When to Consider Ovarian Artery Embolization in UAE

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Contents

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Abstract

The ovarian artery is a major collateral pathway to the uterus and may act as a source of fibroid perfusion. This chapter gives an overview about the ovarian artery anatomy, imaging techniques to visualize collateral arterial supply to uterine fibroids, the technique of ovarian artery embolization (OAE) as well as the risks and benefits of supplemental OAE in the setting of uterine fibroid embolization.

1 Introduction

Uterine artery embolization (UAE) has become an accepted treatment option in patients suffering from symptomatic uterine leiomyomata (fibroids) with a high clinical success rate. Among other causes, incomplete devascularisation of uterine fibroids is considered to be an important negative prognostic factor for the long-term clinical outcome of the procedure (Pelage et al. 2004; Kroencke et al. 2006). Collateral arterial supply to uterine fibroids has been identified as a relevant cause for incomplete fibroid infarction and subsequent clinical failure (Nikolic et al. 1999; Matson et al. 2000). The ovarian arteries represent the major collateral pathway to the uterus with significant supply to uterine leiomyomata in up to 6 % of patients undergoing UAE for symptomatic fibroids according to the literature (White et al. 2007). In addition, the connection between the uterine and ovarian artery circulation, known as the uterine-ovarian anastomoses, represents a potential pathway for nontarget embolization of the ovary during UAE. Furthermore, supplemental ovarian artery embolization (OAE) has been advocated in selected cases of ovarian artery collateral supply to ensure complete fibroid infarction of targeted fibroids. Knowledge about the anatomy of the ovarian artery and recognition of this collateral pathway including types of uterine-to-ovarian artery anastomoses as well as knowledge about the technique, benefits,

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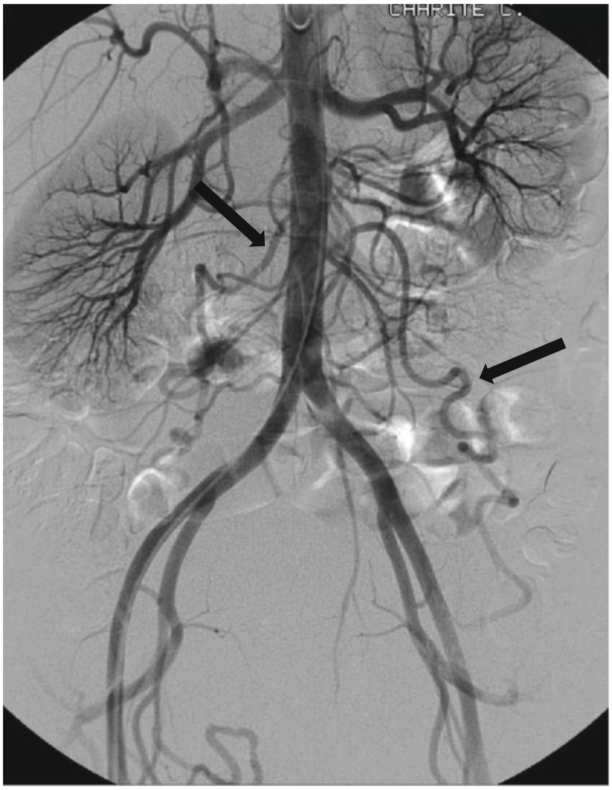

Fig. 1 Flush aortography with a pigtail catheter positioned at the level of the renal arteries shows enlarged ovarian arteries (*black arrows*) which descend retroperitoneally into the pelvis and show the typical corkscrew appearance distally

Fig. 2 Selective angiography of the right ovarian artery shows small branches to the retroperioneum (*black arrow*) and filling of the perifibroid plexus vessels of a leiomyoma

and risks of ovarian artery embolization (OAE) is therefore of importance to interventional radiologists performing UAE.

2 Anatomy of the Ovarian Artery

The ovarian arteries generally originate as paired vessels from the anterolateral aorta below the renal arteries at the level of L2 (see Fig. 1).

They may also arise above or below the renal pedicle from L1 to L4. In cadaveric studies, it is reported that in 6–12 % of cases the ovarian arteries originate directly from the renal arteries, particularly from accessory renal arteries and most commonly from the right (Shoja et al. 2007). In cases where the ovarian artery comes off an accessory renal artery, associated anomalies, such as a common trunk to supply the adrenal gland or replacement of the inferior phrenic artery has been described (Notkovich 1956; Rahman et al. 1993). Seldomly, an ovarian artery may arise in an aberrant fashion from the inferior mesenteric artery (Smoger et al. 2010; Dixon et al. 2012), the common iliac (Kim et al. 2013), external iliac artery (Kwon et al. 2013), or internal iliac artery (Reed and McLucas 2012). The ovarian artery descends retroperitoneally on the psoas muscle, gives branches to the retroperitoneum (Fig. 2) and ureter, which it crosses anteriorly, and can be identified by its characteristical serpentinous ("corkscrew") distal segment before it enters the pelvis (Frates 1969). The ovarian artery then courses medially in the suspensory ligament of the ovary (Syn.: infundibulo pelvic ligament) toward the uterine cornu and gives of branches to the ovary on its lateral border. The tubal branch of the ovarian artery supplies the fallopian tube and variable anastomoses with the uterine artery (uterine-to-ovarian artery anastomoses), lateral and inferior to the junction of the uterine body, and to the fallopian tube (Sieunarine et al. 2005).

3 Classification of the Uterine-to-Ovarian Artery Anastomoses

Razavi proposed a classification of the ovarian artery-to-uterine artery anastomoses (Syn.: uterine-to-ovarian artery anastomoses, UOA) distinguishing three patterns based on selective angiography (Razavi et al. 2002).

According to this classification, a Type I pattern describes an ovarian artery that connects to the intramural uterine artery before branches supply the fibroid(s). Type I can be further subdivided into a Type Ia and Type Ib. In Type Ia, the ovarian artery is a major source of blood supply to the fibroids by means of anastomosis with the intramural uterine artery. In these cases, the flow is toward the uterus, without evidence of retrograde reflux in the direction of the ovary on selective uterine artery angiograms. In Type Ib, the ovarian artery supplies the fibroids in a similar manner as in type Ia. Flow is toward the uterus; however, reflux into the ovarian artery is seen on the selective uterine artery angiogram. In Type II, the ovarian artery supplies the fibroids directly. Although anastomoses to the intramural uterine artery may exist, the flow to the fibroids is anatomically independent of the uterine artery. In Type III, flow is continuous toward the ovary on selective uterine angiograms and washout of the contrast material does not occur from the direction of the ovary within the anastomosis. Theoretically, clinical failure after UAE is likely in patients with Type II and is possible in patients with Type I utero-ovarian anastomosis, whereas those with Type III may rather bear the risk of inadvertent (nontarget) ovarian embolization with resulting ovarian failure. It has to be kept in mind that the uterine-to-ovarian anastomosis has been directly observed in only 40–50 % of cases undergoing UAE using selective uterine artery angiography, while anastomoses between ovarian branches of the ovarian and uterine arteries have been observed in a constant fashion in cadaveric studies employing injection of contrast media under X-ray (Razavi et al. 2002; Kozik 2000; Kim et al. 2006).

Moreover, this classification does not include the anatomic relationship between uterus, fallopian tube, and ovary with variant supply of the fallopian tube which is part of the uterine artery-to-ovarian anastomosis (Dubreuil-Chambardel 1925). This calls into question if standard angiography is sufficient and reliable to visualize this vascular connection. The frequency of the types and the significance of these patterns, as described by Razavi et al., and the impact on fibroid perfusion as well as ovarian function is still debated. In a large study including 202 patients undergoing UAE for symptomatic fibroids Lanciego et al. did not find an association between clinical outcomes and any of the types of ovarian artery-to-uterine artery anastomoses (Lanciego et al. 2012). Other factors, such as patient age, may be far more important to ovarian function than visibility and type of ovarian artery-to-uterine artery anastomoses identified on an arteriogram during UAE (Hu et al. 2011) (see Table 1).

4 Uterine Artery-to-Ovarian Artery Anastomoses, Ovarian Perfusion, and Function in the Setting of UAE

Several articles address the role of the uterine artery-to-ovarian artery anastomoses in the setting of UAE with respect to subsequent ovarian function. Given the fact that embolic particles can reach the ovary via this anastomosis, the type of anastomosis, the flow direction during embolization, and the size of particles may contribute to the extent of nontarget embolization of ovarian stroma and subsequent reduction of ovarian function (Payne et al. 2002). It has been postulated that larger sized particles may prevent nontarget embolization of the ovary but this has not been substantiated in further studies. Coil embolization of prominent uterine artery-to-ovarian artery anastomosis has been proposed as a measure to prevent premature ovarian failure (Marx et al. 2003). This approach is possible only in a minority of cases where the anastomosis can be reached without difficulties and creating spasm.

Ryu et al. assessed the delayed effects of uterine artery embolization on ovarian arterial perfusion by performing ovarian sonography immediately before and after UAE, as well as several months later. They showed that although persistent loss of detectable arterial perfusion after UAE occurs in some women, most patients reestablish arterial perfusion and do not develop symptoms of ovarian failure (Ryu et al. 2003).

Kim et al. reported in a cohort of 124 women (mean age, 43.1 ± 5.7 years) undergoing UAE for symptomatic fibroids patent anastomoses between the uterine and ovarian arteries in 55 patients (44.4 %) detected by angiography (Kim et al. 2006). Changes in basal follicle-stimulating hormone (FSH) level obtained on day three of the menstrual cycle before and 6 months after uterine artery embolization (UAE) were more frequent in women with observed patent anastomoses but also highly age dependent. Salazar et al. compared the incidences of symptom recurrence and permanent amenorrhea following uterine artery embolization (UAE) for symptomatic fibroid tumors in patients with Type I and II utero-ovarian anastomoses with versus without supplemented ovarian artery embolization (OAE) and did not find a significant difference regarding permanent amenorrhea rates but higher symptom recurrence rates were observed when OAE was not performed in the setting of UOA (Salazar et al. 2013) (see Fig. 3).

Table 1 Angiographic classification of Ovarian-to-Uterine Artery Anastomoses according to Razavi et al. based on selective uterine artery angiogram as well as pre- and post-UAE flush aortography

Type I	The ovarian artery is a major source of blood supply to the fibroids by means of anastomosis with the intramural uterine artery
Type Ia	Flow within the anastomosis is toward the uterus. No evidence of retrograde reflux in the direction of the ovary on selective uterine artery angiograms
Type Ib	Flow within the anastomosis is toward the uterus but reflux into the ovarian artery is seen on pre-embolization selective uterine artery angiograms
Type II	The ovarian artery supplies the fibroid(s) directly
Type III	Flow in the anastomosis is continuously toward the ovary with an ovarian blush on selective uterine artery angiograms. Wash out occurs in the direction of the ovary

Adapted from Razavi MK, Wolanske KA, Hwang GL et al. Angiographic classification of ovarian artery-to-uterine artery anastomoses: initial observations in uterine fibroid embolization. Radiology 2002; 224:707–712

Fig. 3 Selective angiography of the left ovarian artery shows collateral perfusion of the uterus. Opacification of vessels supplying the left ovary (*black arrow*) is also noted

Although a plethora of prospective studies have been undertaken to assess the relation between UAE and ovarian function by using hormonal assays and ultrasound measurements, only three of these were comparative studies comparing UAE to surgical treatments such as myomectomy or Hysterectomy. These studies show no differences in FSH level between the treatment groups (Salazar et al. 2013; Healey et al. 2004; Hovsepian et al. 2006; Tropeano et al. 2010). Two studies including the randomized EMMY trial comparing UAE to hysterectomy showed, however, a decrease in anti-mullerian-hormone (AMH), a marker which is cycle independent and considered more sensitive to test the extent of ovarian reserve (Healey et al. 2004; Hehenkamp et al. 2007). Ovarian reserve is a term used to describe the functional potential of the ovary and reflects the number and quality of oocytes within it. To evaluate the extent of ovarian reserve reduction, day three FSH is of limited value since it is an indirect marker, reflecting the hormonal balance between the ovaries and the hypothalamo-pituitary axis and large intercycle variations in basal FSH occur (Maheshwari et al. 2006).

In the EMMY trial, FSH increased significantly compared to baseline at 24 months follow up after hysterectomy and UAE. No differences in FSH values between the groups undergoing UAE versus hysterectomy were found. AMH levels were significantly reduced during the entire follow up period only in the UAE group compared to the baseline values and expected AMH decrease due to aging. However, a significant difference between UAE and hysterectomy group was only observed at 6 weeks follow up but not during the later course of the study. A number of studies assessed clinical and hormonal outcome by age groups and concluded that UAE does not significantly affect ovarian function in women under age of 45 years (Tropeano et al. 2004, 2010; Spies et al. 2001; Tulandi et al. 2002). For an in-depth review on the issue see Kaump et al. (Kaump and Spies 2013).

5 Imaging of the Ovarian Artery and Indication for Ovarian Artery Embolization

As early as 1999, several reports have confirmed the possibility of clinical failure after UAE due to collateral supply of uterine fibroids by the ovarian artery (Nikolic et al. 1999; Matson et al. 2000; Andrews et al. 2000). Pelage et al. reported that ovarian artery supply of fibroids were more common in women with large fundal fibroids, previous tubo-ovarian pathology, or surgery (Pelage et al. 2003). Additional ovarian artery embolization has been advocated for these selected cases (Andrews et al. 2000; Pelage et al. 2003; Barth and Spies 2003; Scheurig-Muenkler et al. 2011). The decision to perform ovarian artery embolization (OAE) should be based on the extent of collateral supply and includes careful analysis of pre-UAE MR angiography, if available as well as unselective and selective catheter angiography during the procedure. Appropriate counseling prior to adjunctive OAE is mandatory (Andrews et al. 2009).

Due to its high spatial and temporal resolution, flush aortography is the reference standard for imaging of the ovarian arteries. However, the standard use of flush aortography prior to UAE is of limited value. Binkert et al. demonstrated ovarian collateral arteries on *Pre-UAE* Aortography in 13/51 patients (25 %), but their detection influenced treatment in only 6 % of the reported cases (Binkert et al. 2001). Furthermore, it has to be kept in mind that flush aortography adds up to 20 % of the total radiation dose of a UAE procedure (White et al. 2007).

Abbara et al. concluded from their study on the use of flush aortography *after* UAE that the presence of residual fibroid perfusion is more likely if the ovarian arteries are large, have rapid flow, or have flow that extends into the pelvis and recommended selective ovarian artery evaluation in these cases to determine the extent of residual fibroid perfusion (Abbara et al. 2007).

In a retrospective study of 1,128 consecutive women undergoing UAE for uterine fibroids, White et al. reviewed *Post-UAE* flush aortographies performed in 1,072 patients (White et al. 2007). Based on the criteria published by Abbara et al., selective ovarian artery angiography was performed to confirm ovarian artery supply to targeted fibroids. Around 17 % of patients undergoing UAE in their retrospective study had at least one visible ovarian artery on Post-UAE flush aortography which either was visible down to the level of the ovaries and/or showed rapid arterial flow. Sixty two (5.8 %) patients were identified at selective ovarian angiography as having collateral OA supply of the uterus and embedded fibroids.

Overall, Post-UAE flush aortography identified fewer than 1 % of patients as having substantial ovarian artery collateral supply and had a sensitivity of less than 25 % in identifying patients with residual uterine perfusion from an ovarian artery. The authors concluded that post-UAE aortography rarely helps to identify patients with substantial residual OA supply to the uterus and is a poor predictor of the extent of that supply, and thus its routine use during UAE is not recommended.

It has been shown that magnetic resonance angiography (MRA) included in a pre-UAE MR imaging exam can visualize enlarged ovarian arteries (Kroencke et al. 2006; Mori et al. 2010). The size of these enlarged ovarian arteries, usually as large or larger than the diameter of lumbar arteries, is an important finding suggesting relevant collateral supply to the uterus (see Fig. 4).

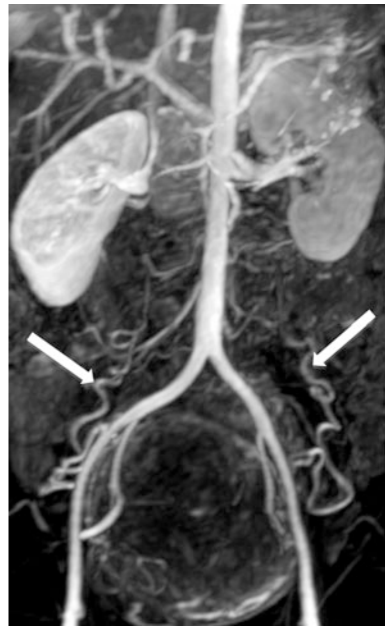

Fig. 4 Contrast-enhanced (ce) magnetic resonance angiography (MRA) prior to UAE. Enlarged ovarian arteries (OA) are detected (*white arrows*). The size of both OA is equal or larger than lumbar arteries in their pelvic segments

However, the direct visualization of enlarged ovarian arteries is an imperfect predictor. It is known that dilated ovarian arteries seen on pre-UAE aortography may not be visible after the embolization of uterine arteries, presumably due to occlusion of the relevant fibroid supply resulting in a reduction in the "sump effect" of the fibroids with reduced demand through the uterine-to-ovarian artery anastomosis or embolic occlusion of the anastomosis after UAE (Binkert et al. 2001). This may be the case in Type I uterine-to-ovarian artery anastomosis representing an OA connected to the intramural UA. In these cases although markedly enlarged OA are identified on preinterventional contrast-enhanced MR angiography, a relevant OA supply may not be confirmed on post-UAE aortography.

A possible explanation is the reduced demand via this collateral pathway after UAE.

In a study conducted by Lee et al., the likelihood of supplemental OAE was significantly higher in those patients who had small or nonvisible uterine arteries on contrast-enhanced MR angiography (Lee et al. 2012). The uterine artery diameter and presence of enlarged ovarian arteries had a high specificity and negative predictive value for ovarian artery collateral supply, but the uterine artery diameter was a better criterion than the detection of enlarged ovarian arteries by MR imaging. CT Angiography with its unchallenged spatial resolution can delineate the ovarian and uterine arteries. It has been advocated for failed

Fig. 5 Flush aortography prior (**a**) and post uterine artery embolisation (**b**) After UAE the right ovarian artery (black arrow in Fig. 5a) is not opacified anymore.

UAE procedures to identify collateral and persistent uterine artery supply but it does add little information compared to MR Imaging and is associated with significant radiation exposure to the patient which does not seem to justify routine use (McLucas 2009).

In summary, integrating MR angiography into the *pre-interventional* imaging exam seems to be currently the best approach to determine the possibility of ovarian artery collateral supply. In case of clinical failure after UAE, interventionists have to look for the possibility of collateral supply of uterine fibroids by previously undetected ovarian arteries.

6 Technical Aspects

Catheterization of the ovarian artery is sometimes challenging due to the steep angle of origin from the abdominal aorta. Reversed-curve catheters such as a 0.038-inch inner lumen Sos-Omni or the Mikkaelsson type configuration give stability within the aorta but also allow to enter the orifice of the vessel (Fig. 6a, b). It is recommended to use a microcatheter which is coaxially advanced over a microwire into the usually straight first part of the ovarian artery. Tension on the vessel must be avoided since the ovarian artery is prone to spasm and spasmolytics should be given if necessary. In most cases, it is neither necessary nor advisable to advance the microcatheter as far as possible since this is usually resulting in spasm due to the tortuosity of the ovarian artery. Limitation of flow should be avoided to ensure that the particulate embolic agent is reaching the perifibroid plexus. Gelatine sponge, nonspherical PVA (355–550 µm), as well as spherical microspheres in the size range of 700 to 900 µm have been used as embolic agents (Hu et al. 2011; Barth and Spies 2003; Scheurig-Muenkler et al. 2011; Kim et al. 2007). The recommended angiographic endpoint for embolization is near-stasis (Fig. 6c, d) with occlusion of the branches feeding the uterus (Hu et al. 2011; Scheurig-Muenkler et al. 2011).

7 Clinical Outcome After Ovarian Artery Embolization

Very few studies have investigated safety and clinical outcome of ovarian artery embolization.

In early reports, the embolization of the ovarian artery was reported to be technically feasible, safe without affecting the menstrual cycle, and successful with alleviation of fibroid-related symptoms of treated patients (Andrews et al. 2000; Pelage et al. 2003). Salazar et al. compared the incidences of symptom recurrence and permanent amenorrhea following UAE for symptomatic fibroids in patients with Type I and II uterine-to-ovarian artery anastomoses with versus without ovarian artery embolization (OAE) and found no statistical differences in permanent amenorrhea rates in the groups studied, but a significantly higher symptom recurrence rate when OAE was not performed (Salazar et al. 2013) Barth et al. analyzed 6 patients treated by supplemental OAE and found no effect on the menstrual cycle of these women and reported clinical success in five of them (Barth and Spies 2003). Our group from Charité reported on 13 patients who underwent UAE and additional OAE and in 10 of the 13 patients, improvement or complete resolution of clinical symptoms was observed.

These women presented with regular menses. Two patients, 47 and 48 years, both treated with additional unilateral OAE, reported permanent amenorrhea directly after

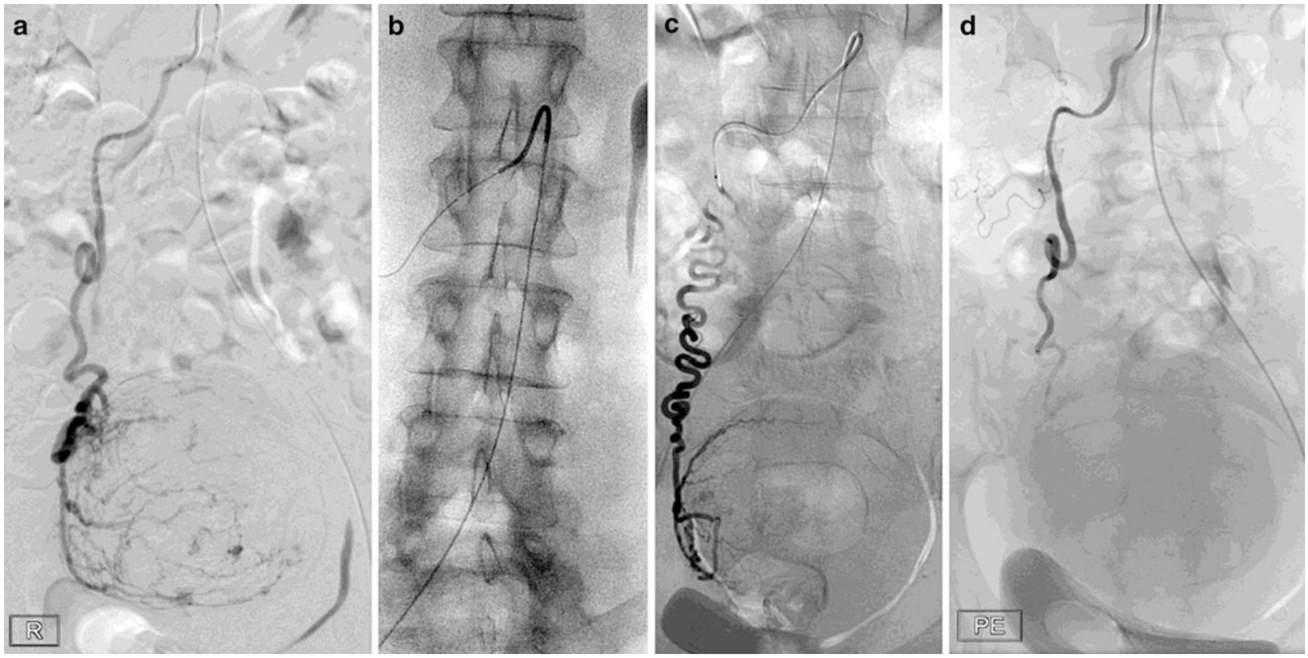

Fig. 6 Selective catheterization of the right ovarian artery by a 0.038 inner lumen Sos Omni catheter (**a**). A microcatheter is coaxially advanced (**b**). Selective angiography through the microcatheter positioned in the straight part of the ovarian artery reveals collateral supply to a fibroid uterus (**c**). Post ovarian artery embolisation (OAE) angiography shows standing column of contrast within the OA and no opacification of terminal uterine branches (**d**)

embolization (Scheurig-Muenkler et al. 2011). In the largest series published to date, 77 patients of a cohort of 1,451 patients (5.3 %) underwent additional OAE (Hu et al. 2011). In this single-center case–control study, 51 patients undergoing OAE were compared to 49 control subjects using the Menopause Rating Scale (MRS), a validated menopausal symptom questionnaire. Compared with standard UAE, the addition of OAE did not precipitate the onset of menopause nor did OAE increase the menopausal symptom severity.

References

Abbara S, Nikolic B, Pelage JP, Banovac F, Spies JB (2007) Frequency and extent of uterine perfusion via ovarian arteries observed during uterine artery embolization for leiomyomas. AJR Am J Roentgenol 188(6):1558–1563

Andrews RT, Bromley PJ, Pfister ME (2000) Successful embolization of collaterals from the ovarian artery during uterine artery embolization for fibroids: a case report. J Vasc Interv Radiol 11(5):607–610

Andrews RT, Spies JB, Sacks D, Worthington-Kirsch RL, Niedzwiecki GA, Marx MV et al (2009) Patient care and uterine artery embolization for leiomyomata. J Vasc Interv Radiol 20(7 Suppl): S307–S311

Barth MM, Spies JB (2003) Ovarian artery embolization supplementing uterine embolization for leiomyomata. J Vasc Interv Radiol 14(9 Pt 1):1177–1182

Binkert CA, Andrews RT, Kaufman JA (2001) Utility of nonselective abdominal aortography in demonstrating ovarian artery collaterals in patients undergoing uterine artery embolization for fibroids. J Vasc Interv Radiol 12(7):841–845

Dixon S, Tapping CR, Chuah PS, Bratby M, Uberoi R, Anthony S (2012) Successful fibroid embolization of pelvic and inferior mesenteric artery collaterals after previous uterine artery embolization. Acta Radiol 53(3):292–295

Dubreuil-Chambardel L (1925) Les variations du corps humain. Flammarion, Paris

Frates RE (1969) Selective angiography of the ovarian artery. Radiology 92(5):1014–1019

Healey S, Buzaglo K, Seti L, Valenti D, and Tulandi T (2004) Ovarian function after uterine artery embolization and hysterectomy. J Am Assoc Gynecol Laparosc 11(3):348–352

Hehenkamp WJ, Volkers NA, Broekmans FJ, de Jong FH, Themmen AP, Birnie E et al (2007) Loss of ovarian reserve after uterine artery embolization: a randomized comparison with hysterectomy. Hum Reprod 22(7):1996–2005

Hovsepian DM, Ratts VS, Rodriguez M, Huang JS, Aubuchon MG, Pilgram TK (2006) A prospective comparison of the impact of uterine artery embolization, myomectomy, and hysterectomy on ovarian function. J Vasc Interv Radiol 17(7):1111–1115

Hu NN, Kaw D, McCullough MF, Nsouli-Maktabi H, Spies JB (2011) Menopause and menopausal symptoms after ovarian artery embolization: a comparison with uterine artery embolization controls. J Vasc Interv Radiol 22(5):710–715.e1

Kaump GR, and Spies JB (2013) The impact of uterine artery embolization on ovarian function. J Vasc Interv Radiol 24(4): 459–467

Kim HS, Tsai J, Patra A, Lee JM, Griffith JG, Wallach EE (2006) Effects of utero-ovarian anastomoses on clinical outcomes and repeat intervention rates after uterine artery embolization. J Vasc Interv Radiol 17(5):783–789

Kim HS, Tsai J, Lee JM, Vang R, Griffith JG, Wallach EE (2006) Effects of utero-ovarian anastomoses on basal follicle-stimulating

hormone level change after uterine artery embolization with tris-acryl gelatin microspheres. J Vasc Interv RadiolJun;17(6):965–971
Kim HS, Thonse VR, Judson K, Vang R (2007) Utero-ovarian anastomosis: histopathologic correlation after uterine artery embolization with or without ovarian artery embolization. J Vasc Interv Radiol 18(1 Pt 1):31–39
Kim WK, Yang SB, Goo DE, Kim YJ, Chang YW, Lee JM (2013) Aberrant ovarian artery arising from the common iliac artery: case report. Korean J Radiol 14(1):91–93
Kozik W (2000) Arterial vasculature of ovaries in women of various ages in light of anatomic, radiologic and microangiographic examinations. Ann Acad Med Stetin 46:25–34
Kroencke TJ, Scheurig C, Kluner C, Taupitz M, Schnorr J, Hamm B (2006) Uterine fibroids: contrast-enhanced MR angiography to predict ovarian artery supply–initial experience. Radiology 241(1):181–189
Kwon JH, Kim MD, Lee KH, Lee M, Lee MS, Won JY et al (2013) Aberrant ovarian collateral originating from external iliac artery during uterine artery embolization. Cardiovasc Intervent Radiol 36(1):269–271
Lanciego C, Diaz-Plaza I, Ciampi JJ, Cuena-Boy R, Rodríguez-Martín N, Maldonado MD, et al (2012) Utero-ovarian anastomoses and their influence on uterine fibroid embolization. J Vasc Interv Radiol 23(5):595–601
Lee MS, Kim MD, Lee M, Won JY, Park SI, Lee do Y, Lee KH (2012) Contrast-enhanced MR angiography of uterine arteries for the prediction of ovarian artery embolization in 349 patients. J Vasc Interv Radiol 23(9):1174–1179
Maheshwari A, Fowler P, Bhattacharya S (2006) Assessment of ovarian reserve–should we perform tests of ovarian reserve routinely? Hum Reprod 21(11):2729–2735
Marx M, Wack JP, Baker EL, Stevens SK, and Barakos JA (2003) Ovarian protection by occlusion of uteroovarian collateral vessels before uterine fibroid embolization. J Vasc Interv Radiol 14(10):1329–1332
Matson M, Nicholson A, Belli AM (2000) Anastomoses of the ovarian and uterine arteries: a potential pitfall and cause of failure of uterine embolization. Cardiovasc Intervent Radiol 23(5):393–396
McLucas B (2009) Collateral supply to uterine leiomyomata from an unnamed vessel: a case report. Minim Invasive Ther Allied Technol 18(2):106–108
Mori K, Saida T, Shibuya Y, Takahashi N, Shiigai M, Osada K et al (2010) Assessment of uterine and ovarian arteries before uterine artery embolization: advantages conferred by unenhanced MR angiography. Radiology 255(2):467–475
Nikolic B, Spies JB, Abbara S, Goodwin SC (1999) Ovarian artery supply of uterine fibroids as a cause of treatment failure after uterine artery embolization: a case report. J Vasc Interv Radiol 10(9):1167–1170
Notkovich H (1956) Variations of the testicular and ovarian arteries in relation to the renal pedicle. Surg Gynecol Obstet 103(4):487–495
Payne JF, Robboy SJ, Haney AF (2002) Embolic microspheres within ovarian arterial vasculature after uterine artery embolization. Obstet Gynecol 100(5 Pt 1):883–886
Pelage JP, Walker WJ, Le Dref O, Rymer R (2003) Ovarian artery: angiographic appearance, embolization and relevance to uterine fibroid embolization. Cardiovasc Intervent Radiol 26(3):227–233
Pelage JP, Guaou NG, Jha RC, Ascher SM, Spies JB (2004) Uterine fibroid tumors: long-term MR imaging outcome after embolization. Radiology 230(3):803–809
Rahman HA, Dong K, Yamadori T (1993) Unique course of the ovarian artery associated with other variations. J Anat 182 (Pt 2): 287–290
Razavi MK, Wolanske KA, Hwang GL, Sze DY, Kee ST, Dake MD (2002) Angiographic classification of ovarian artery-to-uterine artery anastomoses: initial observations in uterine fibroid embolization. Radiology 224(3):707–712
Reed RA, McLucas B (2012) Aberrant right ovarian artery from Proximal Internal Iliac Artery in Uterine artery embolization patient. Open J Radiol. 2(4):117–119
Ryu RK, Siddiqi A, Omary RA, Chrisman HB, Nemcek AA, Sichlau MJ, Vogelzang RL (2003) Sonography of delayed effects of uterine artery embolization on ovarian arterial perfusion and function. AJR Am J Roentgenol 181(1):89–92
Salazar GM, Gregory Walker T, Conway RF, Yeddula K, Wicky S, Waltman AC, Kalva SP (2013) Embolization of angiographically visible type I and II utero-ovarian anastomoses during uterine artery embolization for fibroid tumors: impact on symptom recurrence and permanent amenorrhea. J Vasc Interv Radiol 24(9):1347–1352
Scheurig-Muenkler C, Poellinger A, Wagner M, Hamm B, Kroencke TJ (2011) Ovarian artery embolization in patients with collateral supply to symptomatic uterine leiomyomata. Cardiovasc Intervent Radiol 34(6):1199–1207
Shoja MM, Tubbs RS, Shakeri AB, Oakes WJ (2007) Origins of the gonadal artery: embryologic implications. Clin Anat 20(4):428–432
Sieunarine K, Moxey P, Boyle DC, Ungar L, Lindsay I, Del Priore G, Smith JR (2005) Selective vessel ligation in the pelvis: an invaluable tool in certain surgical procedures. Int J Gynecol Cancer 15(5):967–973
Smoger DL, Kancherla V, Shlansky-Goldberg RD (2010) Uterine fundal blood supply from an aberrant left ovarian artery originating from the inferior mesenteric artery: implications for uterine artery embolization. J Vasc Interv Radiol 21(6):941–944
Spies JB, Roth AR, Gonsalves SM, Murphy-Skrzyniarz KM (2001) Ovarian function after uterine artery embolization for leiomyomata: assessment with use of serum follicle stimulating hormone assay. J Vasc Interv Radiol 12(4):437–442
Tropeano G, Di Stasi C, Litwicka K, Romano D, Draisci G, Mancuso S (2004) Uterine artery embolization for fibroids does not have adverse effects on ovarian reserve in regularly cycling women younger than 40 years. Fertil Steril 81(4):1055–1061
Tropeano G, Di Stasi C, Amoroso S, Gualano MR, Bonomo L, Scambia G (2010) Long-term effects of uterine fibroid embolization on ovarian reserve: a prospective cohort study. Fertil Steril 94(6):2296–2300
Tulandi T, Sammour A, Valenti D, Child TJ, Seti L, Tan SL (2002) Ovarian reserve after uterine artery embolization for leiomyomata. Fertil Steril 78(1):197–198
White AM, Banovac F, Yousefi S, Slack RS, Spies JB (2007) Uterine fibroid embolization: the utility of aortography in detecting ovarian artery collateral supply. Radiology 244(1):291–298