

Case Report

Refractory Postpartum Haemorrhage - A Case Report

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Abstract

A case of uterine haemorrhage in the puerperium where conventional treatment failed with the final diagnosis of arteriovenous malformations which required uterine tamponade, and GnRH agonists. A refractory case of PPH.

Key words: Secondary postpartum haemorrhage, Arterio-venous malformations .

Introduction

Uterine arteriovenous malformations (AVM) are rare lesions with a considerable risk potential. Clinical presentation varies from no signs over various degrees of menorrhagia to massive life-threatening vaginal bleeding. Clinical suspicion is essential for a prompt diagnosis and treatment. Recent reports have mentioned successful conservative management such as surgical removal of AVM, laparoscopic bipolar coagulation of the uterine arteries and long term medical therapy¹. Uterine artery embolization is one effective management for controlling hemorrhage².

Case History

A 20 years old lady, married for 1½ years, was admitted in the department of Gynaecology and Obstetrics in Dr.B.R. Ambedkar Medical College and Hospital, Bangalore, with excessive bleeding per vagina since two months. Obstetric history revealed that she is P1 L1, Last child birth - 2 months back. Patient underwent emergency Caesarean delivery two months ago, in a private institution for IUGR with PROM with Oligohydraminos. Post operative period was uneventful. Twenty four days after surgery, she had excessive vaginal bleeding for one day. She changed about 15 pads in one day.

Bleeding was associated with abdomen pain and passing of clots. She was admitted to another private hospital where 2 units of blood were transfused. Bleeding stopped following symptomatic Rx and patient discharged. Four days later, patient had one more episode of excessive vaginal bleeding which was managed conservatively in another hospital. She had a total of four episodes of heavy vaginal bleeding following which she went to a Govt., hospital where 3 units of blood was transfused and D & C was done. Her serum β hCG values were normal for non pregnant condition (< 0.1 units /ml).

Episodic bleeding per vagina still persisted for 20 days following which patient reported to Dr B R Ambedkar Hospital (02 months from the LSCS). On admission, Hb% was 7.4 gm%. 02 units of blood were transfused. USG with Doppler study: Dilated and tortuous arteries in the anterior wall of lower body of uterus with involvement of left lateral wall- ? Vascular malformation. Diagnostic hysteroscopy showed FLIMSY COBWEB LIKE adhesions in the uterine cavity with multiple big and medium size blood vessels traversing through them. (Fig 1, 2,). Ball cautery at the bases of the vessels was not possible as it was very difficult to visualise the bases of several vessels due to cotton wool adhesions and highly intertwining anastomotic vessels all over in the uterine cavity.

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On POD 3 of diagnostic hysteroscopy, Inj Leuprolide Depot 3.75mg SC was given. --- bleeding apparently stopped. On POD 5, patient had sudden episode of heavy bleeding P/V with passage of clots.

Uterine cavity balloon tamponade was done with Foley's catheter No. 16 with bulb inflated with 30 ml of distilled water. The Catheter was removed on POD 8 (after three days). No fresh episodes of bleeding PV or pain abdomen. Patient was asymptomatic and was discharged on D16 following admission.

Follow up - Patient has regular cycles. No further episodes of excessive bleeding.

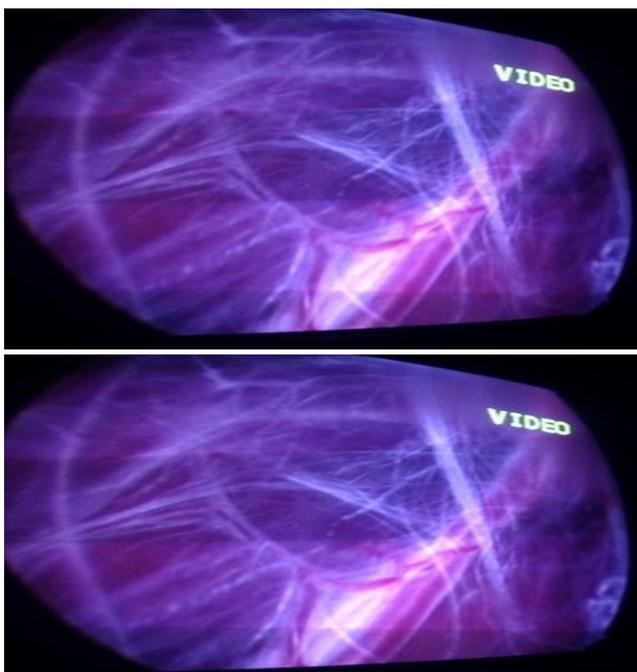


Figure 1&2: Diagnostic hysteroscopy showed FLIMSY COB-WEB LIKE adhesions in the uterine cavity with multiple big and medium size blood vessels traversing through them.

Discussion

The first case of AVM was reported in 1926 by Dubreuil and Loubat³. 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, and necrotic chorionic villi invading venous sinuses.⁴ In our case, uterine AVM was acquired in nature as it started after caesarean section.

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, methyl-ergonovine, 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 reported¹. Recent reports have described successful treatment of uterine artery embolization with different materials used singly or in combination such as autologous blood clot, gel foam, microfibrillar collagen, polyvinyl alcohol, isobutyl cyanoacrylate and steel coil spring occluders⁷.

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 cm⁸. 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 procedures are not available or can be postponed.

Conclusions

Uterine AVMs though rare are potentially life threatening lesions. Though hysterectomy was the only treatment in the past, uterine artery embolization and GnRH agonists are safe and effective method of treatment when uterine function is to be preserved.

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