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Case Series of Uterine Arteriovenous Malformation and Its Varied Treatment Modality

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Abstract:
Uterine arteriovenous malformation (AVM) or enhanced myometrial vascularity is a rare, life threatening cause of abnormal uterine bleeding.It can be congenital or acquired. Uterine AVM typically presents with abnormal uterine bleeding. An AVM is suspected when an abnormal uterine bleeding is followed by post cesarean section, miscarriage, dilatation and curettage. Diagnosis is made by imaging studies such as transvaginal sonography(TVS) with doppler or Magnetic resonance imaging (MRI) pelvis with or without angiography or digital subtraction angiography(DSA). Treatment modality can be conservative or uterine artery embolization or hysterectomy. Here we report a case series of 8 cases,most of them presented with abnormal uterine bleeding and their various treatment modalities.

Keywords: uterine arteriovenous malformation, transvaginal sonography, conservative,uterine artery embolization.

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I. Introduction:

Uterine arteriovenous malformation (AVM) or enhanced myometrial vascularity is a rare life - threateningcondition. The incidence of uterine arteriovenous malformation is unknown. Uterine arteriovenous malformation is an abnormal and nonfunctional fistula formation between arteries and veins without capillary intervening network in the uterine wall. Patient typically presents with vaginal bleeding that can be spotting p/v or massive, life threatening in some instances. If an arteriovenous malformation is suspected imaging technique should be performed. Transvaginal ultrasound with color doppler is the first line imaging technique. MRI pelvis or MRI pelvis with or without angiography, DSA may also be performed. Management of AVM is multifactorial depends upon severity of hemorrhage,fertility status of the patient. Most of them require uterine artery embolization or hysterectomy, some may require conservative management.

Here we are reporting a8 case series of uterine AVM, we had at our institute Sri Ramachandra Institute of Higher Education and Research (SRMC RI/SRIHER,Chennai) during the year of 2015 -2020.

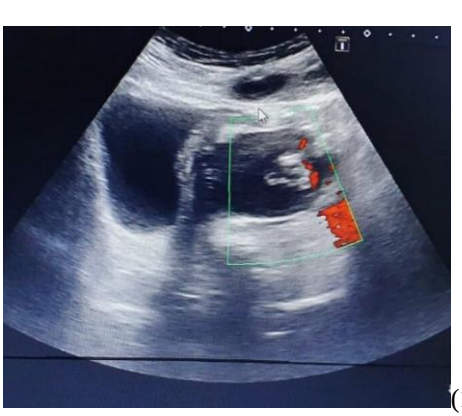
II. Case Series

CASE 1:

A 39yearold P1L1A1 previous LSCS presented with complaints of heavy bleeding p/v associated with passage of clots and pain abdomen. Three weeks earlier, she had history of second trimester medical termination of pregnancy (MTP)(suction and curettage) with laparoscopic sterilization. On examination she was hemodynamically stable with hemoglobin of 10.4g/dl. TVS pelvis showed Thickened and echogenic cavity with an endometrial thickness of 21 mm.There was an increased vascularity of uterus noted suggestive of AVM. Interventional radiologist opinion obtained and then proceeded with Bilateral uterine artery embolization. Post procedure no complications were encountered.

CASE 2:

A 27year old P2L2 previous LSCS presented with complaints of heavy vaginal bleeding associated with passage of clots. 48 days earlier she underwent emergency LSCS with sterilization in view of previous LSCS in labor. Her postnatal period was uneventful. On examination she was pale and stable with hemoglobin of 7.1 g/dl. In-addition her beta hcg was 5.3 mIU/mL. 3unit packed cell was transfused.TVS pelvis done outside showed increased myometrial vascularity with endometrial thickness of 14mm. MRI pelvis done showed an irregular enhancing lesion 1.4×1.3 cm, isointense to endometrium is seen in the fundus of the uterus involving the endo-myometrial interface, causing localized junction distortion. On dynamic enhanced study, tortuous vascular channels are seen within the fundus of the uterus with arterial feeders predominantly from right uterine artery and then proceeded with bilaterally uterine artery embolization. Post embolization patient was stable and no complications were encountered and patient was discharged on POD 3.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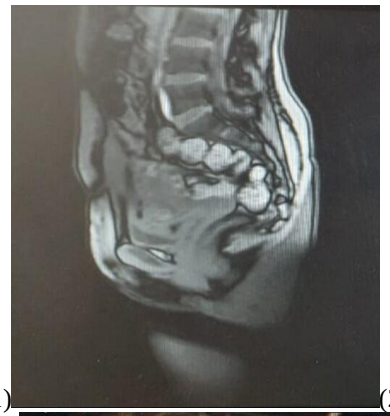
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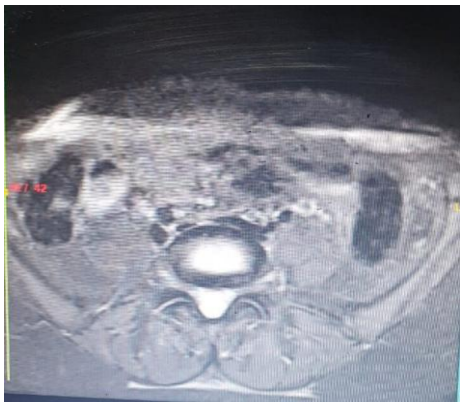
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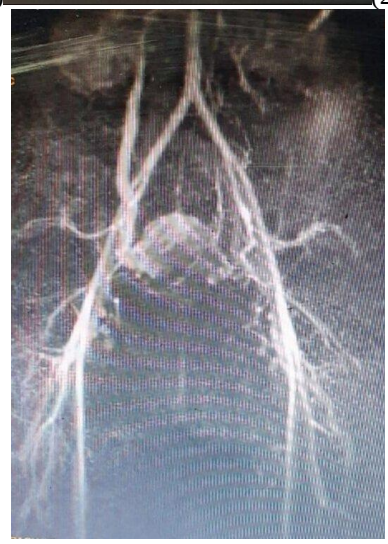
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Figure: 1 TVS pelvis with showing increased vascularity,
Figure 2 to 3 MRI irregular lesion involving endo-myometrial interface and Figure 4-showing tortuous blood vessels from right uterine artery.

CASE 3:

A 28year old P2L2A1 presented with intermenstrual bleeding p/v pain abdomen.She had an history of I trimester medical abortion.She presented with heavy bleeding per vagina managed medically outside after 1 ½ month followed by medical abortion.Upon examination she was hemodynamically stable with hemoglobin of 12.2g/dl , beta hcg-0.8 mIU/mL. TVS with doppler showed ill-defined hypoechoic area in the posterior myometrium. There is venous and arterial flow within the area no evidence of retained products of conception. Managed conservatively with cyclical oral contraceptive pills (OCP) for 3 months.

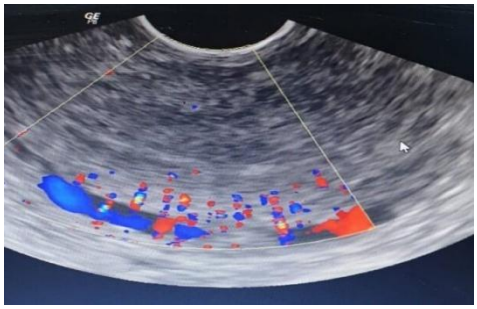

Figure 5 -TVS pelvis with doppler showing AVM in posterior myometrium.

CASE 4:

A 30 year old P2L2 presented with complaints of heavy vaginal bleeding associated with passage of clot.One month earlier she underwent open myomectomy for abnormal uterine bleeding -fibroids.On examinations she was hemodynamically stable and her hemoglobin was of 6.2g/dl. 3 units packed cell were transfused. TVS pelvis showed thickened endometrium with increased vascularity was noted? Arteriovenous malformation and then proceeded with Bilateral uterine artery embolization. Bilateral uterine artery embolization done using gelfoam. Post procedure arteriogram showed complete obliteration of abnormal vessels and no complication was encountered.

CASE 5:

A 36year old P1D2 presented with complaints of heavy bleeding p/v. Two months earlier. she had intrauterine fetal demise (DCDA twin) at 24 weeks delivered vaginally. She had retained placenta for which she underwent manual removal of placenta and 3unit packed cell was transfused. She had history of spotting p/v 1 month back which was managed medically and beta hcg was 38.4mIU/mL. On examination she was pale and her hemoglobin was 9.4g/dl, per speculum bleeding through os present and clots noted. Beta hcg was 48.4mIU/mL. 1unit packed cell was transfused. TVS pelvis showed Cavity appeared thickened with cystic spaces with vascularity noted in color doppler and arterial flow noted in few areas. MRI pelvis showed feature of arteriovenous malformation in uterine cavity of size 4.6*2.3*3.7 cm in the fundal region and myometrium showing few feeders from bilateral uterine artery and then proceeded with bilateral uterine artery embolization. Both uterine arteries were embolized with polyvinyl alcohol and gelfoam. The post procedure arteriogram showed complete obliteration of arteriovenous malformation and no complication were encountered.

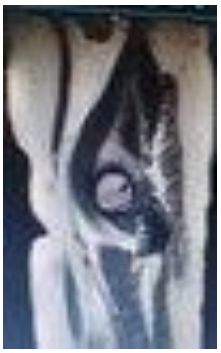

Figure 6 showing AVM in fundal region

CASE 6:

A 23year old A1 with history of missed abortion 2 ½ months earlier which was managed medically presented with complaints of spotting p/v on and off with heavy bleeding p/v for 1 day. Her last menstrual period was 2 weeks earlier. On examination she was stable and her hemoglobin was 10.2g/dl. TVS pelvis showed thickened cavity with endometrial thickness 8.7 mm vascularity noted suggestive of arteriovenous malformation.In view of high suspicion for retained products of conception or arteriovenous malformation consent for suction evacuation and embolization of uterine arteries was taken and proceeded with suction evacuation, no undue bleeding per vagina noted , uterine artery embolization was deferred and proceededwith check curettage. Post procedure she was stable and no complications were encountered.

CASE 7:

A 23yearold A3 presented with complaints of heavy bleeding p/v. One month earlier she had missed abortion which was managed medically. On examination she was stable with hemoglobin of 11.8g/dl and beta hcg was 20.6mIU/mL. TVS pelvis showed retained products of conception with increased vascularity with maximum velocity of 38.3cm/s and RI was 0.5.In view of RPOC with ?AVM planned for conservative management .Consent was obtained and managed with Tablet Misoprostol and Injection Depo Provera.No further episodes of bleeding p/v and patient was discharged .

CASE 8:

A 29year old P1L1A1 presented with complaints of bleeding p/v. Four months earlier, she had spotting p/v and urine pregnancy test was positive, scan showed molar pregnancy and was treated with methotrexate and folinic acid single dose and evacuation done outside. After 3 months now she presented with bleeding p/v. On examination she was pale and hemodynamically stable with hemoglobin of 8.4g/dl, beta hcg was 37.2mIU/mL. 1unit packed cell was transfused. TVS pelvis mass of mixed echogenicity in endometrial cavity with increased vascularity? Invasive mole? Retained product of conception with arteriovenous malformation. MRI pelvis showed hemorrhagic fluid in the uterine cavity with increased vascularity suggestive of? Invasive mole with arteriovenous malformation. PET scan showed metabolically inactive soft tissue density lesion in endometrial cavity with involvement of left lateral wall of myometrium and then proceeded with Bilateral uterine artery embolization. Postoperative period was uneventful.

TABLE NO 1: Summary of all the cases

CASES	PRESENTATION	SECONDARY	SERUM Beta hcg	TVS PELVIS	MRI PELVIS	TREATMENT GIVEN
1	Heavy bleeding p/v with clots	Suction and curettage	-	+	-	Bilateral UAE
2	Heavy bleeding p/v with clots	Post LSCS	-	+	+	Bilateral UAE
3	Intermenstrual bleeding	Medical abortion	+	+	-	Conservative
4	Heavy bleeding p/v with clots	Myomectomy	-	+	-	Bilateral UAE
5	Heavy bleeding p/v	Manual removal of placenta	+	+	+	Bilateral UAE
6	Spotting p/v	Medical abortion	-	+	-	Suction evacuation followed by check curettage
7	Heavy bleeding p/v	Medical abortion	+	+	-	Conservative
8	Bleeding p/v	Molar pregnancy and evacuation	+	+	+	Bilateral UAE

III. Discussion:

The first case of AVM reported in 1926. AVM is a rare life-threatening condition and currently only fewer cases were reported in the literature. AVM can be congenital or acquired. History of dilatation and curettage, cesarean section, myomectomy, treatment for gestational trophoblastic disease are the predisposing factor in acquired arteriovenous malformation. Acquired AVM also associated with infection, gestational trophoblastic disease, gynecological malignancy, retained products of conception, diethylstilbestrol. They typically present with vaginal bleeding ranging from intermittent to profuse bleeding p/v and may become hemodynamically unstable.

In our institute Sri Ramachandra Institute of Higher Education and Research (SRMC RI/SRIHER) during the period of 2015 to 2020 8 cases were reported.

Out of 8 cases, 6 case presented with heavy bleeding p/v with or without passage of clots, 1case presented with spotting p/v on and off, 1 presented with intermenstrual bleeding. Out of 8 cases , 1 case developed uterine AVM in 3 weeks following suction evacuation and curettage, 2 cases developed uterine AVM in 1 month-1 after open myomectomy and 1 followed by medical abortion , 2 cases developed uterine AVM in 1 1/2 months- 1 post cesarean section and 1 following medical abortion , 1 case developed uterine AVM in 2 months following manual removal of placenta , 1 case developed uterine AVM in 2 1/2 months following medical abortion, 1 case developed uterine AVM in 3months followed by molar pregnancy and evacuation. Out of 8 cases, 5 cases were treated with bilateral uterine artery embolization, 2 cases were treated conservatively-1 with cyclical OCP and 1 with Injection Depo Provera, 1 case was treated with suction evacuation followed by check curettage.

Gray scale sonography findings were nonspecific AVM is diagnosed easily by TVS pelvis and color doppler which detect the presence of normal endometrium with hypoechoic area within the myometrium. However other differential diagnosis should be kept in mind such as retained products of conception(RPOC), gestational trophoblastic disease (GTD) because these cases may also present with hyper vascular appearance with turbulent flow. Beta hcg level may be useful in diagnosis. A normal myometrium signal will show a peak systolic velocity (PSV) 9 to 44 cm/ sec and resistive index (RI) 0.6 to 0.8. AVM show high peak systolic velocity and low resistive index. PSV help in management of AVM. A PSV <40cm /sec in AVM are likely to resolve spontaneously and need periodic follow up. CT or MRI can be used to better delineate the anatomy. Digital subtraction angiography (DSA) is the gold standard for diagnosis.

Management of AVM depends on the severity of the hemorrhage, fertility status, location and size of the lesion. Asymptomatic or mild AVM patient can be managed conservatively and with follow up imaging. The methylergonovine maleate have shown successful result in treating AVM. Long term uses of OCPs also shown reduction in AVM lesion. Some studies have described the uses of gonadotropin releasing hormone agonist to treat mild AVM in stable patient. Some may resolve spontaneously if left untreated. The mainstay of treatment is hysterectomy or embolization of uterine arteries.

IV. Conclusion:

Uterine arteriovenous malformation is a rare cause of vaginal bleeding. Uterine arteriovenous malformations are more likely acquired secondary to post cesarean section, dilatation and curettage, miscarriage, infection. DSA is the gold standard technique however TVS pelvis with color doppler,CT, MRI or angiogram can help in diagnosis. Embolization of uterine arteries is the mainstay of management. Hysterectomy may be performed after failure of embolization or based on fertility status. Conservative approach may be appropriate for many patientswho are asymptomatic or with mild lesion,however close clinical follow up and imaging is needed.

ABBREVATION:

AVM- Arteriovenous Malformation
TVS-TransVaginal Sonography
MRI- Magnetic Resonance Imaging
DSA- Digital Subtraction Angiography.
P/V-Per Vagina
LSCS-Lower Segment Cesarean Section
UAE-Uterine Artery Embolization
Hcg-Human Chorionic Gonadotropin
RPOC-Retained Product of Conception
GTD-Gestational Trophoblastic Disease
OCP-Oral Contraceptive Pills
PSV-Peak Systolic Velocity
RI-Resistive Index

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