

Technical Approaches to the Difficult Uterine Artery Embolization

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Uterine artery embolization has been an extremely effective and widely applied method for treating symptomatic fibroids. Much has been written about patient selection, pre- and postoperative management, as well as most details of uterine artery embolization.

For the most part, uterine artery catheterization and embolization is well within the technical skill range of the average interventional radiologist. In particular, the fibroid uterus produces characteristic changes of uterine artery enlargement and flow which generally speaking make catheterization to this vessel quite straightforward using angiographic tools and equipment.

The technically challenging uterine artery embolization however, remains a small but significant (5 to 10 percent) subset of all patients undergoing uterine artery embolization. The ability to recognize, treat, and catheterize these difficult patients may well spell the difference between achieving very high technical success rates (over 98 to 99 percent) or having failure rates of 3 to 5 percent.

Technically challenging cases can be generally described under the following 5 categories.

- 1) Uterine artery spasm.
- 2) Failure to identify the uterine artery origin.
- 3) Managing uterine artery tortuosity.
- 4) Uterine artery anatomic variants and/or alternative pathways of flow to uterine fibroids.
- 5) The patient with recurrent or persistent symptoms after uterine artery embolization.

1) Managing uterine spasm.

The uterine artery, particularly the small to intermediate size uterine artery, has a tendency to go into spasm when traumatized by a catheter or guidewire manipulation. This is particularly true when larger (5 French and greater) catheters and/or hydrophilic guidewires are used. Spasm may result from tortuosity at the origin (see #3 below), but most commonly occurs as the result of repetitive attempts at cannulation of the vessel.

Spasm prevents free-flow embolization to occur and may dramatically change the distribution of embolic particles such that treatment failures may occur when all or portions of fibroids are not adequately embolized.

Recognition of Spasm.

Spasm is usually easily recognized, but spasm that occurs as a catheter is placed and which occurs proximal to the catheter tip (for example at the origin of the vessel) may produce only flow abnormalities. Spasm should be strongly considered when prompt washout of the injected contrast is not demonstrated and persistence of a column of contrast at the catheter tip is seen. In many cases, withdrawing the catheter proximal to the site of spasm will allow recognition and proper treatment.

Prevention and Treatment of Spasm.

Spasm is primarily prevented by gentle catheter manipulations and recognition of anatomic features which might predispose the spasm, such as extreme tortuosity or small uterine artery size. In order to prevent spasm during catheterization, the operator should be aware of these factors and seek to prevent it predominantly through the use of small microcatheters and less traumatic guidewires. At Northwestern, our initial experience indicated that microcatheters were necessary in less than 5 percent of cases but as we have become aware of the negative consequences of spasm, our use of microcatheters has risen to about 15 to 20 percent of all cases, predominantly due to our interest in preventing the time consuming consequences of spasm.

Treatment of spasm once it has occurred is generally through one of 2 interventions: 1) Injection of spasmolytic agents such as nitroglycerin (1 to 200 mg injected directly into the vessel) or nitropaste applied directly to the skin. Other agents which have been described or may be used include priscoline and/or pavaverine. 2) Watchful waiting. Spasm will frequently resolve over the short to intermediate term of the catheter is in place. Simply watching flow patterns and observing complete clearing of contrast may be sufficient to treat spasm.

2) Failure to identify the uterine artery origin.

The uterine artery typically originates off the anterior division of the internal iliac arteries bilaterally and has a characteristic right angle or nearly right angle origin. Recognition of the origin of the uterine artery is usually facilitated by use of the proper oblique. In our experience in over 300 uterine artery embolizations, the use of the ipsilateral anterior oblique is usually the best projection (in over 85 percent of uterine arteries) to demonstrate the origin of the uterine artery. For example, the origin of the left uterine artery is demonstrated in the left anterior oblique and the origin of the right similarly in

the right anterior oblique. If however, the uterine vessel is not seen in the anterior oblique, the contralateral oblique inevitably will demonstrate the vessel to good advantage. This is seen in about 10 to 15 percent of cases.

Failure to demonstrate the origin of the uterine artery may also be caused by anatomic variance (see below) and/or unusual origins such as those which arise as trifurcations or even rarely off the posterior division branches such as the inferior gluteal.

3) Uterine artery tortuosity.

The uterine artery has a tendency to be extremely tortuous; this is particularly true in progressive fibroid disease in which uterine bulk and/or size as well as flow predisposes the uterine artery to not only increase in size but to become highly tortuous. Situations in which uterine artery tortuosity even produces 360 or 720 degree loops in the vessel are not unusual and identification of these highly tortuous uterine arteries is an extremely important factor in not only preventing spasm but insuring technically and successful and complication-free procedures.

The major problem with uterine artery tortuosity is the possibility that either spasm may occur or if a conventional angiographic catheter is used, that the catheter itself may cause kinking and straightening and therefore occlusion of the vessel preventing good free flow embolization.

Dealing with the tortuous vessel predominantly is a function of appropriate catheter guidewire tools.

The following catheters have been particularly useful in cannulating the origin of tortuous uterine arteries:

1. *Microcatheters* such as the Cordis Rapid Transit™ (Cordis Endovascular Inc.) or the Tracker 18™ (Boston Scientific Inc.).
2. A shapeable guidewire such as the Transend™ and/or hydrophilic guidewires such as the Headliner™ (Boston Scientific). The latter guidewire in particular has proved to be particularly useful because of a short right angle bend and a long atraumatic floppy tip length. In general, the more tortuosity, the smaller and/or flexible the microcatheter to be used should be. For example, in highly tortuous vessels we prefer the Rapid Transit™ to the Mass Transit™ (both products from Cordis Endovascular) since the Mass Transit™ may produce kinking and occlusion of the vessel.

4) Anatomic variants.

A great amount has been written about the role of anatomic variance in uterine artery embolization. The variants discussed include uterine supply directly off the aorta or collateral supply from ovarian vessels. Identification of these variants (which also include absent uterine arteries) is an important part of producing technical and clinical result since unembolized uterine segments may obviously show failure of fibroids necrosis and continued symptomatology.

Most authors (our results included) now advise that a search for anatomic variants or alternate routes of flow to the fibroids only begin after a search for conventional uterine arteries fails. In general, a survey of pelvic arteriography, including aortography, is the most useful since the majority of variants and/or alternate flow pathways tend to occur at the aorta or off the aortic branches, such as the renal or ovarian.

Along those same lines, we also strongly encourage that collateral or alternate area of perfusion patterns to the uterus be sought after uterine artery embolization by performing abdominal aortography. Clearly, it has been demonstrated that abdominal aortography performed before embolization of the arteries may well miss collateral pathways which only become evident after uterine artery embolization has occurred.

5) Treatment of the patient with recurrent or persistent symptoms

In all the literature to date, about 90 percent of patients have responded to this therapy. The mechanisms of failure of uterine artery embolization may include failure of fibroids to necrose or regrowth of a fibroid. In general, management of the patient with recurrent or persistent symptoms involves the routine performance of magnetic resonance scanning in these follow-up patients. In my opinion it is paramount that these examinations be conducted with Gadolinium enhancement so that fibroid devascularization and necrosis can be observed and identified properly. In the fibroid which does not show persistent necrosis, alternative pathways and/or perfusion should be sought by repeat arteriography.

The technically challenging uterine artery embolization represents a small but important subset of all patients who undergo uterine artery embolization. Recognition and appropriate management of this difficulty through a combination of the imaging, pharmacologic therapy, and catheter selection is an important part of a successful treatment program for these patients.