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Short-term results of transcatheter arterial embolization for abnormal neovessels in patients with adhesive capsulitis: a pilot study

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Background: Neovessels and accompanying nerves are possible sources of pain. We postulated that transcatheter arterial embolization of abnormal neovessels would relieve pain and symptoms in patients with adhesive capsulitis.

Methods: Adhesive capsulitis was treated by transcatheter arterial embolization in 7 patients. Adverse events, changes in visual analog scale scores for night pain and overall shoulder pain, and changes in range of motion and American Shoulder and Elbow Surgeons scores were assessed at 1 week and at 1, 3, and 6 months after the procedure.

Results: Abnormal neovessels were identified at the rotator interval in all patients. No major or minor adverse events were associated with the procedures. Transcatheter arterial embolization rapidly decreased nighttime pain scores from 67 ± 14 mm to 27 ± 14 mm at 1 week after the procedure, with further improvement at 1 and 6 months (6 ± 8 mm and 2 ± 5 mm, respectively). The American Shoulder and Elbow Surgeons score significantly improved from 17.8 ± 4.5 to 39.8 ± 12.0, 64.3 ± 13.9, and 76.2 ± 4.4 at 1, 3, and 6 months, respectively.

Conclusion: All patients with adhesive capsulitis had abnormal neovessels at the rotator interval. Transcatheter arterial embolization was feasible, relieved unrelenting pain, and restored shoulder function.

Level of evidence: Level IV, Case Series, Treatment Study.

Keywords: Shoulder pain; adhesive capsulitis; abnormal vessels; embolization

Adhesive capsulitis is a condition of uncertain etiology that is characterized by painful restriction of active and passive shoulder motion. The estimated prevalence of adhesive capsulitis is 2% to 3% in the general population[26]

The Institutional Review Board of Edogawa Hospital approved this study (assigned study number 2012-07).

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and 5% to 6% in patients who have been evaluated by a shoulder surgeon.[17]

The overall objective of treating adhesive capsulitis is to relieve pain and restore motion. Treatment regimens for adhesive capsulitis begin with a trial of conservative therapy, including anti-inflammatory medications, injections, and physical therapy.[18] However, residual pain is one of the most important issues in the treatment of adhesive capsulitis. After nonsurgical treatment, 27% of patients had mild or moderate pain at 1.8 years' follow-up,[14] and another study reported that 35% of patients had mild pain at 7 years.[27] When conservative therapy is not effective, more invasive approaches are sometimes required, such as capsular distention, manipulation under anesthesia, or arthroscopic capsular release. Nevertheless, a consensus has not been reached on the optimal treatment for adhesive capsulitis that is resistant to traditional conservative treatments.

Despite the large number of patients affected by adhesive capsulitis, the source of pain has not been defined. Many investigators have described hypervascularity at the rotator interval with adhesive capsulitis. Open surgery and arthroscopic observation have confirmed that angiogenesis most commonly affects the rotator interval.[7,10,30,33] Some groups have histologically confirmed obviously increased vascularity in fibrotic tissues from the rotator interval of patients with adhesive capsulitis.[10,28] Bunker and Anthony[7] described 12 patients who did not have improvement after manipulation. The patients were treated by surgical release of the coracohumeral ligament, and this tissue was hypervascular in all of them. Ryu et al[25] described more intense immunostaining for vascular endothelial growth factor and CD34 (general marker for blood vessels) in the synovium of the rotator interval in patients with diabetic adhesive capsulitis compared with a control group. They postulated that secreted vascular endothelial growth factor might induce adhesive capsulitis. However, the relationship between this abnormal vasculature and symptoms of adhesive capsulitis has not been investigated.

Abnormal neovessels and accompanying nerves are possible sources of pain. Alfredson and colleagues[1,5] showed vascular/neural ingrowth in areas of pain associated with chronic painful tendinopathy and immunohistochemically identified substance P–positive nerves located near the newly formed blood vessels in tendinopathy tissue. Gotoh et al[13] measured levels of substance P in the subacromial bursa of patients with rotator cuff disease and found that the amount of nerve fibers immunoreactive to substance P was increased around the vessels.

Transcatheter arterial embolization is used to occlude blood flow by intra-arterially infusing an embolic agent through a catheter located at target vessels. On the basis of the notion that increased numbers of blood vessels and accompanying nerves are a possible source of pain and that occlusion of abnormal vessels might reduce such pain, We previously applied transcatheter arterial embolization in patients with painful tendinopathy and enthesopathy refractory to traditional nonsurgical management.[21] We located abnormal neovessels at pathologic sites in all patients and showed that embolization of these abnormal vessels resulted in excellent pain relief.

We postulated that abnormal neovessels at the rotator interval play an important role in adhesive capsulitis and that transcatheter arterial embolization of such neovessels could relieve pain associated with this condition. Therefore, this study prospectively evaluated the feasibility and effectiveness of transcatheter arterial embolization in patients with adhesive capsulitis refractory to traditional nonsurgical management.

Materials and methods

This is a prospective study of transcatheter arterial embolization for patients with adhesive capsulitis resistant to conservative therapies. All patients received an explanation about various management modalities and the potential risks, benefits, and outcomes of transcatheter arterial embolization and then provided written informed consent to undergo the procedure. The criteria for inclusion included night shoulder pain; painful restriction of both active and passive elevation to less than 100° and of external rotation to less than 50% of the contralateral side; normal radiologic findings; previous conservative therapies comprising rest, anti-inflammatory drugs, corticosteroid injections, and physical therapy for at least 3 months; persistent moderate to severe pain (visual analog scale [VAS] >50 mm); and completion of a 6-month follow-up period. All patients were assessed by ultrasound or magnetic resonance examinations, and those with full-thickness rotator cuff tears were excluded. Other exclusion criteria included local infection, malignancy, advanced atherosclerosis, rheumatoid arthritis, and prior shoulder surgery. All patients in the final resolving stages were excluded. We enrolled 7 patients (5 women and 2 men; mean age, 50.3 ± 10.4 years; age range, 39-68 years) in this study. Table I shows the baseline patient demographic and clinical data. The mean duration of symptoms was 6.6 ± 3.4 months (range, 3-12 months). All patients had been receiving nonsurgical treatments for symptoms that had persisted for more than 3 months (Table I). All patients had preprocedural physical findings of tenderness at the coracoid process.

All subsequent techniques proceeded with the patients under local anesthesia. We gained arterial access percutaneously using a 3-French introducer sheath (Super Sheath; Medikit, Tokyo, Japan). We selected the radial and femoral arteries as puncture sites in 5 patients and 2 patients, respectively. After the intravenous administration of 2000 IU of heparin sodium (Mitsubishi Tanabe Pharma, Osaka, Japan), we inserted a 3-French angiographic catheter (Multipurpose; Medikit) toward the axillary artery and acquired images using an Allura Xper FD10 angiography system (Philips Healthcare, Best, The Netherlands). Digital subtraction angiography then proceeded by manual injection of 3 to 5 mL of iodinated contrast medium (Hexabrix; Terumo, Tokyo, Japan). We examined the suprascapular artery, thoracoacromial artery, and anterior circumflex humeral artery, as well as the direct branch from the axillary artery, in all patients. Normal blood vessels were defined as tubular anatomic structures in the expected distribution of the known vascular anatomy, as well as normal variants (Fig. 1, *A*). Small arterial branches are undetectable in the normal shoulder. Abnormal neovessels were defined as new periarticular smaller vessels if an additional arterial supply was derived from the normal arteries of the shoulder.[29] After the abnormal