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Enhanced myometrial vascularity secondary to retained pregnancy tissue: time has come to stop misusing the term arterio-venous malformation!

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INTRODUCTION

Historically the term “Arterio-Venous Malformation” (AVM) has been used to describe striking myometrial vascularity identified by Doppler after a recent intrauterine pregnancy. In 2004 Van Schoubroeck et al. introduced the term “Enhanced Myometrial Vascularity” (EMV) to describe this sonographic finding when it is associated with retained pregnancy tissue. AVM and EMV continue to be used interchangeably¹⁻⁴ and has led to much confusion as to the pathophysiology of these two different entities and, more importantly, their management⁴.

Arterio-venous Malformations

Congenital arterio-venous malformations (AVM) are a rare embryological maldevelopment and are not placenta related. These aberrant connections between arteries and veins may occur anywhere in the body including the brain, and do not disappear spontaneously. Treatment is either by surgery or interventional radiology with selective embolization⁵. The term EMV is to be used only in the first weeks postnatally or following a non-viable normally sited intrauterine pregnancy. Using the same term (AVM) for a pregnancy related physiological condition and a congenital vascular condition must be avoided. The former is a natural manifestation of the pathophysiology of retained pregnancy tissue, the latter is a persistent malformation⁶⁻⁸. This is not a semantic discussion^{4,9}. While selective embolization and/or vascular surgery are key to the management of arterio-venous malformations⁵, embolization is rarely indicated in pregnancy related enhanced myometrial vascularity. Serious complications reported after selective uterine artery embolization include thrombo-embolic events, vesico-vaginal and utero-enteric fistulae, buttock and labial necrosis, and septic shock. Obstetrical and fertility related consequences include delayed subsequent pregnancy, abnormal placentation and subfertility due to intrauterine synechiae or decreased ovarian reserve¹⁰⁻¹³. Although these complications may be uncommon, embolization should only be considered in life-threatening indications.

Enhanced Myometrial Vascularity

The reported prevalence of EMV ranges between 1.5% and 6.3%^{6,14}. Progressive upstream remodeling of the spiral and radial arteries starts in early first trimester¹⁵. At the end of the first trimester, the dilated radial and adapted spiral arteries within the myometrium allow for an adequate perfusion of the intervillous spaces in the maternal part of the placenta. In early pregnancy failure an increased flow in the intervillous space has been reported¹⁶. Towards the end of pregnancy, about 700 ml of maternal blood flows each minute to the placenta. Immediately after delivery of the placenta, myoepithelial and myometrial contractions constrict the myometrial vessels, and excessive maternal blood loss is avoided. These vessels are seen on gray scale ultrasonography as anechoic, irregular, tubular structures in the myometrium. Doppler imaging may show unusually high flow velocities, while the flow in the radial and spiral vessels remote from the implantation site is not enhanced (Figure 1 and 2).

According to the laws of hemodynamics, high velocity circulation within the myometrium can only be possible in the presence of a direct connection between the high-pressure arterial system and the low-pressure venous system, as is seen in the placental bed (Figure 3). Once the retained pregnancy tissue is expelled completely - spontaneously or surgically removed - the high velocity vessels disappear as in the normal third stage of labor^{8,17}. Enhanced Myometrial Vascularity beneath retained tissue of pregnancy is therefore part of a normal physiological process, and so is its disappearance once the retained pregnancy tissue has been removed or has passed^{6,8,17}.

In cases of EMV, retained trophoblastic tissue is the most likely explanation of this high velocity flow, but the amount of residual tissue may sometimes be limited. A focal yellowish flat area undetectable on ultrasonography may be seen during hysteroscopy.

Management of EMV

The management outlined in this paper is not applicable in increased myometrial vascularity seen in gestational trophoblastic disease, caesarean scar pregnancy or placenta accreta

spectrum, nor in non-pregnancy related conditions associated with uterine hypervascularity such as uterine malignancy, posttraumatic arterio-venous shunts following intrauterine surgery or the extremely rare case of congenital arterio-venous malformation¹⁸. A 'posttraumatic' arterio-venous shunt is a direct shunt between an artery and a vein, secondary to trauma (Figure 4). It has been described after hysteroscopic surgery, accidental uterine wall perforation or caesarean section at the uterine incision site. Here the placental bed is not involved, and this entity should be distinguished from pregnancy related EMV.

In the absence of heavy bleeding, EMV may be managed expectantly. The EMV will disappear as the retained pregnancy tissue is expelled or resorbed. Although prompt EMV disappearance within minutes during the surgical removal of retained pregnancy tissue has been described by Van den Bosch et al, surgical management is only considered in case of heavy or persistent bleeding and if pregnancy wish is deemed hindered by persistent retained tissue^{14,17}. In cases of extensive EMV the woman should be informed that torrential bleeding may occur, necessitating emergency admission. Should this occur, surgical removal of the retained tissue is indicated (Figure 5). The earlier concept that a high peak systolic flow correlates with the risk of heavy blood loss, is not supported by the literature and should not be used to guide management^{6,17,19}.

Ultrasonography is essential for pre-operative mapping and intra-operative guidance²⁰. Blind tissue removal may lead to incomplete and lengthy procedures, prolonged heavy bleeding, and unnecessary scarring of the endometrial-myometrial junction associated with Asherman syndrome or perforation²¹. The exact position of the retained tissue within the uterine cavity can be deduced from the EMV as it underlies the retained tissue. In the absence of heavy bleeding, operative hysteroscopy with cold loop resection or selective morcellation is often successful. If brisk bleeding obscures the hysteroscopic image, a swift switchover to ultrasound guided tissue removal, either by suction aspiration, ovoid swab forceps removal or with a blunt curette, is required. Both transabdominal and transrectal ultrasound guidance may be used, depending on the patient.

As soon as the retained tissue is removed completely, the EMV disappears and the bleeding usually stops ¹⁷. If bleeding persists, incomplete removal of the retained tissue should be suspected. The spontaneous filling of the cavity with fresh blood, as this is a negative contrast agent, optimizes the detection of residual tissue by ultrasound. Thus, aspiration should be temporarily stopped whilst the tissue is located.

Persistent bleeding in the absence of visible retained pregnancy tissue requires standard measures to optimize uterine contraction and hemostasis, including emptying the bladder, bimanual uterine massage, administration of uterotonic drugs and tranexamic acid.

If bleeding still remains problematic, an intra-uterine balloon catheter is the next step. The balloon should be inserted under ultrasound guidance up to the level of the active bleeding site and then inflated. The balloon catheter is left in situ for 2 to 24 hours, depending on the clinical situation.

If life-threatening bleeding persists despite all the above mentioned, selective embolization should then be considered, to avoid a lifesaving hysterectomy. In our experience, this is a rare occurrence.

Conclusion

To conclude, the presence of focally enhanced high velocity/low resistance vessels in the myometrium following miscarriage should be termed “enhanced myometrial vascularity” (EMV). EMV is a reliable sign for the presence of retained pregnancy tissue and can be managed expectantly. Complete resorption, expulsion or surgical removal of the retained tissue will lead to rapid resolution of the EMV, irrespective of the initial peak systolic velocity. If treatment is indicated, the retained tissue should be removed. The cornerstone of a safe surgical removal of the retained tissue is accurate preoperative mapping of the EMV and a procedure performed under hysteroscopic or ultrasound guidance in a center that is experienced in dealing with bleeding complications on the rare occasions that they arise.

Key Points

1. EMV represents a natural manifestation of the pathophysiology of retained pregnancy tissue
2. After a miscarriage or delivery, EMV is an indication of retained pregnancy tissue
3. If treatment is indicated, it should focus on the removal of the retained tissue, under ultrasound or hysteroscopy guidance
4. The risk of excessive bleeding during treatment is limited. Persistent (heavy) bleeding can be managed with uterotonics and, if necessary, an intra-uterine balloon.
5. Embolization is not advised as first-line or prophylactic treatment and should only be considered in the rare case of persistent life-threatening bleeding.

FIGURE LEGENDS

Figure 1: Ultrasound images (gray scale and power Doppler) of retained pregnancy tissue (*) with underlying enhanced myometrial vascularity (EMV): notice the tortuous dilated hypo-echogenic vessels on gray scale, corresponding to the enhanced myometrial vessels visible on power Doppler.

Figure 2: Enhanced myometrial vascularity (EMV) underlying retained pregnancy tissue (*).

Figure 3: Normal vascularization of the placental bed.

Figure 4: Posttraumatic AV-shunt (NOT pregnancy related): an iatrogenic cavity within the myometrium (*) into which an artery flows and a vein starts.

Figure 5: Management of enhanced myometrial vascularity associated with retained pregnancy tissue. Hysteroscopic management is considered in case of persistent bleeding and if pregnancy wish is deemed hindered by persistent retained tissue. EMV: enhanced myometrial vascularity; IU: intrauterine

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