A CASE OF PREGNANCY WITH AV MALFORMATION BRAIN

N. Sumathi

1Professor, Department of Obstetrics and Gynaecology, Government Rajaji Hospital, Madurai.

ABSTRACT

BACKGROUND
Arteriovenous malformation brain is an abnormal tangle of blood vessels. In pregnancy, cardiac output is increased leading to increased blood flow and the chance of rupture of AVM is high. Hence, careful monitoring of pregnant patient is necessary for both maternal and foetal survival.

CASE PRESENTATION
27 years, G2A1, married since 6 years with 36 weeks of gestation, a known case of arteriovenous malformation presented with headache. MRI brain showed features suggestive of pial AV malformation, left parieto-occipital region. Patient was conservatively managed and was taken up for elective LSCS under high neurosurgical risk with appropriate antiepileptic cover. An alive female body with good Apgar score delivered. Patient had an uneventful intrapartum and postpartum period.

CONCLUSION
Rupture of AVM in pregnancy can have both maternal and foetal consequences. Hence, if a patient with AVM wants to be pregnant, surgery is advised before pregnancy. Also if AVM is discovered during pregnancy, decision should be made regarding treatment risk versus the risk of haemorrhage during the remainder of pregnancy if left untreated.

KEYWORDS
Pregnancy, AV Malformation, Maternal Survival.

Financial or Other Competing Interest: None.
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Case History
27 Yr. female G2A1/Married since 6 years with 36 weeks of gestation, presented to our hospital with complaints of headache for the past 3 days. She is a known case of multi-compartmental large diffuse AV malformation in left parieto-occipital region diagnosed 7 years before in a private hospital, planned for embolisation of arteriovenous malformation, but due to social reasons procedure was abandoned. Angiography was done which revealed AVM at left parieto-occipital region 45 x 40 x 35 mm. The arterial feeders were from bilateralACA, left MCA, left PCA and bilateral occipital arteries venous shunting present with early drainage to superior sagital sinus and straight sinus planned for hypofractionated stereotactic radiotherapy, but since it was difficult (as the lesion was large) and chance of rupture are more after SRT/embolisation, it was considered that she can be kept on follow up and the procedure was abandoned.

Patient attended OP at GRH Madurai with LMP - 27/07/14 and EDD - 03/05/15. Patient had no other history of diabetes, hypertension, tuberculosis, asthma or thyroid disorder. She had history of one induced abortion one year back at 2 months due to the high risk of AVM rupture.

On admission patient was afebrile, no pallor, no pedal oedema, PR - 88/mt, BP - 100/80 mmHg, CVS - S1, S2 heard. RS - NVBS present. Per abdomen - uterus was found to be 36 weeks, not acting, head mobile. FHR - 136/mt. She was then subjected to basic investigations, which revealed Hb - 12.3 gms%, urine albumin/sugar - nil, platelets - 1.95 lakhs/mm3, RBS - 64 mg%, S. urea - 17 mg%, S. creatinine - 0.7 mg%, T. Bilirubin - 0.7 mg/dl, SGOT - 19 IU/L, SGPT - 10 IU/L, ALP - 56 IU/L all of them were found to be within normal limits. Antenatal ultrasound showed SLLUG, cephalic presentation, GA was 35 - 36 weeks, liquor - adequate, placenta - fundal posterior Grade II maturity and FHR - 146/mt.

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MRI Brain Taken

Evidence of serpiginous nidus of vessels noted involving left posterior parieto-occipital region supplied by left MCA branches and left PCA branches and dilated veins draining into superior sagittal sinus, left transverse and straight sinus. The serpiginous nidus measures 6 * 4 cm, minimal focal atrophy of the left parieto-occipital region. No evidence of mass lesion, thrombosis or haemorrhage. Other structures - medulla, pons, midbrain, basal ganglion - normal, internal capsule - normal.

Impression - features suggestive of pial AV malformation in the left parieto-occipital region is present.

MRI Brain - AV Malformation in Left Parieto-occipital Region

Neurophysician suggested for adequate hydration, monitoring of vitals and to take Tab. Paracetamol. Patient was kept under careful observation and was planned for elective LSCS in view of CPD. During the patient’s hospital stay, there was a favourable clinical condition without any neurological deterioration. Neurosurgeon opined that the patient was under high neurological risk and she could be taken up for elective LSCS under increased risk and antiepileptic cover under appropriate anaesthesia.

Patient was then assessed and taken up for elective LSCS indication being CPD/AV malformation in left parieto-occipital region, Inj. Phenytoin 2 amp given preoperatively. LSCS was done and an alive term female baby delivered whose Apgar score was 4/10 and 6/10 at 1 minute and 5 minutes respectively. Patient was then shifted to Intensive Care Unit for observation. Both her intra-operative and immediate post-operative period were uneventful. Post-operatively, she was treated with antibiotics and was discharged on 19th post-operative day. She was advised to continue the same line of management and for regular followup.

Currently, she maintains followup with Department of Obstetrics and Gynaecology and also Department of Neurosurgery.

DISCUSSION

An Arteriovenous Malformation (AVM) is a complex tangle of abnormal arteries and veins linked by one or more direct connections called fistulas or shunts. They have a high rate of bleeding than normal vessels. They can occur anywhere in the body. Brain AVM’s are of special concern, because of the damage they cause when they bleed. Their occurrence is very rare and is less than 1% in general population. AVM’s that occur in the covering of brain are called dural AVM. AVM’s occurs due to the abnormal development of blood vessels in utero and may be present since birth. Most AVM’s are not inherited. Dural AVM’s in adults are an acquired disorder that can occur following an injury. AVM can occur in people of all races and sexes in almost equal proportion. The typical time of discovery is between 20 - 40 years.

When on AVM bleed, there is 10% - 15% risk of death related to each bleed and a 20% - 30% chance of permanent brain damage the risk of bleeding is higher in the first years after first bleed. In about 50% of the patients, the presentation is sudden haemorrhage/bleeding into the brain in the form of stroke. Other potential complications include seizures, headache and stroke like symptoms including weakness/paralysis on one side of the body, numbness and tingling sensation, problems with vision, hearing balance, memory and personality changes. The most important complication following AVM rupture in pregnancy is the possibility of subsequent re-bleeding.[1] AVM’s are usually diagnosed by CT angiography, where an AVM’s will show up an abnormal tangle of blood vessel and also to detect the bleeding and fluid spaces around the brain. This is the most accurate test. MRI Brain which utilises the pulse sequences is specifically designed to show the arteries and veins of the brain as well as AVM. CT scan is to detect the bleeding and fluid spaces around the brain.

There is an increased risk of haemorrhage from an AVM during pregnancy, usually after the first 3 months of pregnancy. This haemorrhage does not occur only during labour. It is thought to be due to increased blood circulation that occur during pregnancy. [2] Haemorrhage during delivery has been a major concern of obstetrician and patient; however, the studies suggest that in most cases vaginal delivery does not carry a higher risk for haemorrhage than delivery by caesarean section. There are no data available to address whether the caesarean section helps to reduce the already low incidence of AVM associated complications during labour, although there is evidence that increased venous pressure during Valsalva manoeuvre is not directly transmitted to the draining veins. On the other hand, if a women anticipates pregnancy and has a known AVM, treatment should be considered before the pregnancy. If the lesion is discovered during pregnancy, a decision should be made regarding the treatment risk versus the risk of haemorrhage during the remaining period of pregnancy of the lesion. Unruptured AV malformation in pregnant women generally needs only conservative management due to low rupture risk. However, surgery is often indicated due to increased risk of re-rupture and associated mortality.[3]

- Individuals with cerebral AVMs are at a moderately higher risk for seizures; risk depends on the location of the AVM and the history of intracranial haemorrhage or focal neurologic deficit. No evidence suggests the use of antiepileptic medications for prophylaxis of individuals who have an AVM, but have never had a seizure.

- Headache of acute onset without localising neurological signs may be the presenting sign of a haemorrhage, either intraventricular or subarachnoidal, and need immediate assessment by neuroimaging.

- For AVM-associated headaches that are not associated with an intracranial haemorrhage, standard analgesia for
headache may be used, either nonspecific or migraine specific.

Invasive treatment of AVMs may include endovascular embolisation, surgical resection and focal beam radiation, alone or in any combination. The surgical treatment risk has traditionally been estimated by the Spetzler-Martin grading scale, which includes grades I-V.

The current American Heart Association Multidisciplinary Management Guidelines for the treatment of brain AVMs recommend the following approach.

Spetzler Martin GR I and II
Surgical extirpation.

Spetzler Martin GR III
Embolisation followed by surgical extirpation.

Spetzler Martin GR IV and V
Combination of embolisation, radiotherapy, radiotherapy and surgery.

Surgical resection is the mainstay of definitive treatment and is most effective with more easily accessible lesions of smaller size.

Arterial feeders are isolated and ligated. Then the nidus is resected. The draining veins are ligated last, so that the pressure is not increased while the nidus is being resected. Arterial aneurysms may be clipped surgically as well. Intraneural aneurysms are resected with the AVM. Distal aneurysms are usually flow related and resolve when the AVM is resected.

Post-surgical angiography is done routinely to ensure that no residual AVM exists.

Endovascular Embolisation
- Superselective endovascular treatment includes delivery of thrombosing agents such as quick-acting acrylate glue (N-butyl cyanoacrylate [NBCA]), thrombus-inducing coils, Onyx liquid embolic fluid or small balloons into the AVM nidus.

- The presence of embolic material within the AVM make surgery and radiosurgery safer and more accurate.

Radiosurgery
Radiosurgery is an option that is generally used to treat AVMs that are approximately 3 cm in diameter or less.

Endovascular embolisation is widely accepted as an important component of contemporary, multimodal therapy for rupture of arteriovenous malformations.

If left untreated, this also must include the potential risk to the future during detention wherever it be by embolotherapy, surgical extirpation, radiation and the associated diagnostics test.

CONCLUSION
Cerebral arteriovenous malformations infrequently complicate pregnancy. Conservative management is generally recommended for low risk and unruptured arteriovenous malformation and careful monitoring of these cases in antenatal period is necessary. However, surgical mode of treatment preferably endovascular embolisation is generally opted in ruptured cases, since the chance of re-rupture is high in these cases.

REFERENCES