ALL IS WELL THAT ENDS WELL IN MTP

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ABSTRACT

This is an important and worthwhile case to write about because it demonstrates an unusual complication of surgical termination of pregnancy captured and shown here on direct high-quality hysteroscopic video, adding evidence to the light weight of literature that recognises an association between instrumentation of the uterine cavity and acquired arteriovenous malformations (AVMs). It highlights that reserving an index of suspicion for AVM of the uterus is very important, because through treatment such women can be relieved of the devastating physical, psychological, emotional and material impacts on their quality of life that may otherwise go undiagnosed for longer. It also exhibits the use of arterial embolisation in a young lady and follow-up of such patients may impart information about the effects of such treatments on future fertility.

KEYWORDS

Arterio-Venous Malformations, Pregnancy, Colour Doppler, Hysterectomy, Ultrasound.

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INTRODUCTION

Uterine Arteriovenous Malformations (UAVM) are abnormal connections between uterine arteries and veins.[1] UAVM can be congenital or acquired/traumatic. The clinical presentation of UAVM are variable–the classical clinical feature is intermittent, heavy vaginal bleeding which can be life threatening. UAVM can also be fairly asymptomatic.[2] The incorporation of necrotic villi in the venous sinuses of scar tissue is thought to cause acquired UAVM.[3] An acquired UAVM almost always occurs after a uterine trauma, such as dilation and curettage or caesarean section but can also be associated with normal vaginal birth or malignancy.[4] UAVM may also be congenital, occurring rarely in the uterus and usually invading surrounding structures.[5] Congenital lesions classically present with severe menorrhagia, unresponsive to conventional therapy.

UAVM can be diagnosed with grey scale and colour Doppler ultrasonography, MRI and CT angiography; radiographic angiography remains the gold standard.[6] There can be considerable overlap in the presentation and appearance of UAVM and other pathologies related to pregnancy and uterine trauma, such as subinvolution of the placental bed (SPB).[3] retained products of conception (RPOC) and gestational trophoblastic disease (GTD).[7] UAVM can also occur concurrently with or post resolution of a spontaneous incomplete miscarriage as well as trophoblastic disease.[7] Accurate diagnosis is critical, as the treatment for RPOC and GTD (generally dilation and curettage) is contraindicated in UAVM, as there is the risk of causing massive haemorrhage and death.[6] UAVM are characterised by a negative Beta Human Chorionic Gonadotrophin (BHCG) value, which is usually weakly elevated in RPOC and grossly elevated with GTD.[7] RPOC, GTD, UAVM and SPB can present with vaginal blood loss or haemorrhage.[1] with RPOC and SPB exclusively occurring post pregnancy.[2] GTD and UAVM can be discovered during or post pregnancy.[3] UAVM appear as multiple anechoic spaces in the myometrium (Figure 1) with a typical spectral Doppler appearance of turbulent flow (Figure 2), low resistance, high velocity. A colour mosaic pattern is seen with colour Doppler imaging.[1] A key feature is that the colour Doppler images are vastly more informative than the grey scale imaging. SPB appear similar to UAVM.[3] RPOC are seen on ultrasound as a focal echogenic mass in the endometrium with low resistance flow.[7] while GTD has anechoic spaces and an absence of a fetus.[1]

Standard of treatment for UAVM was previously hysterectomy, current fertility preserving techniques, mainly Uterine Artery Embolisation (UAE) are becoming more widely accepted.[1] The uterine artery is embolised with microparticles, coils, glue or alcohol, while collateral supply maintains uterine perfusion thus preserving fertility.[6,7]

There have been several case studies of pregnancy after embolisation for UAVM in the literature, but only a few reviews and no classification scheme for best diagnostic methods and outcome incorporating clinical presentation and past medical history.

DISCUSSION

In our study, three cases were studied. One case-a case of G4P2L2A1 with 6 weeks of amenorrhoea with failed medical management of abortion who underwent MVA at a Private Hospital. During MVA, she started bleeding profusely. So procedure was stopped and she was revived. Her USG showed AV fistula at right cornual end of uterus. After 6 weeks without any provocation she started bleeding heavily (Inter-menstrual bleeding) and was admitted, underwent hysterectomy immediately.

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In Second Case

A case of G3 P2 L1 D1 with 6 weeks with AV fistula in uterine vessels. She had failed medical management of abortion. She had completed her family, hence taken for total abdominal hysterectomy as embolization facilities are not available in District place like Ballari.

In Third Case

A case of G3 P1 L2 A1 with 7 weeks of gestation with previous LSCS with Medical Management of Abortion taken 3 weeks back, presented with menorrhagia. Serial monitoring showed that she had AV fistula in the right cornual end. Five months later USG and Colour Doppler showed that it was an intramural fibroid, but on laparotomy a huge hematoma on the cornual end was detected. On the histopathological examination, it revealed tropoblastic tissue in that site with AV malformation.

Uterine arteriovenous malformations are a rare cause of abnormal vaginal bleeding, most commonly seen in women who have been pregnant and experienced some type of uterine trauma such as D and C.[1] UAVM can be difficult to diagnose, not only because they are rare, but because they may present similarly to or in conjunction with other pregnancy related pathologies, such as sub-involution of the placental bed, retained products of conception and gestational trophoblastic disease.[5] It is important to make an accurate diagnosis of UAVM, as treatment for RPOC and GTD may include D and C, which is contraindicated in UAVM.

The gold standard of diagnosis of UAVM is angiography, although colour Doppler ultrasound is now the most widely used first line of investigation. Angiography has the benefit of allowing treatment (In the form of embolisation) at the time of diagnosis.

It has been proposed by Timmerman, et al.[3] that SPB and UAVM constitute a spectrum of disease rather than distinct pathologies Timmerman.[3] suggests the term uterine vascular malformations for those diagnosed by ultrasound with arteriovenous malformation describing only those lesions seen on angiography to have the appropriate characteristics (Early venous filling). Aside from angiography, the clinical picture and ultrasonographic features of uterine vascular malformations are very similar: hypervascular areas within the myometrium showing turbulent, low resistance flow often associated with varying degrees of vaginal bleeding.[3]

An AVM is an abnormal connection between an artery and a vein that bypasses the capillary system. Under normal circumstances, the venous system is subject to a low-pressure, monophasic blood flow. However, in an AVM, high-pressure pulsating arterial blood flows directly into the venous system. Due to the subsequent haemodynamic changes the veins undergo a process of arterialisation. Despite the attempted vessel wall adaptation, these malformations are vulnerable to rupture and haemorrhage. Bleeding from a uterine AVM is compounded by the dynamic physiological alteration that occurs in the endometrium every month.

Rapid vascularisation and shedding of this glandular tissue, which is controlled by the hormones of the female reproductive axis make uterine AVMs that abut the endometrium particularly prone to bleeding.

AVM can be classified as either congenital or acquired. Congenital AVMs are particularly rare and generally consist of multiple small vascular connections thought to be a result of major local disturbance to the angiogenic process during development.[1] These usually become apparent at menarche with the onset of menses when they become more prone to bleeding. Acquired AVMs, however, are generally morphologically different. They are typically a single large connection between an artery and a vein.

The aetiology of acquired uterine AVMs is poorly understood. The majority are associated with some form of iatrogenic uterine trauma, such as dilatation and curettage or myomectomy, although Caesarean section and even normal vaginal delivery have proven traumatic enough to lead to the formation of an AVM. Rarely, AVMs have also been associated with infection, trophoblastic disease and malignancies of the uterus and cervix.

Uterine AVM is a rare, but potentially life-threatening condition. In the past, they have usually presented with severe per vaginal bleeding that is refractory to usual methods of treatment. The incidence of uterine AVM is unknown, though considered rare.

Cases similar to this one have been reported. Molvi et al.[2] reported two cases of patients with abnormal uterine bleeding after abortions followed by dilatation and curettage. In both instances, Doppler ultrasound yielded the diagnosis. Both patients were treated by transcatheter embolisation and went on to have normal menstrual cycles. One of these patients went on to have an uncomplicated pregnancy 2.5 years after embolisation. This triggers the next question: how safe is uterine artery embolisation?

Wang et al.[3] recently evaluated the efficacy of embolotherapy in patients with iatrogenic traumatic uterine AVMs in 42 patients. All the patients underwent transcatheter embolisations of both uterine arteries and they measured two main complications: haemorrhage and outcome of subsequent pregnancies. Bleeding from the malformations was successfully controlled in 83% of the patients after the first embolisation procedure. Thirteen patients became pregnant post-embolisation and eight of these had uneventful pregnancies carried to term. So perhaps the final important question to consider is whether surgical intervention is always necessary to treat these lesions.

Interestingly, despite many reported cases presenting with horrific haemorrhage from a friable nidus of abnormal blood vessels, some uterine AVMs are actually asymptomatic. Degani et al. found that 9 of 12 patients, diagnosed with uterine AVMs after abortion procedures were indeed asymptomatic and were managed expectantly for 4–10 weeks without future complications.[4]

It seems that there is a broad spectrum of presentation severity associated with AVMs of the uterus from asymptomatic patients to those like our patient who suffered almost catastrophic uterine haemorrhage. The diagnosis of uterine AVM is scarcely expected. In many cases of AVM, it has become apparent that the initial investigation to yield an abnormal finding is Doppler ultrasound scan of the pelvis showing vascular flow abnormalities.[6] The key finding is early filling of the dilated veins, thus providing evidence of arteriovenous shunting.

Before the 1980s, uterine AVMs were managed predominantly by hysterectomy. Now, there is growing

evidence that transcatheter uterine artery embolisation is a safe and effective intervention that returns a favourable chance of successful pregnancy.

CONCLUSION

In conclusion, UAVM are rare. Potentially, life threatening causes of vaginal bleeding that can be effectively treated with UAE, but some may also be safely managed conservatively. Ultrasound is a safe and reliable method for suggesting an UAVM, although angiography remains the gold standard for definitive diagnosis. Every treating obstetrician should have high degree of suspicion of the existence of UAVM and be ready to handle such emergencies and save lives.

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