Uterine artery embolization for adenomyosis without fibroids


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AIM: To evaluate the potential usefulness of transcatheter uterine artery embolization as a treatment for symptomatic adenomyosis in patients without uterine fibroids.

MATERIALS AND METHODS: Uterine artery embolization using polyvinyl alcohol particles sized 250–710 μm was performed in 43 patients (mean; 40.3 years, range; 31–52 years) with dysmenorrhoea, menorrhagia, or bulk-related symptoms (pelvic heaviness, urinary frequency) due to adenomyosis without fibroids. All patients underwent pre-procedural and 3.5 months (range 1–8 months) follow-up magnetic resonance imaging (MRI) with contrast enhancement. Clinical symptoms were also assessed at the time of MRI before and after embolization.

RESULTS: Significant improvement of dysmenorrhoea (95.2%) and menorrhagia (95.0%) was reported in most patients. Contrast-enhanced MRI revealed non-enhancing areas suggesting coagulation necrosis of adenomyosis in 31 patients (72.1%), decreased size without necrosis in 11 patients (25.6%), and no change in one patient (2.3%). The mean volume reduction of the uteri after uterine artery embolization was 32.5% (from 321.7 ± 142.9 to 216.7 ± 130.1 cm³).

CONCLUSION: Transcatheter uterine artery embolization is an effective therapy for the treatment of symptomatic pure adenomyosis, and may be a valuable alternative to hysterectomy.

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Introduction

Adenomyosis is characterized by the presence of heterotopic endometrial glands and stroma deep within the myometrium with adjacent myometrial hyperplasia. The frequency of adenomyosis reported in the literature ranges widely, from 8.8 to 31%.1,2 Although patients with adenomyosis may be asymptomatic, they frequently present with symptoms of menorrhagia, dysmenorrhoea and bulk-related symptoms due to uterine enlargement. The extent of disease roughly correlates with the severity of symptoms. Hysterectomy is considered to be the definitive treatment for adenomyosis. Conservative treatments have been proposed such as endometrial ablation, endometrial resection and hormonal suppression.3 Uterine artery embolization (UAE) has recently been applied to the management of symptomatic myomas.4-8 The purpose of this study was to evaluate the therapeutic effectiveness of UAE for the treatment of symptomatic pure adenomyosis of the uterus without fibroids.

Materials and methods

The Institutional Review Board of Bundang CHA General Hospital, S. Korea, approved this study. Between 1998 and 2002, 103 women underwent UAE...
for either adenomyosis, fibroids or both. All patients had magnetic resonance imaging (MRI) as part of the pre-UAE evaluation. Forty-one had uterine myomata, 19 had both uterine myomata and adenomyosis and 43 had only adenomyosis. The 43 patients (age range 31–52 years; mean age 40.3 years) with adenomyosis only were retrospectively studied. Pre-procedural and 3.5 months follow-up MRI (1.5 T Supermagnet, Magnetom Vision, Siemens, Erlangen, Germany) of the pelvis were performed in all patients. Clinical data were based on medical records, and MRI images were reviewed by two radiologists. Diagnostic criteria of adenomyosis on MRI include poorly defined, low signal intensity mass with or without bright foci on T2-weighted image and thickening of junctional zone more than 12 mm. Diffuse adenomyosis was defined as diffuse ectopic growth of the endometrium into the myometrium with either diffuse or focal widening of endometrial-myometrial junctional zone. Focal adenomyosis was defined as an actual subcorticated mass within the myometrium.

Symptoms caused by the adenomyosis were dysmenorrhoea (n = 42; 97.7%), menorrhagia (n = 40; 93.0%), and bulk-related symptoms such as pelvic heaviness (n = 32; 74.4%) and urinary frequency (n = 31; 72.1%). Cases of adenomyosis combined with myoma were excluded in this study.

The right common femoral artery was used for arterial access in all patients. Non-selective pelvic arteriography was performed, and if the entire course of uterine artery could not be evaluated, additional internal iliac arteriography was performed. After this, both uterine arteries were catheterized.

Co-axial 3 F catheters (Tracker-18 Infusion Catheter, Boston Scientific, Fremont, USA) were used to get as distal as possible into the uterine artery. Polyvinyl alcohol (PVA) particles (250–710 mm; Contour, Boston Scientific, Fremont, USA) mixed with 40 mL of 1:1 saline-contrast mixture were used. Embolization was performed until there was complete cessation of blood flow in the ascending uterine artery with residual flow in lower uterine segment. Between 0.5 and 4.5 vials of PVA (mean 1.87 vials) were used for the embolization.

Clinical response was assessed by questionnaire, including symptomatic changes in menorrhagia, dysmenorrhoea, and bulk-related symptoms, and satisfaction about the procedure. In view of menorrhagia and dysmenorrhoea, patients were asked to report improvement of symptoms on a scale of 0 (little bleeding, no pain) to 10 (severe menorrhagia, severe pain) according to the number of pads used, analgesics and experiences. We compared pre-UAE and post-UAE scales in each case. Wilcoxon signed rank test was used in statistical analysis for menorrhagia and dysmenorrhoea.

Pre and post-procedure contrast-enhanced MRI examinations were performed in all patients who underwent axial fast spin-echo T2-weighted image, T2-weighted sagittal image using half-Fourier single-shot turbo spin-echo (HASTE) and contrast-enhanced T1-weighted sagittal image. The following imaging parameters were used for HASTE sequence: repetition time (TR) = 4.4 ms, echo time (TE) = 90 ms, 30 cm field of view, 160 × 256 matrix, one acquisition, 6 mm slice thickness.

Enhanced MRI was performed 2 min after intravenous infusion of 10 ml gadolinium (Dotarem, Guerbet, Aulnay-sous-Bois, France) with FLASH (fast low-angle shot) sequences (TR/TE 117.3/4.1 ms; flip angle 80°; matrix 140 × 256; section thickness 5 mm). Follow-up contrast-enhanced MRI was performed at 3.5 month (range 1–8 months).

MRI findings were classified into four categories as follows: total necrosis, partial necrosis, decreased size of uterus without necrosis, and no change. The presence of necrosis was defined as high signal intensity on T1-weighted image, low signal intensity on T2-weighted image, and no contrast enhancement on enhanced T1-weighted images. Partial necrosis was considered to have occurred when some adenomyotic tissue remained though necrosis was clearly present in other areas.

Volume measurements were obtained on the uterus in each patient using MRI. The formula for volume was that of a prolate ellipse (length × width × depth × 0.5233).

Results

With use of microcatheter, UAE was successfully performed in all patients without spasm. Thirty-eight (95.0%) of the 40 patients who had complained of menorrhagia experienced improvement of the symptom after embolization. By Wilcoxon signed rank test, the median change in score was −4 (p < 0.001). Forty (95.2%) of the 42 patients who had complained of dysmenorrhoea showed improvement after embolization with a median change in score of −5 (p < 0.001). Twenty-five (78.1%) of the 32 patients who had complained of pelvic heaviness, and 15 (48.4%) of 31 patients with urinary frequency showed symptomatic improvement after embolization.

Forty (93.0%) of 43 patients were satisfied with UAE. The causes of dissatisfaction with the
Figure 1  Total necrosis of focal adenomyosis. (a) T2-weighted sagittal image shows asymmetrical thickening of uterine posterior wall (arrows) with multifocal high signal spots (arrowheads). (b) Gadolinium-enhanced T1-weighted sagittal image shows diffuse enhancement of adenomyotic tissue. (c) On T2-weighted sagittal image, a dark signal intensity is noted at the adenomyotic region after UAE. The uterine volume was 631.9 cm$^3$ before embolization and 271.4 cm$^3$ after embolization, a volume reduction of 57.0%. (d) Gadolinium-enhanced T1-weighted image shows no enhancement at the adenomyotic region (arrows) suggesting ischaemic necrosis, but remaining normal myometrium (asterisk) shows enhancement.
Figure 2  Partial necrosis of diffuse adenomyosis. (a) T2-weighted image shows diffuse enlargement of uterus with multifocal bright spots (arrowheads). (b) Gadolinium-enhanced T1-weighted sagittal image shows diffuse enhancement of adenomyosis. (c) T2-weighted sagittal image after UAE shows multifocal dark signal intensity (arrows) and marked decrease in uterine size. The uterine volume was 398.7 cm³ before embolization and 189.9 cm³ after embolization, a volume reduction of 52.4%. (d) On gadolinium-enhanced T1-weighted image, multifocal non-enhancing portions (arrows) are seen in uterus.
Figure 3  Decreased size of uterus without necrosis in focal adenomyosis. (a) T2-weighted sagittal image shows an ill-defined, focal low signal intensity of mass (arrows) on the anterior uterine wall. (b) The contrast-enhanced MR image shows focal enhancement (arrowheads) of the adenomyotic tissue. On follow-up MRI after UAE, T2-weighted (c) and contrast-enhanced T1-weighted (d) images show decreased thickness of the focal adenomyotic region without necrosis.
procedure were permanent amenorrhoea after embolization \((n = 1)\), and no symptomatic improvement \((n = 2)\). One patient (40 years old) became amenorrhoeic permanently, and MRI revealed partial necrosis of diffuse adenomyosis of the uterus after UAE and a volume reduction of 39.7%. One of two patients without symptomatic improvement complained of aggravated back pain, although volume reduction was 22.8% after UAE and MRI showed partial necrosis of the uterus. The other showed no change of uterine volume without necrosis after UAE. No patient underwent hysterectomy after embolization due to uncontrolled infection and other symptoms.

At the 3.5 month follow-up, the contrast-enhanced MRI showed total necrosis (Fig. 1) of adenomyotic tissue in 19 patients (44.2%), partial necrosis (Fig. 2) in 12 (27.9%), decreased size of uterus without necrosis (Fig. 3) in 11 (25.6%) and no change in one (2.3%). Total necrosis was found only in focal adenomyosis. The mean volume of the uterus before embolization was 321.7 \(\pm\) 142.9 cm\(^3\) and was 216.7 \(\pm\) 130.1 cm\(^3\) after embolization, a volume reduction of 32.5%.

**Discussion**

Adenomyosis is a common disorder in women and is considered to be an important cause of menorrhagia. Clinical manifestations are similar to those of uterine fibroids and include dysmenorrhoea and menorrhagia. The frequency of adenomyosis by hysterectomy specimens reported in the literature ranges from 8.8 to 31%. The prevalence of disease is largely dependent on the histological criteria used for establishing the diagnosis and the number of sections obtained at the time of histopathological evaluation.

The mechanism of increase in menstrual flow in adenomyosis is still unclear. Some hypotheses have been proposed. Increased menstrual flow might depend on increased endometrial surface resulting from the overall enlargement of uterus. Further, the contractile function of the myometrium would be compromised by dysfunctional hypertrophic smooth muscle. According to Ota et al., in adenomyosis the total surface area of capillaries, as well as the number of capillaries, in the endometrium increased up to 11.6 times that of the controls.

The clinical diagnosis of adenomyosis is difficult because of the non-specific signs and symptoms and frequent coexistence of other pelvic disease. Until recently, adenomyosis could be diagnosed only at surgery, now non-invasive diagnosis is possible using transvaginal ultrasonography and MRI. On T2-weighted MR images, bright foci are seen in areas of abnormal low signal intensity within the myometrium in approximately 50% of patients with adenomyosis. These foci correspond to islands of heterotopic endometrial tissue, cystic dilatation of heterotopic glands, or haemorrhagic foci. Junctional zone thickening and low signal intensity of myometrial mass with ill-defined margins are other characteristic MRI findings.

Hysterectomy is definitive treatment for adenomyosis. Other treatment options currently available for symptomatic patients include drug therapy or conservative surgery such as endomyometrial ablation, laparoscopic myometrial electrocoagulation or excision. Drug therapy is relatively ineffective in treating adenomyosis. Women who pursue hysterectomy for adenomyosis have nearly all been treated previously with drugs for menorrhagia and/or dysmenorrhoea. In endometrial ablation, the success rate was high at first, but with long-term follow-up a significant failure rate became evident, particularly, in deep adenomyosis. Other conservative surgery has proven to be effective in more than 50% of patients, although no long-term follow-up is currently available.

Recently, UAE has been reported as being successful in managing symptoms caused by uterine myomas. There is, however, a failure rate of 10-20%. Adenomyosis of the uterus is reported as one of the possible causes of clinical failure after embolization. However, the therapeutic efficacy of UAE in the management of adenomyosis had been reported. The results of our study showed dramatic improvement of symptoms, suggesting that UAE has the potential as a first-line treatment in patients with adenomyosis.

We used a microcatheter coaxial system to 4 or 5 F catheters to prevent spasm of uterine artery and to gain access to distal uterine artery where possible. In conclusion, increasing evidence suggests that UAE is safe and effective in the treatment of uterine myomata. This study suggests that it is also safe and effective as a uterus-conserving procedure for adenomyosis. The low complication rates, shorter hospital day and rapid recovery time are other favourable features of UAE. Further investigation of the long-term results is indicated.

**References**