery and draining through a tributary of the left femoral vein (Figure 3).



Figure 3: Feeding vessel in Angiography

Microcoils were used to embolize the primary arterial feeder endovascularly (Figure 4).



Figure 4: Post embolization

After three months of management, post coiling angiography revealed a significant decrease in contrast opacification of the AVM. No additional complications were seen throughout six consecutive months of routine follow-up.

DISCUSSION

AVMs may occur in any region of the body and, regardless of their location, they can rupture and cause a variety of issues. Intracranial AVM rupture has more serious consequences. Peripheral AVM most frequently occurs in the extremities and pelvis. Along with bleeding and discomfort, pelvic AVMs may cause sexual dysfunction, while AVMs in the extremities may cause varicosities and phlebitis [2]. In patients with uterine AVM, delayed secondary bleeding may occur after birth or abortion [3]. There are different perspectives on the increased risk of AVM rupture. According to investigations conducted by Robinson *et al.*, in 1974 on 24 instances of burst cerebral AVM during pregnancy, the risk of cerebral AVM rupture was fourfold that of the general population [4].

Whereas Horton *et al.*, 1990, reported 451 female patients with AVM suggested that the hemorrhagic risk in pregnant women was comparable to that in non-pregnant women [5].

Maternal hemodynamic changes, such as increased blood volume, cardiac output, and blood pressure, also have an effect on AVM, as the majority of vascular malformation ruptures during pregnancy occur between 20 weeks and 6 weeks after delivery [6].

Additionally, changes in the arterial wall, metabolic, and endocrine aspects during pregnancy may contribute to the weakening of the vascular lesion's walls. However, no unequivocal evidence exists to demonstrate an increased risk of AVM rupture during pregnancy. If an AVM is identified in a female prior to pregnancy, intervention is indicated because to the risk of bleeding and high-output heart failure, which is increased in individuals with a history of hemorrhage. If a rupture occurs during pregnancy, early intervention is advised [7].

The goal of the treatment is to obstruct the blood flow in the nidus. Symptomatic AVMs were formerly treated surgically by excision of the nidus or ligation of the principal feeding vessels. Due to recurrence, surgical excision did not provide good outcomes. Endovascular intervention with a catheter has been shown to be very effective [2].

Stereotactic Radiosurgery is also effective. Due to the potential of radiation exposure, these treatments are chosen after fetal birth to reduce fetal radiation exposure. However, in circumstances when rapid intervention is absolutely necessary, such as a ruptured cerebral AVM, these treatments should be performed immediately [8].

Peripheral AVMs present in a variety of ways, and therapy is determined by the situation's acuity, nidal architecture, and anatomic location. Treatment is often a lengthy procedure that involves numerous rounds of embolization and lifetime monitoring [10].

However, if treated correctly, most patients will experience at least symptomatic improvement after endovascular therapy, though recurrence could be a common problem. So, emphasis on the use of a multidisciplinary team is highly advocated.

CONCLUSION

AV malformations are focal abnormal conglomerations of dilated arteries and veins with disruption of the sub arteriolar space. They lack capillaries and as a consequence, suffer from AV shunting. Divergent perspectives have been noted about the increased risk of rupture of AVMs regardless of their location during pregnancy. In our research, we discovered that the AVM grew in size gradually during pregnancy, with eventual bleeding from the lesion, which supports the notion of an elevated risk of AVM hemorrhage during pregnancy. While bleeding may be treated conservatively during labor and delivery, embolotherapy remains the primary stay of treatment after birth.

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