Tangled: A Pictorial Review of Ultrasound and Angiography of Postpartum Hemorrhage due to Uterine Arteriovenous Malformations and Sub-Involution of the Placental Bed

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Post-partum hemorrhage (PPH) can occur in up to 6% of deliveries and is a major cause of maternal mortality. First line conservative management includes uterotonics (oxytocin/misoprostol), and extra and/or intrathecetan compression. Failure of conservative management in PPH progresses to uterine artery embolization (UA) or surgical management. UAE is preferred for PPH after failure of conservative treatment, as UAE can be performed in an emergent manner and can be repeated if necessary. Multiple studies have shown fertility is usually preserved after UAE, while surgical management is more aggressive utilizing either LU ligation or hysterectomy. UAE is effective for multiple types of PPH, and 24 hours after delivery, arteriovenous malformations (AVM) are one of the most common type to require UAE. Embolization with gelatin microspheres is the preferred but in refractory or severe bleeding, N-butyl cyanoacrylate or microspheres can be used. Coils are generally reserved for pseudoaneurysms.

Sub-involution of the placental bed (SIBP) as an etiology for PPH is a relatively underrecognized etiology in both diagnostic and interventional radiology as it is a diagnosis of exclusion. SIBP describes a failure of regression of large placental vessels within the myometrium, with PPH occurring greatest at 2-weeks post-partum. Generally, they can be thought of as focal uterus atony and can respond to uterotonic agents. However, embolization may be required in severe bleeding and, as shown in our imaging, they can recur and cause re-bleeding.

**Clinical Findings**

The appearance of uterine AVM and SIBP in the setting of PPH is indistinguishable on ultrasound. Both manifest as a vascular myometrial based focus with high flow and low-resistance on spectral Doppler. However, uterine vessel angiography can clearly diagnose a uterine AVM with tortuous and hypertrophied uterine arteries and an early draining vein.

We present several cases of PPH with uterine AVM diagnosed on ultrasound that were confirmed with angiography and embolized successfully. We contrast those cases to patients with PPH and features that were consistent with AVM on ultrasound, whom had no evidence of AVM on angiography, thus suggestive of SIBP. Despite the lack of AVM found on angiography, these cases were embolized via gelatin foam or microspheres with successful hemostasis of the PPH.

In our cases, the PVS of both AVM and SIBP are in the 30-60 cm/s range with a low RI. AVMs are more likely to be after instrumentation and will show a focal tangle of vessels with a early draining vein on angiography. SIBP, in contrast, will show only a normal post-partum uterine vascularopathy on angiography without focal vascular lesion or early draining vein. In our cases of SIBP one was after surgical abortion, one was after medical abortion and one was after a C-section with the other two after NSVD. All AVM and SIBP cases had negative beta-HCG at time of ultrasound and UAE.

**Conclusion**

Both SIBP and AVM are a cause of secondary PPH which are indistinguishable on doppler and spectral ultrasound but can be differentiated on pelvic angiography. Both entities can be successfully embolized, in our experience with different sizes of microspheres +/- gelatin foam. SIBP treated with conservative management can regress and recur that then may require embolization for refractory bleeding.

**References**