Pregnancy Following Embolisation of Uterine Arteriovenous Malformation – A Case Report

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Abstract

***Introduction**: Pregnancy following successful embolisation of a uterine arteriovenous malformation (AVM) is rare. Hypovascularity of treated areas affecting placentation and fetal growth has been postulated to be the cause for adverse pregnancy outcomes. **Clinical Picture**: A 37-year-old multiparous lady presented with anaemia from repeated heavy vaginal bleeding from an iatrogenic uterine AVM. This was diagnosed with power Doppler ultrasonography and embolised after pelvis angiography with hystoacryl and lipiodol. Following this she had a spontaneous pregnancy with a normal outcome. **Conclusion**: There are no distinguishing clinical features of a uterine AVM. An index of suspicion and power Doppler sonography help in reaching a diagnosis. Dilatation and curettage should be avoided as this can precipitate torrential vaginal bleeding.*

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***Key words:** Power Doppler, Super selective embolisation, Vaginal bleeding*

Introduction

Arteriovenous malformations (AVMs) of the uterus are rare but potentially life-threatening lesions. The common presentation is vaginal bleeding and, if not diagnosed correctly, can result in torrential bleeding, especially following dilatation and curettage. They may be congenital, but can be secondary to malignancies or trauma to the uterus. In the past, most have been managed surgically by hysterectomy with or without hypogastric artery ligation. The conservative management of such lesions by embolisation conserves reproductive function. We report a case of a successful pregnancy after transarterial embolisation of an AVM in a 37-year-old lady.

Case Report

A 37-year-old Indonesian lady, para 2, was referred for further management of menometrorrhagia. She had 2 previous deliveries by Caesarean section followed by a first trimester miscarriage in April 2001 for which a curettage was done. In May and June 2001, she reported normal menses. However, from July 2001, she began to experience menometrorrhagia. A gynaecological and ultrasound examination of the pelvis showed what appeared to be blood clots within the endometrial cavity. She was treated with tranexamic acid with symptomatic improvement. In September, however, she relapsed with prolonged heavy vaginal bleeding despite compliance with tranexamic acid therapy. A repeat ultrasound examination of the pelvis revealed a hypoechoic mass within the uterine cavity. Her haemoglobin level then was 7.9 g/dL and she had symptoms of anaemia. She underwent a dilatation and curettage (D&C) but the procedure was abandoned halfway due to profuse vaginal bleeding. Histology of the curettings was non-diagnostic. A computed tomography (CT) scan of the abdomen and pelvis was arranged and revealed an enlarged uterus with blood clots. Her serum beta hCG was negative. She was given blood transfusion and her post transfusion haemoglobin level was 9.4 g/dL. She was subsequently referred to our centre for further management on 8 November 2001.

Upon arrival, she had an ultrasound examination of her uterus which showed a bulky uterus with a thick-walled cystic area measuring 2.6 x 2.0 x 2.3 cm in the lower right uterine wall, adjacent to the endometrium. Power Doppler showed intracavity blood flow with arterial and venous waveforms. A clinical diagnosis of a uterine AVM was made and the patient was prepared for a therapeutic embolisation of the AVM.

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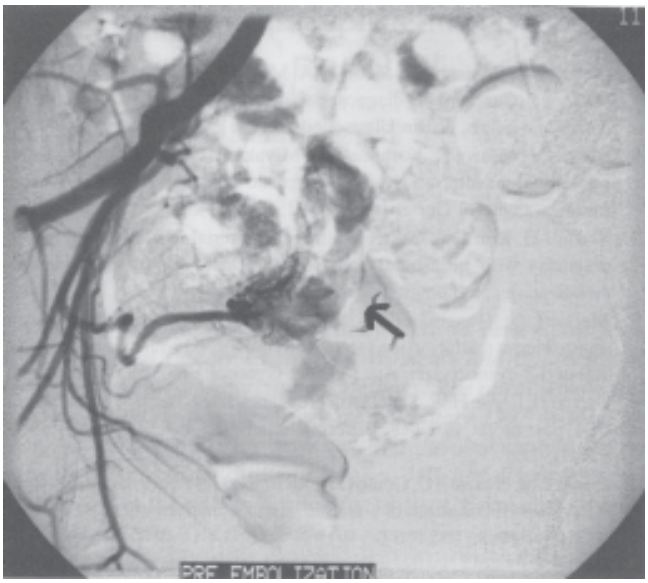

Fig. 1. Right internal iliac angiogram demonstrating hydrophied vessels arising from the tip of the right uterine artery with early arteriovenous shunting indicative of an AVM (arrow).

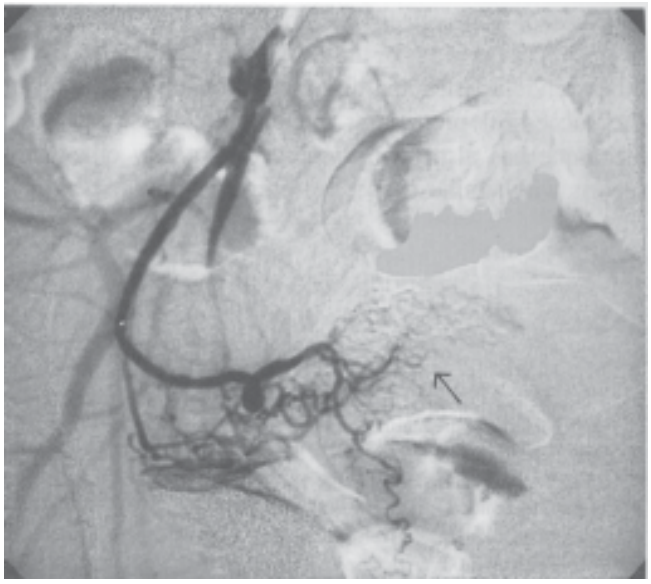

Fig. 2. Selective right uterine angiogram after superselective embolisation shows successful obliteration of the AVM.

She underwent an angiogram of the uterine vessels on 9 November 2001 and this confirmed the presence of a right uterine AVM (Fig. 1). The lesion was successfully embolised with a 0.5 mL mixture of histoacryl and lipiodol (1:1) via a microcatheter placed superselectively at the feeding artery. With the histoacryl and lipiodol mixture, it was possible to completely occlude the nidus of the AVM (Fig. 2).

After the procedure, she recovered uneventfully. There were no further episodes of abnormal vaginal bleeding and she resumed her regular menstrual periods the following month. Nine months after the procedure, the patient conceived spontaneously. An ultrasound scan done at 16 weeks amenorrhoea showed a viable fetus corresponding to dates and no abnormal vessels were noted at the site of the previous AVM. Her antenatal progress had since been uneventful. She went on to term and delivered a healthy baby girl with a birth weight of 3.5 kg at 39 weeks' gestation via an elective lower segment Caesarean section for 2 previous Caesarean sections. She did not experience any postoperative complications. She is currently well at 3 months postpartum.

Conclusion

Clinically significant vascular malformations of the uterus are uncommon. There are less than 100 cases reported in the literature worldwide.[1-3] Although they can be congenital or acquired, the majority of reported cases are acquired and occur after uterine curettage. Other predisposing conditions include gestational trophoblastic disease,[1] endometriosis, uterine fibroids and habitual abortion.[2] The commonest clinical presentation is abnormal vaginal bleeding which can be profuse. There is often a lack of other physical signs and clinical diagnosis is often difficult and delayed. In the past, a diagnosis was usually made only at laparotomy or after histologic examination of the hysterectomy specimen. Presently, diagnosis is often suggested by ultrasound (presence of AVM is suggested by thickening of the uterine wall with numerous cystic lesions on ultrasound) and Doppler studies[4-7] but the gold standard of diagnosis is pelvic angiography.[2] Magnetic resonance (MR) imaging has been shown to be of value and may be the modality of investigation in the future. Clinicians should exercise a high index of suspicion in patients with predisposing condition(s) presenting with abnormal vaginal bleeding, especially if profuse and unresponsive to medical therapy, e.g., menometrorrhagia. At a minimum, an ultrasound scan should be performed before embarking on further endometrial evaluation. Curettage is non-therapeutic and often aggravates bleeding as encountered in this case report.

Embolisation of uterine AVM using various embolic materials has been employed as a definitive therapeutic modality to preserve reproductive capability.[1,2,8] The latter is important as many such lesions occur in young women. Successful treatment has been previously reported. Nevertheless, pregnancy following embolisation of uterine vascular malformations is rare.[2,3,8-10] Poor pregnancy outcome, e.g., increased fetal wastage and stillbirth following treatment, is common. Hypovascularity of treated areas affecting placentation and fetal growth has been postulated to be the cause for adverse pregnancy outcomes.

The use of superselective embolisation, with complete occlusion of the nidus of the AVM and preservation of the other pelvic arteries and branches, probably accounts for the successful pregnancy outcome in this case. Such technique has also been employed in a few other case reports with similar successful pregnancy outcome.[1,10]

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