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Case Report Open Access

Transcatheter Percutaneous Embolotherapy of Uterine Arteriovenous Malformations: A Report of 2 Cases

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Abstract

Purpose: Uterine AVMs are difficult to treat using conventional surgical techniques due to the high patient morbidity secondary to complex vascularity. Trans catheter embolotherapy has become a suitable option in the setting of large pelvic arteriovenous malformations as a significantly safer alternative to surgery offering great patient outcomes and shorter recovery times. We report two cases of large uterine arteriovenous malformations successfully embolized with N-butyl cyanoacrylate with near complete remission of patient symptoms.

Case Reports: Two patients are presented one premenopausal and one postmenopausal both of whom had large uterine arteriovenous malformations which were resulting in significant bleeding.

Results: The first patient had a large AVM which was treated solely through transcatheter methods with N-butyl cyanoacrylate (n-BCA) glue embolotherapy of the right ovarian artery and bilateral uterine arteries. The second patient had a markedly complex AVM draining into a hypertrophied left gonadal vein which was treated with a combination of transarterial and transvenous catheter embolotherapy with coils and n-BCA as well as direct percutaneous puncture with STS foam sclerotherapy of a dominant nidus. Both had good outcomes with the second patient having a 4 year follow-up arteriogram revealing no further filling of the AVM.

Conclusion: A combination transcatheter and percutaneous sclerotherapy approach with embolization of the nidus, outflow vein and arterial inflow can eradicate uterine AVMs and dramatically improve patient outcomes, preserving patient fertility with limited morbidity and high success rates. In general, particulate embolization should be avoided in large AVMs due to the high risk for AV-shunting.

Introduction

Uterine arteriovenous malformations are a rare cause of menorrhagia in premenopausal and a rare cause of heavy bleeding in postmenopausal women. They can be notoriously difficult to treat by conventional surgical techniques due to the marked vascularity and high risk for life-threatening hemorrhage. Transcatheter embolotherapy has become a suitable option in the setting of large pelvic arteriovenous malformations as a significantly safer alternative to surgery offering great patient outcomes and shorter recovery times. We report two cases of large uterine arteriovenous malformations successfully embolized with N-butyl cyanoacrylate (n-BCA) with near complete remission of patient symptoms.

Case Reports

Case 1

A 25 year old G2P0 presented with a history of menorrhagia and acute anemia requiring two transfusions in the past four years and an abnormal transvaginal pelvic ultrasound. She had a history significant for molar pregnancy 5 years prior for which she had a dilatation and curettage and then chemotherapy but was then lost to follow-up. She returned 4 years later and reported irregular menses in the year following her D&C with unpredictability and increased duration of her periods consistent with menometrorrhagia. She specifically reported heavy bleeding lasting 5 to 9 days and occasionally having to change pads up to 3 times per hour during the heaviest times as well as soaking through her clothing preventing her from going to work or school.

Ultrasound showed findings suggestive of uterine AVM (Figure 1a-1c).

She was taken for hysteroscopy which revealed an anteverted uterus with two large pulsating masses involving the anterior uterine wall consistent with uterine arteriovenous malformations (Figure 2a and 2b).

Approximately one month later she was referred for pelvic MRI which revealed a 6 x 5 cm arteriovenous malformation involving the anterior uterine wall (Figure 3a-3d).

Approximately two months after hysteroscopy she was referred to us in interventional radiology for possible embolization. A diagnostic arteriogram was performed revealing a very large uterine arteriovenous malformation fed by both uterine arteries and draining via the gonadal veins. The uterine arteries were selected using conventional catheter techniques and embolized with N-butyl cyanoacrylate. Notably after embolization of the right uterine artery, the distal right ovarian artery was noted to be filling retrograde. Therefore, aortography was performed revealing a hypertrophied right ovarian artery also feeding portions of the AVM. This was then selected and embolized with n-BCA as well. Particles were not utilized due to the high risk for AV-shunting and the greater viscosity of n-BCA offered safer rapid therapy and complete embolization of the AVM.

The patient was admitted overnight for post-operative pain control and discharged the following day. She followed up 2 months later noting dramatic changes and that her periods had normalized and she no longer had problems with heavy bleeding.

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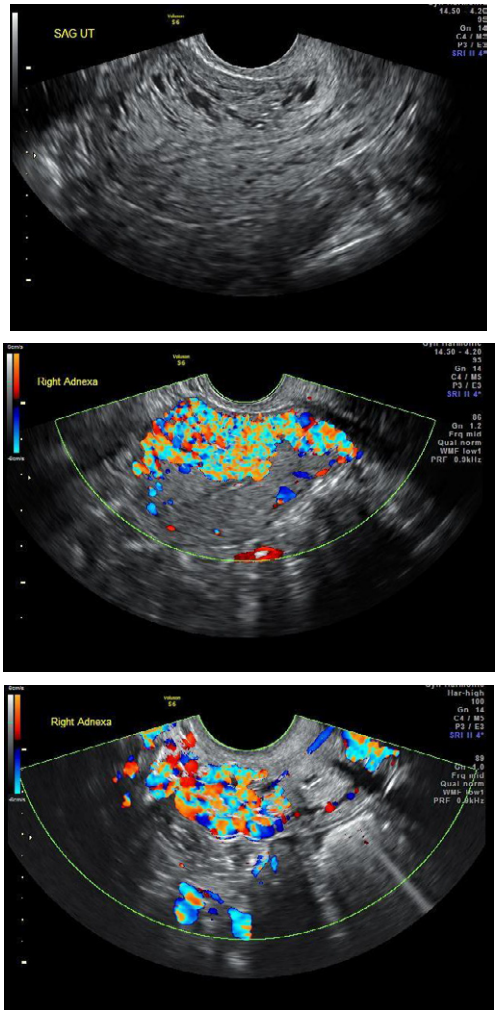


Figure 1: (a) Real-time grayscale ultrasound demonstrates multiple hypoechoic areas within the anterior uterine wall. (b and c) Color Doppler interrogation demonstrates filling in with color flow consistent with a high flow arteriovenous malformation.

Case 2

A 62 year old postmenopausal female presented with heavy vaginal bleeding occurring sporadically. She reported intermittent bouts of large volume vaginal bleeding which was lifestyle-limiting in nature. She had a full obstetric workup including endometrial biopsy which revealed no evidence for endometrial carcinoma.

Pelvic MRI was performed which revealed a large 11 x 11 cm AVM involving the majority of the uterus as well as a fundal fibroid (Figure 4a-4d).

She was referred to interventional radiology for possible embolization. Initial aortography and pelvic arteriography revealed hypertrophied bilateral uterine arteries and ovarian arteries feeding a large uterine AVM with draining into an enlarged left gonadal vein (Figure 5a-5c).

It was decided initially that given the large size of the AVM it would need to be treated in a staged fashion. Given the large amount of AV-shunting, particles were not felt to be a suitable option for treatment. Initially multiple large microcoils were packed into the AVM to fill the dominant niduses (Figure 5d and 5e).

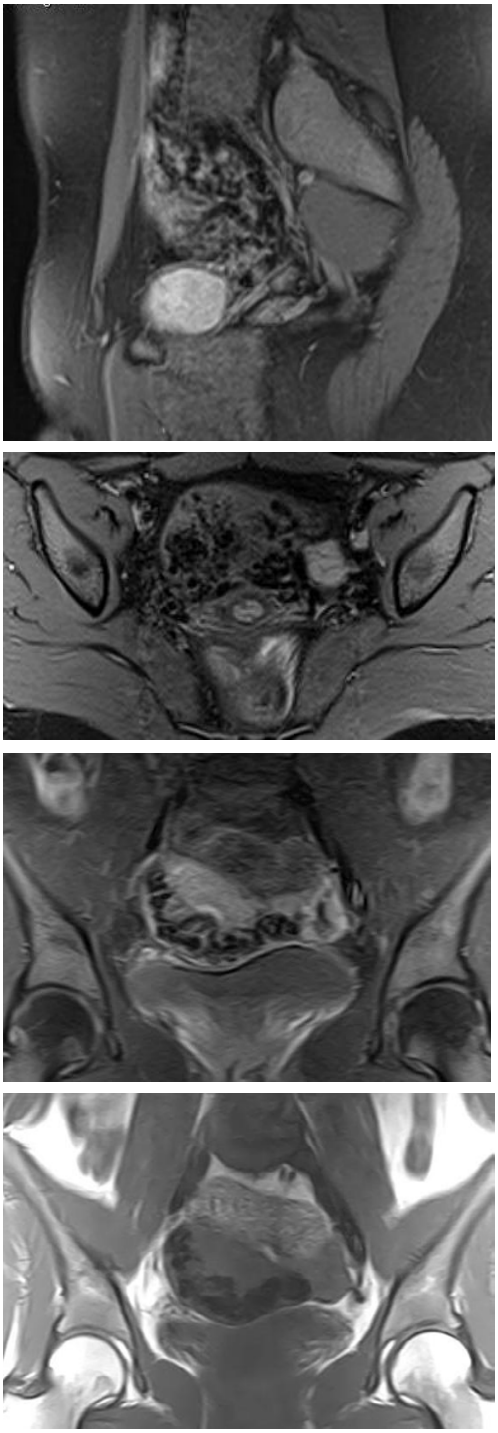


Figure 2: (a and b) Hysteroscopic evaluation demonstrated large pulsating masses along the anterior uterine wall consistent with uterine arteriovenous malformations.

N-butylcyanoacrylate glue was then utilized to slow down the flow through the AVM further (Figure 5f-5h).

Post-embolization arteriography revealed there was still substantial flow through the AVM. It was elected to stop further embolization at this session and bring the patient back at a later date for additional embolization of the AVM.

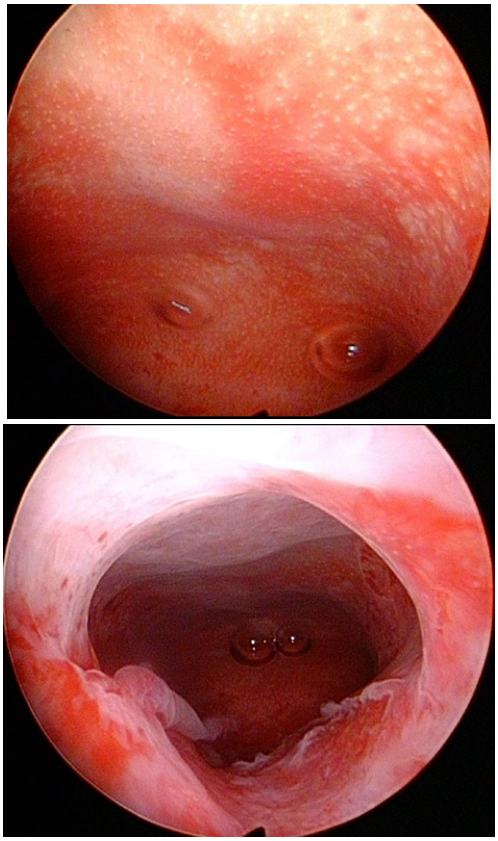


Figure 3: (a-d) Sagittal, axial, and coronal MRI demonstrates T2 hypointense flow voids which demonstrate serpentine enhancement consistent with vascular channels and a high flow AVM. Several large niduses are noted.

Approximately 1 month later the patient was brought back. Pelvic arteriography was performed revealing substantially decreased flow through the AVM, however still notable flow is present (Figure 6a and 6b).

The ovarian arteries and gonadal veins were also noted to be hypertrophied. Right common femoral vein accessed was obtained and via the left renal vein, the left gonadal vein was selected. A catheter was advanced through the gonadal vein down into the uterine AVM (Figure 6c).

Additional N-butyl cyanoacrylate glue embolization for permanent venous outflow occlusion of the AVM was then performed. This was combined with direct percutaneous access of the AVM nidus under fluoroscopic guidance with STS/n-BCA sclerotherapy of the remainder of the nidus after it was confirmed with contrast injection that there was no further filling of the embolized draining gonadal vein. Post-embolization arteriography revealed no further rapid filling of the uterine arteriovenous malformation and essentially near complete embolization (Figure 6d and 6e).

The patient was admitted for post-operative pain control and discharged the following day. She was followed for approximately 1 year noting no further bleeding episodes and then she was lost to follow-up. However, she returned 4 years later presenting with mild pelvic pain and further arteriography was requested to ensure that the AVM had not returned. Repeat pelvic arteriography revealed no residual filling of the AVM and complete embolization (Figure 7a and 7b).

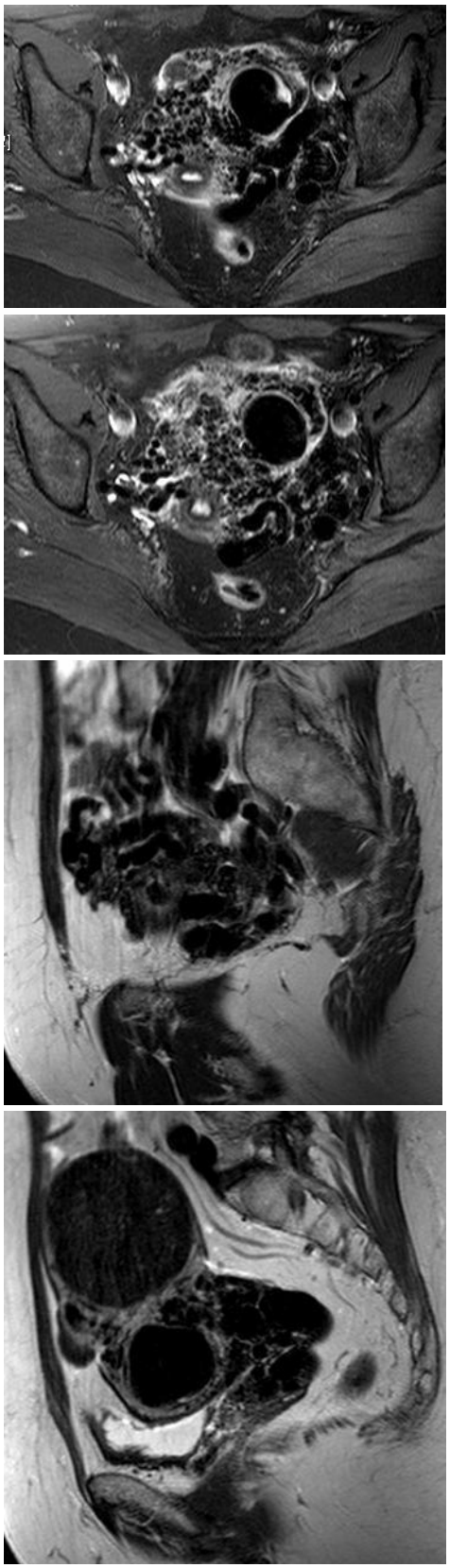


Figure 4: (a-d) MRI images demonstrate a very large 11 x 11 cm uterine AVM with numerous T2 hypointense flow voids consistent with dilated vascular channels.

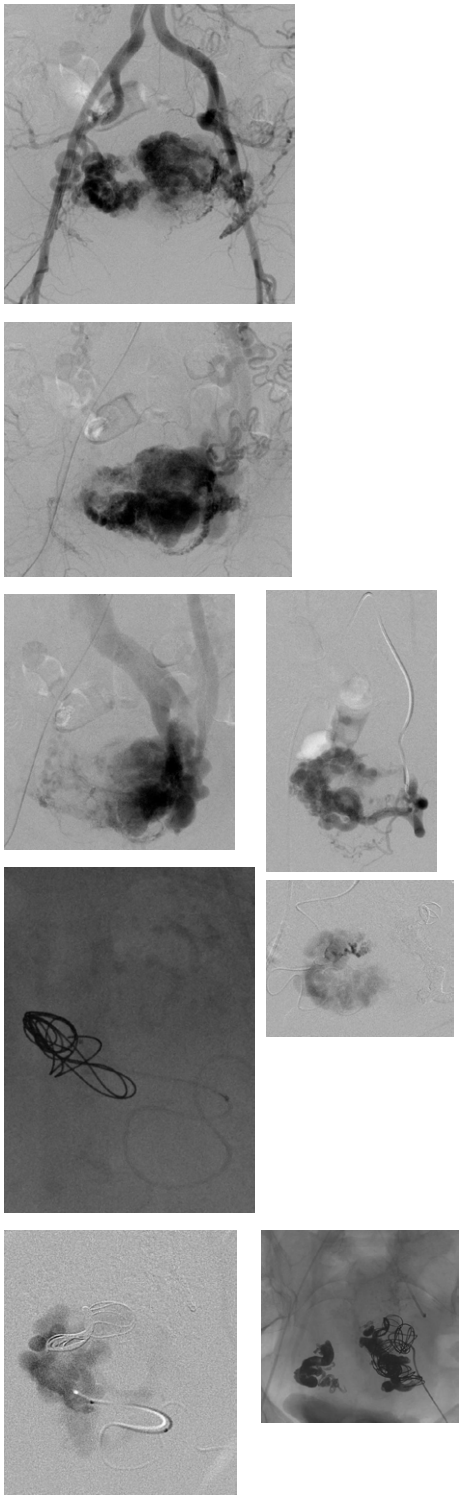
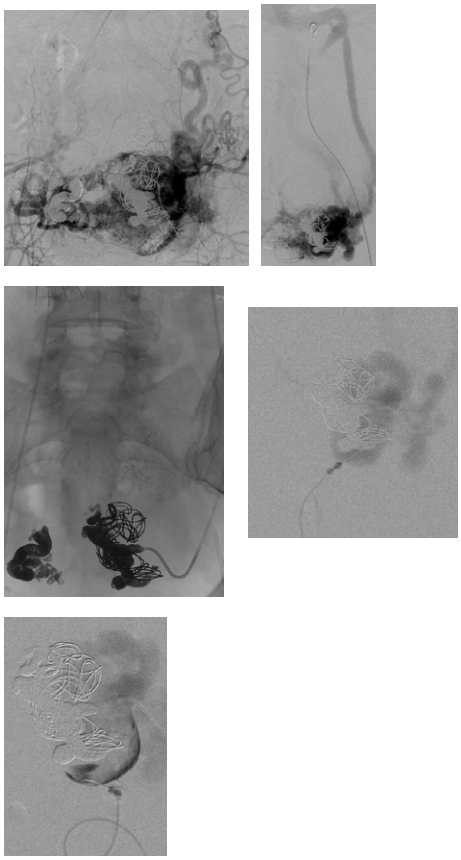


Figure 5: (a-h) Initial images, Figures 5a-c demonstrated pre-treatment planning pelvic aortography and demonstrate very large dilated vascular spaces with large AVM nidus and drainage to a dilated left gonadal vein. Figures 5d and 5e demonstrate subselection of the uterine arteries with a combination 5 French diagnostic catheter and microcatheter with large coil embolization to fill the nidus. Subsequently due to the high flow and the need for a very viscous embolic agent, N-butyl cyanoacrylate glue embolization was then performed demonstrated in Figures 5f-h. n-BCA has been mixed with lipiodol and demonstrates adequate staining and filling of the abnormal vascular spaces with reduction in flow through the AVM.



Figures 6: (a-e) Initial images demonstrate pelvic arteriography 1 month after the initial embolization. There is still obvious residual flow identified within niduses and hypertrophy of a large draining gonadal vein. Retrograde access into the gonadal vein by catheterizing the right femoral vein and placing a catheter into the renal vein and down into the gonadal vein was then performed as demonstrated in Figure 6c. Additional glue embolization was performed. Percutaneous access of additional enlarged nidal channels was then performed as seen in Figure 6d-e with injection of foam sclerosant, sodium tetradecyl sulfate (STS).

Discussion

Uterine AVMs are rare and notoriously difficult to treat. Given the popularity of uterine fibroid embolization, nearly all interventional radiologists are comfortable with selecting the uterine arteries. It is important to perform dedicated selective power-injected uterine arteriography to ensure incidental AVMs are detected during fibroid embolization. One case report in the IR literature has reported a patient fatality from particulate embolization of an unrecognized uterine AV fistula during fibroid embolization [1]. Smaller AVMs discovered incidentally and not causing significant symptomatology can often be watched and may resolve spontaneously. Careful large particle embolization could be considered during fibroid embolization if a small AVM were detected. Larger AVMs require more of a sclerotherapy approach with a combination of glue, coil and STS foam embolotherapy.

Uterine AVMs can be congenital or acquired as with AVMs anywhere in the body. Congenital AVMs consist of an abnormal tangle of vessels with feeding arteries, draining veins and a nidus which can be variable in size [2]. Acquired AVMs most commonly actually represent AV fistulas with abnormal arteriovenous communications typically secondary to biopsy, dilatation and curettage or trauma [3].

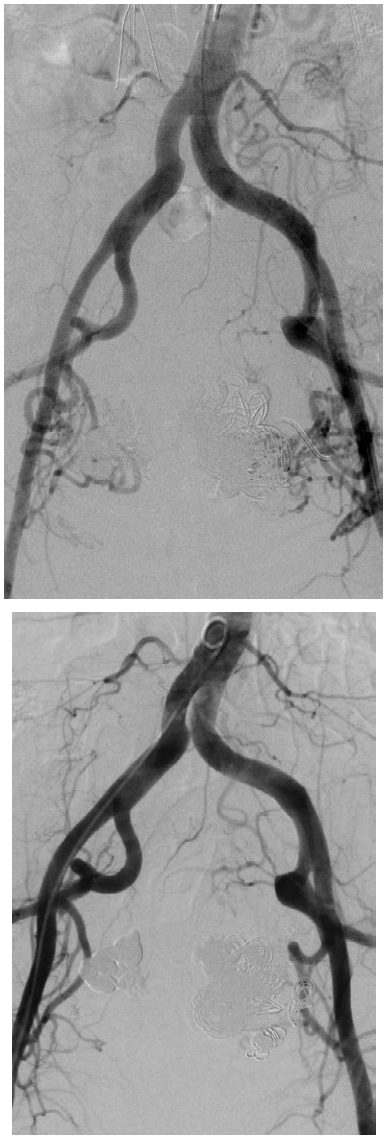


Figure 7: (a and b) Patient returned 4 years later for follow-up pelvic arteriography which demonstrates no further flow within the arteriovenous malformation and complete remission. On the subtracted image, Figure 7b, the lipiodol, n-BCA, and sodium tetradecyl sulfate cast can be seen within the pelvis consistent with embolization of the AVM.

These typically do not have the characteristic nidus that accompanies congenital AVMs.

Patients can present with intermittent massive bleeding which can occasionally be life-threatening. This has been theorized to occur during times of menstruation as uterine sloughing exposes the abnormal arteriovenous channels to the endometrial cavity resulting in vaginal bleeding [4,5]. Oftentimes patients with uterine AVMs have such significant bleeding that it can be lifestyle limiting forcing patients to stay home during the worst bleeding episodes. Hormonal medications such as estrogens and prostaglandins have been used to attempt to decrease bleeding by stimulating proliferative endometrium to cover and thus tamponade the bleeding vessels. Methylergonovine maleate has been used to cause collapse of the abnormal blood vessels and consequently suppress bleeding [3,5].

Imaging of uterine AVMs typically begins with transabdominal and transvaginal pelvic ultrasound with Doppler which demonstrates abnormal vascularity within the uterine myometrium. This is typically followed with enhanced pelvic MRI which on spin-echo sequences demonstrates corresponding abnormal flow voids within the myometrium. Often MRA/MRV imaging can be helpful for pre-operative planning demonstrating the feeding arteries which are typically a combination of the uterine and ovarian arteries and the draining veins including the gonadal veins and pelvic veins.

Hysteroscopy can also be performed as was performed in our first patient which revealed abnormal pulsating masses along the anterior uterine wall. Coagulation of the AVM can be performed hysteroscopically [6] as well as laparoscopically however this can be difficult and may not have as high success rates as direct transcatheter therapy of the nidus. Surgical ligation of the internal iliac arteries has also been reported in the literature as treatment for uterine AVMs, however this would likely not afford a good long term solution as the nidus would still remain and collateralize with time. Furthermore, ligation of the internal iliac arteries proximally would make future transcatheter treatments difficult, limiting treatment to either complex surgical resection possibly including hysterectomy or percutaneous direct puncture sclerotherapy methods if refractory to treatment.

Combination transcatheter and direct puncture sclerotherapy is an emerging method which has been reported in the literature to treat uterine AVMs successfully [3], affording high success rates, preserved fertility, low complication rates and short hospital stays. Typical agents which are utilized include coils, N-butyl cyanoacrylate mixed with lipiodol, and STS foam. Particulate embolization should in general be avoided particularly when there are large arteriovenous communications. The goal of treatment should include permanent venous outflow occlusion, direct nidal embolization and arterial embolization in order to provide the greatest chance for long term success.

Following embolization, patients report symptoms similar to those occurring after uterine artery embolization for fibroids, including post-operative pain and nausea. These symptoms are well controlled with PCA pumps and IV antiemetic medications and typically a 23-hour overnight admission was sufficient. Patients may also develop post-embolization syndrome following discharge to include low-grade fevers and pain. As with any other embolization patient's presenting with unremitting high fevers postoperatively should be admitted and carefully monitored for signs of infection and sepsis. Neither of our two patients had significant post-embolization syndromes despite the large amount of embolic agents that were utilized [7-10].

Conclusion

A combination transcatheter and percutaneous sclerotherapy approach with embolization of the nidus, outflow vein and arterial inflow can eradicate uterine AVMs and dramatically improve patient outcomes, preserving patient fertility with limited morbidity and high success rates. In general, particulate embolization should be avoided in large AVMs due to the high risk for AV-shunting.

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