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Acquired uterine arteriovenous malformation - a rare cause of genital bleeding post lower segment caesarean section (LSCS)

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Abstract: Uterine Arteriovenous malformation (AVM) are uncommon vascular lesions, but may cause potential life threatening genital bleeding following lower segment caesarean section. It is diagnosed by Color Doppler, CT, MRI and angiography. Previously hysterectomy was the only option for its treatment but uterine artery embolization provide a good, fertility saving alternative treatment. Here we are presenting a case of uterine bleeding, post lower segment caesarean section (LSCS). She was investigated and found to have acquired uterine arteriovenous malformation which was treated successfully with uterine artery embolization.

Keyword: Uterine arterio venous malformation, Uterine artery embolization

INTRODUCTION:

The first case of uterine arteriovenous malformation (AVM) was reported in 1926 by Dubreuil and Loubat(1). Uterine arteriovenous malformation are classified as congenital and acquired. Congenital AVMs, may be isolated or may occur in association with arterio venous malformation in other organ.Congenital AVMs represent an abnormal embroyologic differentiation of primitive vascular structure and/or the lack of development of capillary plexus, which result in direct connections between arteries and vein without the interposition of a capillary bed(2,3). Congenital AVMs may extend beyond the anatomic limits of the uterus into the pelvis (2). Acquired uterine arterivenous malformation (AVMs) are formed by communication between the uterine arteries and myometrial vein. It is caused by an iatrogenic events or a pathological condition like previous uterine trauma such as uterine curettage, gestational trophoblastic disease, caesarean section, intra uterine contraceptive device and necrotic chorionic villi invading venous sinuses(4).

CASE REPORT:

A 23 year old lady, P1L1 presented to us with complaint of profuse bleeding per vaginum, acute in onset of 3 days

duration, in the form of daily soaking of sanitary pad, passage of clot, restriction of social life and easy fatiguability. She had no family history of bleeding disorder. She underwent lower segment caesarean section 8 month back (indication -fetal distress). Post operative period was uneventful. Her antenatal scan done before were normal with no evidence of uterine AV malformation. She resumed menstruation 2 month after childbirth and hormal menstrual cycle of 3/30 till this episode.

On examination she was pale but hemodynamically stable. Pelvic examination revealed bulky uterus, bilateral fornices free and bleeding from cervical os was noticed. Urine pregnancy test was negative. Hemoglobin was 6.4gm%, platelet -2.4 lakh, B-hCG not detectable, congulation profile, thyroid, liver and kidney function test were normal. Transabdominal and transvaginal ulrasonography showed 4.1x3.7x4.1cm sized heterogenous mass with multiple cystic areas in the anterior wall of lower uterine segment close to the scar (fig.1). Colour flow mapping revealed increased vascularity with in the mass suggestive of arteriovenous malformation, confirmed with MRI (fig.2) and angiography.

Diagnostic angiogram showed moderate to low flow uterine AV malformation with feeder from spiral artery of left uterine artery draining into left internal iliac vein. Mildly increased blush and filling of arteriovenous malformation from right uterine artery noted (fig.3). Initial management of anaemia correction was done to stabilize patient immediately.

In total she was given five unit of packed cell. As the patient was young and willing for conservative management for preservation of her fertility, case was discussed with interventional radiologist and the decision of uterine artery embolization was taken. Diagnostic angiogram showed uterine arteriovenous malformation. Bilateral uterine artery embolization was done and on follow up patient was relieved of her complaint. She resumed a normal menstrual cycle a month later. Follow up ultrasonography with doppler showed no residual uterine arterio venous malformation(fig.4).



Figure 1: Ultrasound demonstrates heterogenous mass with multiple cystic areas in the anterior wall of lower uterine segment with increased vascularity.



Figure 2 : MRI Pelvis showing moderate sized mixed intense signal lesion projecting from anterior body of uterus and communication with uterine cavity.







Figure 4 : Follow up ultrasound scan with doppler showed no residual uterine arterio venous malformation.

DISCUSSION:

We present a case of acquired uterine arteriovenous malformation in 23 years old women that occurred secondary to lower segment caesarean section. Uterine arteriovenous malformation is a rare cause of uterine bleeding. The true incidence of which is unknown

An Initiative of The Tamil Nadu Dr. M.G.R. Medical University University Journal of Surgery and Surgical Specialities but with increased use of ultrasound, O'Brien et al(5) proposed a rough predicted incidence of 4.5%. Uterine AV malformation can be congenital and acquired. In our case uterine arteriovenous malformation was acquired in nature as patient's symptoms started eight month after lower segment caesarean section and her previous antenatal scan showed no evidence of uterine arteriovenous malformation. Clinical scenario varies from silent, non harmful condition to potentially life threatening condition. An accurate diagnosis is important for prompt management. 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. Ultrasonography will often show non specific heterogenous or anechoic tortuous space in the myometrium. Color and spectral Doppler USG shows further detailing of a tangle of vessels producing a "color mosaic" pattern with multi directional high and low velocity flow (5,6). Endometrial curettage a procedure commonly performed in patient with abnormal vaginal bleeding aggravates the genital hemorrhage of a uterine AV malformation (7). Management depends on hemodynamic stability, amount of bleeding, patient's age and her desire to preserve fertility. Acute management include measures to stabilize the patient, uterine tamponade with foley's catheter or rolled gauze packing and medical therapies and like estrogen, progestins, danazol 15-methyl-prostaglandin F2 alpha. Management of AVM has evolved from hysterectomy to uterine artery embolization. Uterine artery embolization is a safe and effective method of treatment. It has several advantages over surgery, which includes higher success rate. less morbidity, and preservationof fertility [8] Uterine artery embolization (UAE) was first described by RAVINA et al in 1995(9). Arteriography can define the main arterial supply to the AVM, the presence of a niders, the size of arterovenous stunting and venous drainage. Early venous drainage into the endometrial or myometrial vein is an angiographic sign of arteriovenous shunting seen in AVM (10).

CONCLUSION:

Vascular lesion of the uterus in the form of aquired arterio venous malformation following lower segment caesarian section are rare but they should be considered in those who present with profuse genital bleeding, since these lesion can be treated effectively with uterine artery embolization but may be worsened by uterine curettage. **REFERENCES:**

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