



Early Results of Transcatheter Arterial Embolization for Relief of Chronic Shoulder or Elbow Pain Associated with Tendinopathy Refractory to Conservative Treatment

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ABSTRACT

Purpose: To evaluate the effectiveness and safety of transcatheter arterial embolization to relieve pain associated with shoulder and elbow tendinopathy refractory to conservative treatment.

Materials and Methods: This study included 13 patients (15 cases) who underwent embolization between November 2015 and December 2016 to treat chronic shoulder pain (6 with rotator-cuff tendinopathy, 2 with calcific tendinitis) or elbow pain (7 with lateral epicondylitis) refractory to conservative treatment. Microspheres were used in the first 4 cases, and imipenem/cilastatin sodium was used in the remaining 11. Visual analog scale (VAS) score changes were recorded. Decrease in VAS score and degree of enhancement on digital subtraction angiography were compared.

Results: The technical and clinical success rates were 100% (15/15) and 73% (11/15), respectively. The mean VAS scores at baseline, 1 day, 1 week, 1 month, and 4 months after embolization were 6.1, 5.8, 5.1, 4.3, and 2.5, respectively ($P < .05$ after 1 wk). Pain improved in 9 of 10 cases (90%) with “evident” enhancement and 3 of 5 cases (60%) with no evident enhancement. The VAS scores in the evident enhancement group decreased more than those in patients with no evident enhancement (4.5 vs 1.8; $P < .05$). Forearm cutaneous erythema was noted in 1 patient treated with microspheres.

Conclusions: Transcatheter arterial embolization may be an option for relieving pain associated with chronic shoulder and elbow tendinopathy refractory to conservative treatment. The degree of angiographic enhancement might be a possible factor affecting the degree of pain relief after embolization.

ABBREVIATIONS

ESWT = extracorporeal shockwave therapy, IPM/CS = imipenem/cilastatin sodium, VAS = visual analog scale

Primary tendinopathy is a common disorder, and it accounts for a high proportion of visits to rheumatologists and orthopedic surgeons (1). In the general population, the

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prevalence rates of lateral epicondylitis, rotator-cuff tendinopathy, and calcific tendinitis have been reported to be 2.8%, 2%, and 2.7%, respectively (2–4). In addition, tendinopathy accounts for approximately 7% of all physician visits in the United States (1). Although there has been a lack of direct and indirect cost analysis for various tendinopathies, 5% of patients with epicondylitis took sick leave from work for a duration of 29 days in 1 year in the United Kingdom. In addition, costs related to absenteeism resulting from lateral epicondylitis in the United Kingdom were estimated at £27 million in 2012 (5,6). In the United States, total direct medical cost related to lateral epicondylitis was reported at \$660 per patient during the 1-year period after diagnosis (7).

Although tendinopathy is considered a self-limiting condition, it may become resistant to conservative therapies and involve a long recovery time. Approximately one third of patients with tendinopathy experience significant pain and limitation of activities at 6 months after the onset of symptoms (7). Traditional treatment modalities have been focused on the inflammatory process of tendinopathy (8). However, there have been reports describing overuse tendinopathy with minimal or no inflammation (9,10). As the pathophysiology of tendinopathy has not yet been fully established, a multitude of treatment options have been proposed for this disorder, including nonsteroidal anti-inflammatory drugs, physical therapy, corticosteroid injections, extracorporeal shockwave therapy (ESWT), sclerotherapy, growth factor treatment, stem cell treatment, and surgery (9,11). However, most treatment options are limited by inconsistent outcomes.

Surgery focuses on the excision of the fibrotic adhesion and areas of the affected tendon. Open debridement, arthroscopic management, and percutaneous procedures have been reported for surgical treatment of tendinopathy (9,11). However, even though surgery is the last option in the treatment of tendinopathy, the results may not be completely satisfactory, with a wide range of reported failure rates as follows: lateral epicondylitis, 3%–31% (open), 3%–28% (arthroscopic), and 4%–26% (percutaneous); rotator cuff tendinopathy, 29% (arthroscopic); and calcific tendinitis, 6%–21% (arthroscopic) (9,11–14).

Alfredson et al (15) have suggested that neovessels and accompanying nerves are possible sources of inflammation and pain in chronic Achilles tendinosis. Based on such a theoretical background, transcatheter arterial embolization has been performed for adhesive capsulitis (16,17), tendinopathy (18), and osteoarthritis of the knee (19,20). Results of these studies (16–20) have suggested that embolization of these abnormal vessels is effective for pain reduction and clinical symptom improvement without causing complications in treatment for adhesive capsulitis, tendinopathy, and osteoarthritis. However, only a few studies (16–18) have been conducted on transcatheter arterial embolization for pain associated with shoulder and elbow disease. In addition, these studies mainly involve adhesive capsulitis. Transcatheter arterial embolization for shoulder and elbow tendinopathy has been reported in only 3 cases of which we are aware (18). In addition, factors affecting the degree of pain reduction after embolization are unknown in the literature (16–20), and our hypothesis is that the degree of enhancement of the lesion on angiography may affect pain reduction after embolization.

Therefore, the objectives of the present study are to assess the effectiveness and safety of transcatheter arterial embolization to relieve pain associated with shoulder and elbow tendinopathies refractory to conservative treatment and to examine the effect of the degree of angiographic enhancement on pain alleviation.

MATERIALS AND METHODS

Patients

This retrospective study was performed at a tertiary-care center and with institutional review board approval. The informed consent requirement was waived in view of the study's retrospective nature.

Between November 2015 and December 2016, a total of 613 patients who visited the orthopedic surgery outpatient department with shoulder or elbow pain were diagnosed with shoulder tendinopathy (rotator cuff tendinopathy, $n = 262$; calcific tendinitis, $n = 148$) or elbow tendinopathy (lateral epicondylitis, $n = 203$) based on shoulder or elbow joint magnetic resonance imaging or ultrasonography.

Among these patients, those who had persistent pain for more than 6 months and pain refractory to conservative treatment were included in the present study. The exclusion criteria were local infection at the site of pain, age younger than 18 years, and history of previous tendon surgery or trauma. Patient selection was accomplished by a multidisciplinary approach in collaboration with orthopedic surgeons and interventional radiologists.

As a result, 13 patients (15 cases; 10 women and 3 men with a mean age of 52.4 y; range, 27–75 y) were referred for transcatheter arterial embolization and included in the present study. Two patients had shoulder and elbow pathologic conditions at the same time and underwent a single embolization session. The etiologies were shoulder pain (rotator cuff tendinopathy, $n = 6$; calcific tendinitis, $n = 2$), and elbow pain (lateral epicondylitis, $n = 7$). Conservative treatments previously performed included administration of pain relievers (nonsteroidal anti-inflammatory drugs, $n = 3$; tramadol, $n = 9$), corticosteroid injection ($n = 12$), and ESWT ($n = 5$). The mean duration of symptoms was 16.7 months \pm 15.3 (range, 6–68 mo; median, 13 mo). Patients' demographic and clinical data are summarized in the **Table**.

Embolization Procedure

Under local anesthesia, percutaneous arterial access was obtained by using a 5-F introducer sheath (Terumo, Tokyo, Japan) via the common femoral artery ($n = 11$) or a 4-F sheath via the brachial artery ($n = 2$). Baseline selective subclavian, axillary, and/or brachial arteriography was performed by using a 5-F (H1; Cook, Bloomington, Indiana) or 4-F angiographic catheter (Glidecath, non-taper angle; Terumo) to identify abnormal enhancement around the shoulder or elbow area.

After identifying abnormal enhancement, superselective arteriograms of corresponding feeding arteries were obtained by using a coaxial 2.0-F microcatheter (Parkway Soft; Asahi Intecc, Nagoya, Japan) and microguidewire (Meister; Asahi Intecc). Tris-acryl microspheres (Embosphere; Merit Medical, South Jordan, Utah) with a diameter of 40–120 μ m were used in the first 4 cases. Imipenem/cilastatin sodium (IPM/CS) suspension was used in the following 11 cases as an embolic agent. IPM/CS suspension was prepared by

Table. Summary of Clinical Manifestations and Treatment Outcomes

Pt. No./ Sex/Age (y)	Body Part	Pain Duration (mo)	Initial VAS	Clinical Diagnosis	Previous Treatment (n)	Target Arteries	Embolitic Agent	Embolitic Volume (mL)	Staining	Procedure Duration (min)	VAS Decrease	Treatment at Final Follow-up (4 mo)
1/F/48	Elbow	13	5	Lateral epicondylitis	ESWT (4), CSI (5), NSAID	Right RRA	Embosphere	1.8	NE	32	3	None (NSAID for 3 mo after treatment)
2/F/48	Elbow	7	5	Lateral epicondylitis	CSI (2), tramadol	Left RRA	Embosphere	0.5	Evident	22	3	NSAID
3/M/75	Shoulder	16	5	Rotator cuff tendinopathy	CSI (5), tramadol	Left TAA	Embosphere	5.2	Evident	40	4	None (tramadol for 3 mo after treatment)
4/F/27	Elbow	9	7	Lateral epicondylitis	CSI (1), NSAID	Left RRA	Embosphere	1.9	Evident	40	5	None (NSAID for 1 mo after treatment)
5/F/56	Elbow	18	7	Lateral epicondylitis	CSI (2), NSAID	Right RRA, Muscular branch	IPM/CS	5.8	Evident	60	5	None (NSAID for 1 mo after treatment)
6/F/67	Shoulder	8	3	Rotator cuff tendinopathy	Tramadol	Right PCHA, CSA	IPM/CS	2.5	NE	55	2	None (tramadol for 1 mo after treatment)
	Elbow	6	6	Lateral epicondylitis	ESWT (1), tramadol	Right RRA	IPM/CS	1.2	Evident		5	
7/F/75	Shoulder	13	5	Rotator cuff tendinopathy	CSI (6), tramadol	Right TAA, PCHA	IPM/CS	7.8	Evident	41	0	CSI, Tramadol
8/F/47	Shoulder	68	7	Calcific tendinitis	ESWT (3), CSI (7), Tramadol	Right PCHA	IPM/CS	4.6	Evident	76	5	None (tramadol for 2 mo after treatment)
9/M/40	Shoulder	22	7	Rotator cuff tendinopathy	ESWT (1), CSI (4), tramadol	Left PCHA	IPM/CS	5.4	Evident	28	7	None (tramadol for 3 mo after treatment)
10/F/50	Shoulder	9	6	Rotator cuff tendinopathy	CSI (2), tramadol	Right TAA, CSA	IPM/CS	6.3	Evident	71	4	Tramadol (for elbow pain)
	Elbow	7	6	Lateral epicondylitis	Tramadol	Right RRA	IPM/CS	3.8	NE		0	
11/F/57	Elbow	12	7	Lateral epicondylitis	CSI (2), tramadol	Right RRA	IPM/CS	4.4	NE	30	4	None (tramadol for 2 mo after treatment)
12/M/31	Shoulder	24	7	Rotator cuff tendinopathy	CSI (8), tramadol	Left PCHA	IPM/CS	4.2	NE	37	0	CSI, tramadol
13/F/60	Shoulder	19	8	Calcific tendinitis	ESWT (3), CSI (4)	Left CSA, PCHA	IPM/CS	10.6	Evident	55	7	None (pt. had ulcerative colitis)

CSA = circumflex scapular artery; CSI = corticosteroid injection; ESWT = extracorporeal shockwave therapy; IPM/CS = imipenem/cilastatin sodium; NE = not evident; NSAID = nonsteroidal anti-inflammatory drug; PCHA = posterior circumflex humeral artery; RRA = recurrent radial artery; TAA = thoracoacromial artery; VAS = visual analog scale.



Figure 1. Left axillary arteriographic imaging before and after transcatheter arterial embolization with IPM/CS in a 60-year-old woman (patient 13; [Table](#)) with chronic shoulder pain (19 mo) caused by calcific tendinitis. **(a)** Preembolization arteriography shows evident enhancement (asterisk) in the region of the supraspinatus tendon supplied by the circumflex scapular artery (white arrows) and the posterior circumflex humeral artery (black arrows). **(b)** Postembolization arteriography shows disappearance of abnormal enhancement. At 4-month follow-up, VAS score decreased from 8 to 1. No additional treatment was applied.

mixing 500 mg of IPM/CS (500 mg Prepenem injection; JW Pharmaceutical, Seoul, Korea) with 5 mL iodixanol 320 mgI/mL (Visipaque; GE Healthcare, Little Chalfont, United Kingdom) via a pumping method with multiple passages (20 times). The suspension was injected in 0.2-mL increments followed by flushing with the same amount of normal saline solution. The endpoint of embolization was complete or near-stasis of the feeding artery without reflux of embolic agent to undesired arteries. Patients were observed for adverse events and then discharged the day after the procedure. Oral pain medication taken before the embolization procedure was prescribed to patients for pain medication after discharge. Patients resumed activities freely 1 day after the procedure.

Assessment and Follow-up

Clinical information and imaging data of patients were reviewed from the electronic medical record and picture archiving and communication system. Technical success was defined as superselective catheterization and embolization of at least 1 feeding artery to the enhanced lesion in the shoulder or elbow area. Patients were assessed for pain after the embolization procedure by using a 10-point visual analog scale (VAS), with 0 indicating no pain and 10 indicating the maximum pain intensity. The VAS scores were assessed at baseline, 1 day, 1 week, 1 month, and 4 months after embolization. The use of previous or new conservative treatment was also assessed. Clinical success was defined as improvement in pain (a decrease in VAS score of more than 50% vs baseline) and discontinuation of pain medication at 4 months after embolization.

Abnormal enhancement was defined as tumor blush–type enhancement that appeared in the arterial phase as described in previous studies (16,20). The degree of abnormal enhancement of the lesion on angiographic images was dichotomized into “evident” enhancement and lack thereof. Evident enhancement had strong staining with obvious margins, whereas no evident enhancement showed poor staining with obscure margins. Two independent interventional radiologists (S.W.P. and J.H.H.) assessed the degree of enhancement by evaluating all series of angiographic images. Interobserver differences were resolved by consensus. Changes in VAS scores according to the degree of enhancement were also evaluated.

Adverse events were assessed according to the Society of Interventional Radiology classification (21). Newly developed pain, muscle weakness, paresthesia, and skin changes were also recorded.

Statistical Analysis

Categorical variables are expressed as percentages, and continuous variables are expressed as means and standard deviations. The Wilcoxon signed-rank test was used to compare baseline and follow-up VAS scores. The level of interobserver agreement for the degree of enhancement was determined by calculating a κ coefficient.

The Student *t* test was used to analyze the differences in pain improvement according to the degree of enhancement of the lesion. A *P* value of less than .05 was considered statistically significant. All statistical analyses were performed by using SPSS software (version 17.0; SPSS, Chicago, Illinois).



Figure 2. Left brachial arteriographic imaging before and after transcatheter arterial embolization with microspheres in a 48-year-old woman (patient 2; [Table](#)) with chronic elbow pain (7 mo) as a result of lateral epicondylitis. **(a)** Preembolization arteriography shows evident enhancement (white arrows) fed by the recurrent radial artery (black arrows) in a region adjacent to the lateral epicondyle. **(b)** Postembolization arteriography shows disappearance of hyperenhancement adjacent to the lateral epicondyle. At 4-month follow-up, the patient continued the pretreatment medication regimen despite a reduction in VAS score from 5 to 2.

RESULTS

Technical success was achieved in 100% of cases (15 of 15). The mean procedure time was 45.2 minutes \pm 16.8 (range, 22–76 min). A decrease in VAS score was noted in 12 of 15 cases (80%). Clinical success was noted in 11 of 15 cases (73%) at 4 months after embolization. One patient with elbow pain (patient 2; [Table](#)) continued to receive the preprocedural medication regimen despite more than 50% reduction in pain after embolization.

The mean VAS scores at baseline, 1 day, 1 week, 1 month, and 4 months after embolization were 6.1 ± 1.3 , 5.8 ± 1.4 , 5.1 ± 1.6 , 4.3 ± 2.0 , and 2.5 ± 2.0 , respectively. The mean VAS scores at 1 week after embolization and thereafter were significantly ($P < .05$) lower than those before embolization. Among the 12 cases with pain relief, 7 patients (58%) experienced quick pain relief within 1 week and 9 (75%) experienced pain relief within 1 month. The remaining 3 cases experienced pain relief at 1–4 months after embolization.

There was interobserver agreement ($\kappa = 0.857$; $P < .001$) for the degree of angiographic enhancement. Pain score improved in 9 of 10 cases with evident enhancement ([Figs 1, 2](#)), compared with only 3 of 5 cases in which there

was no evident enhancement ([Fig 3](#)). The VAS score in the evident enhancement group was decreased more than that in the group with no evident enhancement (4.5 vs 1.8; $P < .05$).

No major adverse events were reported following the embolization procedure. Forearm cutaneous erythema was noted in 1 patient (patient 4; [Table](#)) after embolization with the use of microspheres to treat elbow pain. The erythema resolved within 1 month in that patient without specific treatment. There was no report of newly developed or aggravated pain, muscle weakness, or paresthesia.

DISCUSSION

Although more basic studies are needed, pain relief achieved by treatment with transcatheter arterial embolization can be explained by 2 mechanisms. As angiogenesis is required for the development and maintenance of inflammation, abnormal and increased vessels on pretreatment angiography would suggest ongoing inflammation ([16,22](#)). Therefore, occlusion of abnormal vessels could decrease the influx of inflammatory cells and proinflammatory cytokines, and that would result in reduction of the inflammatory

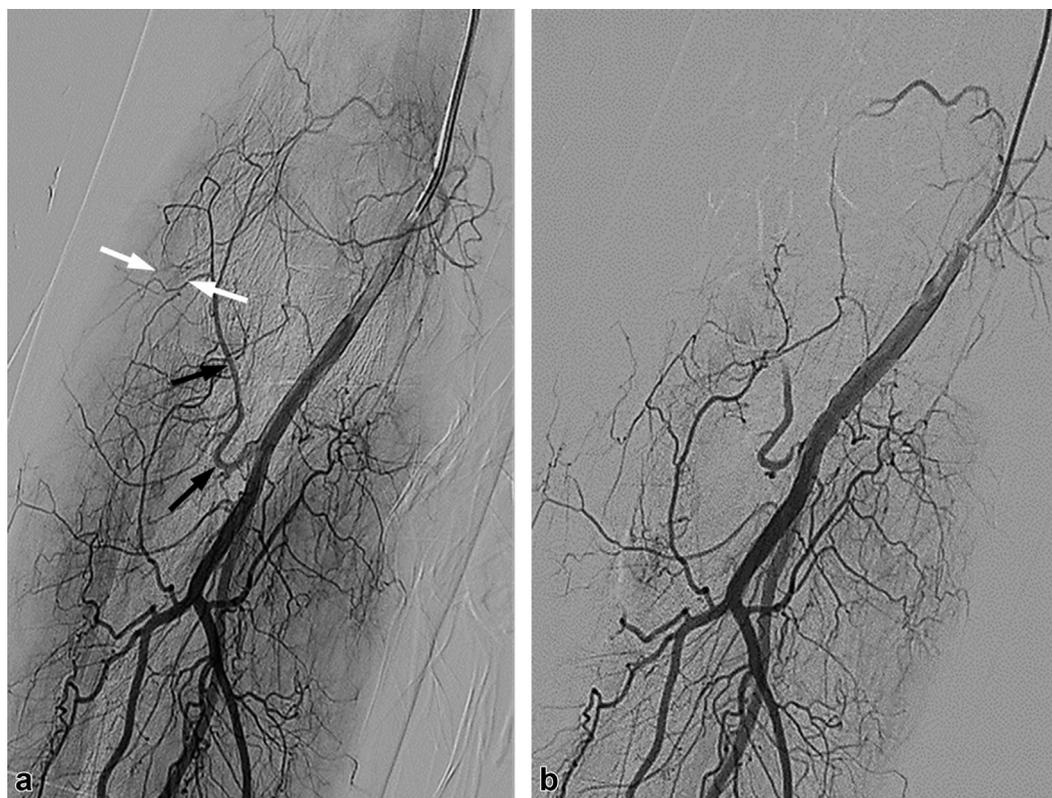


Figure 3. Right brachial arteriographic imaging before and after transcatheter arterial embolization with IPM/CS in a 50-year-old woman (patient 10; [Table](#)) with chronic elbow pain (7 mo) as a result of lateral epicondylitis. **(a)** Preembolization arteriography shows no evident enhancement (white arrows) in a region adjacent to the lateral epicondyle fed by the recurrent radial artery (black arrows) arising from the brachial artery. **(b)** After embolization of the recurrent radial artery, subtle enhancement (ie, not classified as evident) adjacent to the lateral epicondyle disappeared. At 4-month follow-up, there was no change in VAS score compared with the baseline score (6 vs 6).

process (16). The other mechanism is that stimulation of new unmyelinated sensory nerve growth near abnormal blood vessels affected by angiogenesis could be reduced after embolization (16,23). In a previous study (24) regarding immunohistochemical analysis of Achilles tendinosis, the tissue showed positivity for substance P and calcitonin gene-regulated peptide, which are associated with pain or pain transmission near blood vessels, suggesting a possible association between pain and blood flow. Pufe et al (25) demonstrated that increased vascularity might be involved in the pathogenesis of degenerative tendinopathy. Okuno et al (18) proposed that decreasing blood flow at sites of pathologic processes might relieve pain and prevent further tissue degeneration. Moreover, these authors showed practical evidence for pain relief of tendinopathy in various parts of the body after transcatheter arterial embolization (18). In the present study, 11 of 15 body parts (73%) with chronic shoulder or elbow pain associated with tendinopathy showed clinical success. In addition, more than a 50% improvement in VAS score versus baseline was achieved in 12 body parts at 4 months after embolization. One patient with elbow pain (patient 2; [Table](#)) continued the pretreatment medication regimen despite more than 50% reduction in pain after embolization.

In one study conducted by Okuno et al (18), pain relief was achieved in all 7 patients with tendinopathy. Although

inferior to these results, the present study reports a clinical success rate of 73%, which may be acceptable considering that all 13 patients involved in the study had tendinopathy refractory to conservative treatments such as pain relievers, corticosteroid injections, and ESWT. Considering that the failure rates associated with surgical treatment for tendinopathy are inconsistent (9,11–14), transcatheter arterial embolization might be a helpful option for patients with chronic shoulder or elbow pain related to tendinopathy before considering surgery if they do not benefit from conservative treatment.

There may be a concern that transcatheter arterial embolization for benign musculoskeletal disorder might bring about unexpected adverse results. However, according to the midterm results of a prospective single-center study of transcatheter arterial embolization for adhesive capsulitis (16), there were no symptom recurrences or late-onset adverse events. These authors also reported continuous improvement in the range of motion and shoulder function. Another study of transcatheter arterial embolization in 7 patients with tendinopathy and enthesopathy (18) has also shown excellent pain relief lasting for 4 months. Additionally, in 1 study of midterm clinical outcomes of transcatheter arterial embolization in knee osteoarthritis (20), cumulative clinical success rates at 6 months and 3 years were reported to be 86.3% and 79.8%, respectively. Although the present

study was focused on short-term results (4 mo), there was no evidence of pain aggravation, and there was steady pain reduction over time among the 12 cases with pain relief after embolization. Therefore, transcatheter arterial embolization might not inhibit the natural recovery process of tendinopathy. However, further research is needed to prove this assumption.

In previous publications (16,20), abnormal vessels seen during embolization for adhesive capsulitis were characterized as having a tumor blush-type enhancement. These are found on the arterial phase, often accompanied by early venous filling. However, we are aware of no study that has evaluated the difference in pain relief according to the degree of angiographic enhancement. Considering that abnormal neoangiogenesis is an important factor in inflammation, we hypothesized that greater alleviation of pain could be achieved after embolization in patients with more severe abnormal neovascularization. In the present study, all 15 patients showed abnormal enhancement of a lesion on digital subtraction angiography, including evident enhancement in 10 cases and no evident enhancement in 5. Improvement of pain was noted in 9 of 10 cases (90%) with evident enhancement. However, only 3 of 5 cases (60%) with no evident enhancement showed pain improvement. In addition, VAS scores in the evident enhancement group were decreased more ($P < .05$) than those in the group with no evident enhancement. These results suggest that the degree of lesion enhancement on digital subtraction angiography might be a factor to predict the degree of pain relief after embolization. However, further study is needed to clarify this possibility.

IPM/CS has been approved by the Food and Drug Administration as an antibiotic agent. IPM/CS is slightly soluble in water, but, when it is mixed with a contrast agent, crystalline particles will form, which can induce temporary embolization (18). Woodhams et al (26) used 0.5 g IPM/CS suspended in 5 mL contrast agent as an embolic agent (10–70 μm) for embolization of gastrointestinal tumor bleeding from the superior mesenteric artery of swine. Its use resulted in embolization with less necrosis compared with gelatin sponge suspension. Okuno et al (16–20) have also chosen IPM/CS as an embolic agent to embolize neovessels of musculoskeletal components because they consider that IPM/CS is safer than traditional embolic agents such as gelatin sponges or microspheres. They reported transient cutaneous color changes in 4 of 7 patients treated with microspheres during embolization for knee osteoarthritis (20). There were no complications reported in the treated area (eg, peripheral paresthesia, muscle weakness, tissue necrosis, and skin changes) in 25 cases with adhesive capsulitis (16), 7 cases with tendinopathy (18), or 88 cases with knee osteoarthritis (20) when IPM/CS was used as embolic agent. In the present study, 1 of the first 4 cases treated with microspheres as an embolic agent showed erythematous change on the forearm skin at 1-week follow-up. Although this skin change was improved at 1-month follow-up without any specific treatment, we decided to use IPM/CS

for the subsequent procedures. Adverse events did not occur thereafter. This suggests that IPM/CS seems to be safer than traditional embolic agents, consistent with the suggestion of Okuno et al (16–20). However, considering the small number of patients included in the present study and the absence of randomized controlled studies or large studies, further evaluation is needed to determine whether IPM/CS is safer than traditional embolic agents.

The present study has several limitations. First, this study was a retrospective and not a comparative study, with a small number of patients. Second, follow-up was available for only 4 months. Midterm or long-term follow-up results were not obtained. Third, there was a lack of uniformity in the embolic agent used. Finally, patients were allowed to continue their medication after embolization if the pain persisted. Continued medication might have affected outcomes regarding pain relief after embolization.

In conclusion, transcatheter arterial embolization might deserve consideration as a treatment option to relieve pain related to chronic shoulder and elbow tendinopathies refractory to conservative treatment. The degree of angiographic enhancement might be a possible factor affecting the degree of pain relief after embolization.

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