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Case Report

Uterine Arteriovenous Malformation-A Rare Cause of Abnormal Uterine Bleeding

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Abstract

We report a rare case of arteriovenous malformation(AVM) presented as abnormal uterine bleeding diagnosed following two spontaneous abortions. Patient was referred from a private hospital for excessive uterine bleeding with severe anemia following spontaneous miscarriage. Clinically incomplete miscarriage was diagnosed. Ultrasound(USG) confirmed and revealed retained products of conception along with presence of A-V fistula. Evacuation was performed and severe anemia was treated with one unit of blood transfusion. A week later patient presented with another episode of heavy bleeding. Repeat USG revealed the presence of retained products with ?AVM or ?Vesicular mole. Vesicular mole was excluded by estimation of serum β HCG. The patient was managed medically using hormones and two units of blood transfusion.

Key words: Arteriovenous malformation, abnormal uterine bleeding, severe anemia.

Introduction

Uterine arteriovenous malformation (AVM) is not a common entity with a predicted rough incidence of about 4.5% as quoted by O'Brein et al[(1)]. Exact incidence is not known.AVM may present as a rare cause of menorrhagia or abnormal uterine bleeding. It should be considered in patients who with profuse or torrential genital bleeding and in refractory cases of menorrhagia not responding to conventional measures. AVM can be diagnosed by color Doppler, CT, MRI & Angiography. Angiography is the gold standard for diagnosis. Catheter angiography and embolization are very effective in defining the vascular anatomy and treating uterine vascular abnormalities. In the past, hysterectomy was the only remedy. Recent reports have mentioned successful conservative management such as long term hormonal therapy, surgical removal of AVM or laparoscopic bipolar coagulation of the uterine arteries.

Case History

A lady aged 27 years, A_2P_0 with marital life of 3 years, was referred from a private hospital for further management of heavy bleeding per vagina following spontaneous miscarriage with severe anemia. She had her 1st conception after 6 months of marital life resulting in spontaneous complete miscarriage at third month. It was followed by profuse vaginal bleeding for which she was treated with 2 units of blood transfusion. Thereafter patient had regular menstrual cycles for 2 years.

Her second conception also resulted in spontaneous miscarriage at 2 months of gestation for which she underwent evacuation at private hospital. A week later presented with another episode of profuse uterine bleeding. She was thus referred to our hospital for control of bleeding and correction of anemia. On admission she was hemodynamically stable and was diagnosed to have incomplete miscarriage. USG revealed retained products of conception along with presence of AVM.

Hemoglobin (Hb) was 6.7g%. Evacuation was done with 1 unit of O+ve blood transfusion. Bleeding was controlled and patient got discharged with an advice to come for follow up 2 weeks later.

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However patient presented a week later with another bout of severe bleeding. On readmission (20/8/12), investigations revealed Hb 5g%, Platelets-1,50,000, Urine pregnancy test and CRP negative, Serum β HCG within normal limits. Repeat USG and color Doppler reinstated the presence of AVM with minimal endometrial collection. Severe anemia was corrected with 2 units of blood transfusion .Persistent bleeding was controlled by oral Medroxyprogesterone 30mg/day. Second opinion reconfirmed the presence of AVM.

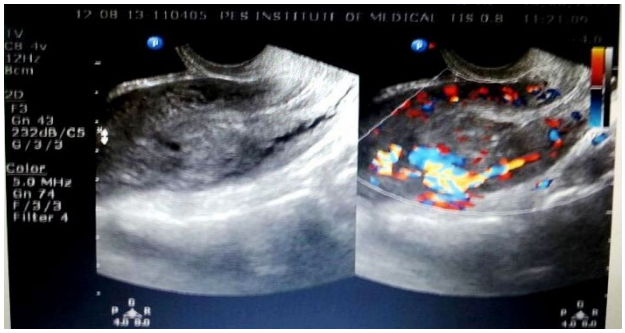

Figure 1: USG – Thickened endometrium with heterogenous increased vascularity. Possibility of retained products/ Invasive vesicular mole with A-V Fistula.

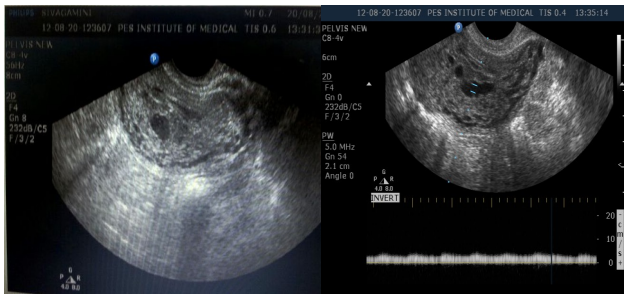

Figure 2: Uterus 6.98x6,6.x 4.5cm. Endometrial collection+. ET- 7 mm. At the junction of endomyometrial junction there is serpigenous dilated channel possibility of AVF to be considered . No evidence of retained products of conception Right ovary-5.2X3.6X3cm,large cyst seen, Left Ovary – 2.8X1.5 cm.

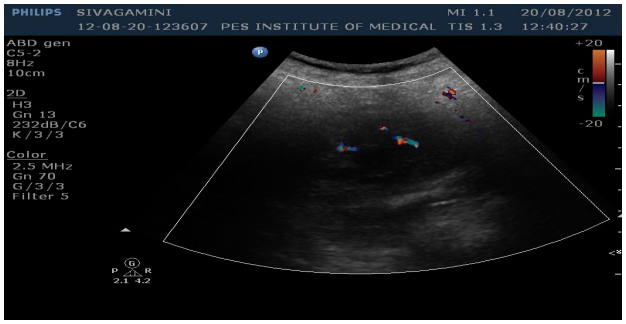

Figure 3: Ultra sound scan (color Doppler) showed increased myometrial color flow having a mosaic pattern. Showed high velocity increased diastolic flow, having low resistance index, suggestive of arterio venous malformation.

The first case of AVM was reported in 1926 by Dubreuil and Loubat.[2] Uterine AVM are classified as congenital or acquired. Congenital AVMs may be isolated or may occur in association with AVM in other organs. It could be due to abnormal development of primitive vessels that result in connections between pelvic arteries and veins in the uterus without an intervening capillary bed.[3] Acquired uterine AVMs are formed by communications between the uterine arteries and the myometrial veins. It is caused by an iatrogenic event or a pathological condition like previous uterine trauma (such as uterine curettage), gestational trophoblastic disease, caesarean section, intrauterine contraceptive devices, and necrotic chorionic villi invading venous sinuses. [3]

In our case, it is difficult to conclude as we do not have previous scan/color Doppler of first miscarriage event. Presence of uterine AVM may increase the size of uterus and a pulsatile mass may be felt in the vagina. Bruit may also be heard on auscultation. Though angiography remains the gold standard imaging technique for diagnosis of uterine AVM,[4] our case was diagnosed by color doppler sonography. Wiebe and Switzer favored the diagnosis of AVM by color Doppler sonography.[5] Management depends on the age of the patient, her desire for future fertility and severity of bleeding, site / location of AVM in uterus. [6,7] In the past, treatment had been confined to hysterectomy. In the last decade, an increasing number of women have been treated conservatively with success and hysterectomy is no longer considered essential.[8]

In a stable patient, expectant management may have a role with spontaneous resolution of the lesion. [5,6] Acute management includes measures to stabilize the patient, uterine tamponade with Foley's catheter or rolled gauze packing or medically with estrogens, progestins, methylergonovine, danazol, and 15-methyl -prostaglandin $F_2\alpha$.[6,7,9] Other conservative methods like surgical removal of an AVM, laparoscopic bipolar coagulation of the uterine blood vessels and long-term medical therapy with combined oral contraceptive pills are also available. Uterine artery embolization with different materials used singly or in combination is another method to control the hemorrhage in AVM.

Obstetric importance of AVM is yet to be explored in detail. Vascular malformations as a cause of PPH (postpartum haemorrhage) has been reported by K.Hayes.[10] Most often acquired AVM produces secondary PPH rarely congenital may be responsible for primary PPH.A few articles have reported the association of AVM with placenta accreta with torrential haemorrhage. Patients with recurrent miscarriage have been reported to have AVM diagnosed by HSG &

MRI. Selective transcatheter embolization of AVM resulted in successful IVF twin pregnancy. [11] Suggested complications like growth restriction and malpresentations may occur. Uterine rupture may also occur in treated uterine AVM.[12]

Conclusion

Uterine AVMs though rare are potentially life threatening lesions. Although angiography is considered to be the gold standard for diagnosis, USG with color Doppler is effectively a noninvasive practical approach in detection of AVM. Angiography can be selectively used for elective management. Conservative management of AVM has evolved from hysterectomy to uterine artery embolization which is a safe and effective method of treatment.

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