

Case Report

Uterine Arteriovenous Malformation As A Rare Cause Of Menorrhagia

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Abstract:

Uterine arterio venous malformation is uncommon cause of menorrhagia. We report a rare case of arteriovenous malformation diagnosed after 18 years of suffering from menorrhagia.

Key Words: Uterine arteriovenous malformation, Menorrhagia

Introduction:

Uterine arteriovenous malformation (AVM) is a rare cause of menorrhagia. It is important to keep in mind the possibility of uterine AVM in refractory cases of menorrhagia not responding to conventional measures. AVM can be diagnosed by color Doppler ultrasonography, computed tomography, magnetic resonance imaging and angiography. In the past hysterectomy was the only remedy. Recent reports have mentioned successful conservative management such as surgical removal of AVM, laparoscopic bipolar coagulation of the uterine arteries and long term medical therapy.¹ Uterine artery embolization is also effective in controlling hemorrhage.²

We report a case of uterine AVM, where hysterectomy was performed

Case Report:

A 40 years old lady, married for 22 years, was admitted in the department of Gynecology and Obstetrics in N.R.S Medical College with history of menorrhagia for 18years. Obstetric history revealed that she conceived 4 years after marriage which resulted in spontaneous abortion for which curettage was done. Since then she developed menorrhagia. Blood loss was so heavy that she was compelled to be admitted in hospital for several times for blood transfusion, in spite of taking different hormonal preparations.

She had been investigated for secondary infertility also and reports showed no abnormality in ovarian and tubal factors.

On admission she was severely anemic. Hemoglobin was 4gm%. She received 6 units of blood transfusion. Ultra sound scan (color Doppler) showed grossly dilated and tortuous vessels in both adnexal regions, more prominent on the right side. There was increased myometrial color flow having a mosaic pattern. Flow in the vessels of myometrium and adnexal region showed high velocity increased diastolic flow, having low resistance index, suggestive of arterio venous malformation.

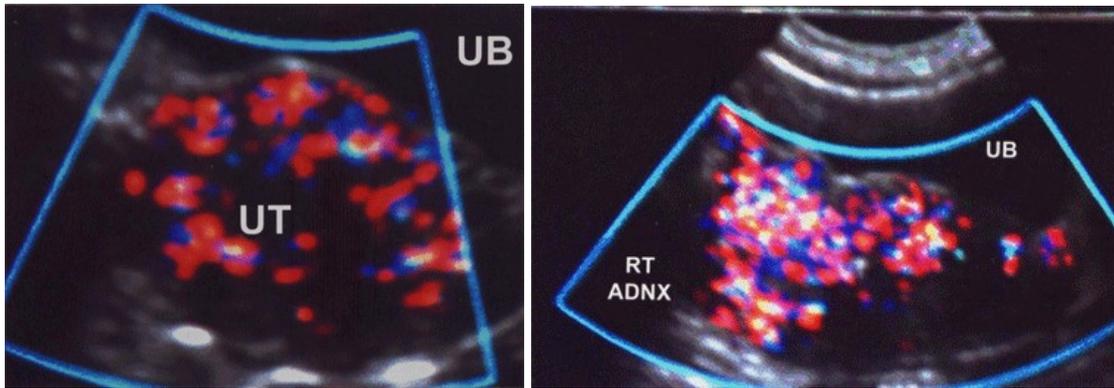


Figure 1 (Left): Doppler ultrasonography of uterus with increased myometrial colour flow having mosaic pattern; Fig 2: Grossly dilated and tortuous vessels in both adnexal regions,more prominent or right side

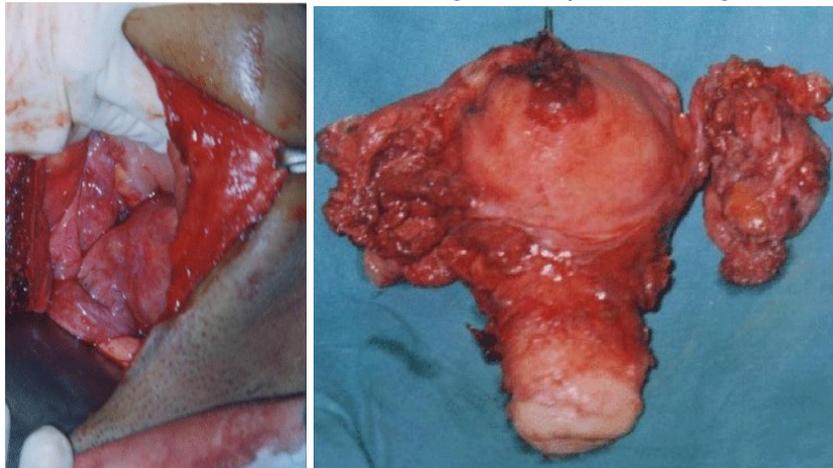


Fig 3 (Left): Part of uterus with congested blood vessels in right adnexa during operation; Fig 4: Uterus and both adnexae after operation



Figure V: Histological sections of myometrium showing dilated and congested vascular spaces lined by endothelial cells in absence of any specific lesion (H&E, X100)

Pelvic angiography and MRI were not done as these facilities were not available in our hospital.

Total hysterectomy with bilateral salpingo oophorectomy was done after counselling the patient (patient was not desirous for child and she wanted to get rid of the uterus).

Histopathological examination of uterus and ovaries showed plenty of congested blood vessels and irregular blood spaces in myometrium without any specific lesion. There were features of chronic cervicitis. In the ovaries also there were fair number of congested blood vessels. No neoplastic lesion was seen.

Discussion:

The first case of AVM was reported in 1926 by Dubreuil and Loubat.³ It consists of proliferation of arterial and venous channels with fistula formation and admixture of small capillary like channels. In many cases, distinction between artery and vein becomes blurred due to secondary intimal thickening in the veins as a result of increased intraluminal pressure.

Uterine AVM may be congenital or acquired. Congenital uterine AVM may be isolated or may occur in association with AVM in other organs. Acquired AVMs may be due to previous uterine trauma (such as uterine curettage), gestational trophoblastic disease, caesarean section, intrauterine contraceptive devices, necrotic chorionic villi invading venous sinuses.⁴

In our case uterine AVM was acquired in nature as it started after post abortal curettage. Uterus was normal in size. We did not get any audible bruits or pulsatile masses at vaginal examination. Though angiography remains the gold standard imaging technique for diagnosis of uterine AVM⁵, our case was diagnosed by colour doppler sonography. Wiebe and Switzer reported seven cases of AVM diagnosed by colour Doppler sonography.⁶

Management depends on the age of the patient, her desire for future fertility and severity of bleeding. 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. Acute management includes measures to stabilize the patient, uterine tamponade with Foley's catheter or rolled gauze packing, and medical therapies like estrogens, progestins, methylergonovine, danazol, and 15-methyl-prostaglandin F₂alpha. In stable women, expectant management, surgical removal of an AVM, laparoscopic bipolar coagulation of the uterine blood vessels, and long-term medical therapy with combined oral contraceptive pills are reported.¹ Recent reports have described successful treatment of uterine artery embolization with different materials used singly or in combination such as autologous blood clot, gelfoam, microfibrillar collagen, polyvinyl alcohol, isobutyl cyanoacrylate and steel coil spring occluders.⁷ Gonadotropin-releasing hormone agonists have been used as an adjunct to embolization and 6 months of therapy reduced the size of a uterine AVM from 5.1 x 3.8 cm to 1.4 x 1.0 cm.⁸ Subsequent uterine artery embolization resulted in complete disappearance of the AVM, and normal cycles were resumed 3 months later. The authors concluded that gonadotropin-releasing hormone agonist therapy may be useful in situations where embolization needs to be postponed.

Our patient was 40 years old and not desirous of having child. Moreover she was so much disgusted with the continued suffering from menorrhagia, that she refused any kind of conservative management and so hysterectomy was planned for. Fleming et al reported six cases of AVM who underwent total abdominal hysterectomy for life threatening bleeding.⁷ Successful uterine artery embolization with polyvinyl alcohol particles in three cases was reported by AA Nicholson et al. All of them resumed normal menstruation and one of them had successful pregnancy also.² Prabhu et al also reported a case of uterine AVM treated with spring coil, who resumed normal menstruation with the procedure.⁹

Conclusion:

Uterine AVMs though rare are potentially life threatening lesions. Though hysterectomy was the only treatment for it in the past, uterine artery embolization is a safe and effective method of treatment when uterine function is to be preserved. The case is reported here not only for its rarity but also to highlight the delay in its diagnosis. For long eighteen years she was treated by different doctors with various hormonal preparations. Had she been diagnosed earlier and treated by uterine artery embolization, her uterus would have been saved and she might conceive. So while dealing with a case of refractory type of menorrhagia one should always consider the possibility of uterine AVM.

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