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| Review Article |

Partial-Thickness Rotator Cuff Tears

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Although the incidence of partial-thickness rotator cuff tears (PTRCTs) was reported to be from 13% to 32% in cadaveric studies, the actual incidence is not yet known. The causes of PTRCTs can be explained by either extrinsic or intrinsic theories. Studies suggest that intrinsic degeneration within the rotator cuff is the principal factor in the pathogenesis of rotator cuff tears. Extrinsic causes include subacromial impingement, acute traumatic events, and repetitive microtrauma. However, acromially initiated rotator cuff pathology does not occur and extrinsic impingement does not cause pathology on the articular side of the tendon. An arthroscopic classification system has been developed based on the location and depth of the tear. These include the articular, bursal, and intratendinous areas. Both ultrasound and magnetic resonance image are reported with a high accuracy of 87%. Conservative treatment, such as subacromial or intra-articular injections and suprascapular nerve block with or without block of the articular branches of the circumflex nerve, should be considered prior to operative treatment for PTRCTs. (Korean J Pain 2011; 24: 69-73)

Key Words:

injections, intrinsic, nerve block, rotator cuff.

INTRODUCTION

Shoulder pain is the third most common musculoskeletal complaint and 1% of adults visit their physicians with new shoulder pain each year [1]. The most common shoulder ailments include impingement syndrome, frozen shoulder, bicipital tendinitis, acromioclavicular arthritis, and glenohumeral arthritis. Impingement syndrome refers to symptoms that result from compression of either the rotator cuff muscles or the subdeltoid bursa.

Although Neer [2] reported that most partial-thickness rotator cuff tears (PTRCTs) correlate with external rotator cuff impingement, the currently accepted theory is that PTRCTs derive from intrinsic tendon degeneration [3]. Like other shoulder diseases, PTRCTs are closely related to age and are especially common in diabetic patients. Even in the absence of high-quality data on the treatment of PTRCTs, the majority of these conditions are amenable to conservative therapy.

INCIDENCE

Although an accurate incidence of PTRCTs is unknown, an estimation has been made from numerous

imaging and cadaveric studies. Sher et al. [4] reported that 4% of individuals younger than 40 years old and more than 50% of individuals older than 60 without shoulder pain showed partial and full thickness rotator cuff tears on magnetic resonance imaging (MRI). Löhr and Uhthoff [5] found an incidence of 19% for full thickness tears and 32% for partial thickness tears in 306 cadaveric studies. Fukuda [6] reported 13% partial thickness supraspinatus tears, with 18% bursal side partial thickness supraspinatus tears, 55% intratendinous partial thickness supraspinatus tears, and 27% articular-sided partial thickness supraspinatus tears in 249 cadaveric studies.

PATHOPHYSIOLOGY

The causes of PTRCTs may be explained by either extrinsic or intrinsic theories. Intrinsic causes include age-related, metabolic, and vascular changes which lead to degenerative tearing and differential shear stress within the tendon leading to intratendinous tears [7]. Hashimoto et al. [8] performed histopathologic, histochemical, and morphometric studies on 80 medical stumps of torn rotator cuff tendons to clarify the causes of tears. A high prevalence and diffuse distribution of degenerative changes were observed in the rotator cuff tendons, including thinning and disorientation of collagen fibers, myxoid degeneration, hyaline degeneration, chondroid metaplasia, calcification, vascular proliferation, and fatty infiltration. Evidence suggests that intrinsic degeneration within the rotator cuff is the principal factor in the pathogenesis of rotator cuff tears [8]. Nho et al. [9] also found decreased cellularity, fascicular thinning and disruption, accumulation of granulation tissue, and dystrophic calcification. The causes of PTRCTs may result from intrinsic failure in the articular side of the tendon. It may lead to superior translation of the humeral head and irritation of the bursal tissue, increasing the strain on the coracoacromial ligament and the acromion.

Extrinsic causes include subacromial impingement, acute traumatic events, and repetitive microtrauma [10]. The extrinsic theory behind subacromial impingement suggests cuff tendinopathy resulting from impingement of the rotator cuff against subacromial osteophytes or the coracoacromial ligaments [11]. However, Ozaki et al. [7] found, in their cadaveric study, that the undersurface of the acromion was almost always intact in articular-sided tears.

Although acromioplasty has been reported with success rates of 80−90% [12,13], it produced no clinical improvement at 6 months or more compared to conservative management [14,15]. Hyvönen et al. [16] reported that acromioplasty does not prevent the progression of impingement syndrome on a rotator cuff tear. Gill et al. [17] revealed no significant association between acromial morphology and rotator cuff pathology in patients who were over 50 years of age. Ogata and Uhthoff [3] reported that the majority of PTRCTs were intrasubstance or articular side tendon tears and not on the upper bursal side of the tendon adjacent to the acromion. Considering the results of these studies, the authors propose that acromially initiated rotator cuff pathology does not occur and extrinsic impingement does not cause pathology on the articular side of the tendon.

CLASSIFICATION

Ellman [18] developed a classification system based on the location (A: articular, B: bursal, C: intratendinous) and depth (grade 1: < 3 mm, grade 2: 3−6 mm, grade3: < 6 mm) of the tear based on arthroscopic findings. Among 130 patients who had arthroscopic subacromial decompression, 20 patients with PTRCTs were encountered [18]. Another classification system was proposed by Snyder et al. [19] based on tear location and size (0−4, normal to tear > 3 cm) in 31 patients who underwent bursoscopy. Conway [20] described a shoulder pain common in athletes who engage in excessive throwing, which is referred to as PAINT (partial articular tears with intratendinous extension).

CLINICAL FINDINGS

Typical symptoms of PTRCTs are shoulder pain, stiffness, or dysfunction on the affected side. Patients often report a painful arc of motion, resting pain, and night pain. Examination findings include supraspinatus weakness, weakness in external rotation and signs of impingement. Patients over 60 years old with only 2 of the above mentioned findings still have a 98% chance of rotator cuff tear [21].

Fukuda [6] reported that patients with PTRCTs complain of more severe pain compared to patients with full thickness tears. Bursal-sided tears were reportedly more

painful than articular-sided tears [6]. Neer's test, Hawkins' test, the empty can test or the full can test may help diagnose PTRCTs. However owing to their relative nonspecificity, the tests may yield positive results for other pathologic entities, such as acromioclavicular joint arthritis or biceps tendinitis.

IMAGING STUDIES

Anteroposterior radiographs show that large rotator cuff tears are associated with an elevation of the humeral head, especially with decreased subacromial space to less than 5-7 mm. An axillary view is helpful in excluding a shoulder dislocation. The supraspinatus outlet view allows visualization of the bony structures of the scapulohumeral motion interface and shows acromial spurs or calcification of the coracoacromial ligament that might compress the underlying rotator cuff.

Ultrasound is a noninvasive, readily available, and inexpensive diagnostic tool but with a variable correlation with MRI, ranging from 58-83% [22,23]. Brenneke and Morgan [24] preoperatively analyzed 120 patients with ultrasonography and compared this with arthroscopic findings to reveal a 95% sensitivity in full thickness tears but only a 41% sensitivity in partial thickness tears. Preoperative ultrasonographic findings of a mixed hyperechoic/hypoechoic focus in the supraspinatus tendon was found to have a sensitivity of 93%, a specificity of 94%, a positive predictive value of 82%, and a negative predictive value of 98% (van Holsbeeck et al. [25]). Milgrom et al. [26] reported a prevalence of full and partial thickness tears of 5% to 11% in asymptomatic patients under 50 years of age. These tears markedly increased after 50 years of age to reach 80% in patients over 80. Therefore, treatment should be based on clinical findings rather than image findings.

MRI has become the imaging modality of choice for most clinicians and investigators with a high sensitivity of 38% to 84% reported [27,28]. Meister et al. [27] found preoperative gadopentate contrast MR arthrography to have a sensitivity of 84%, a specificity of 96%, a positive predictive value of 93%, and an overall accuracy of 91%. To overcome shortcomings of direct MR arthrography, noninvasive indirect MR arthrography was reported to have lower sensitivities of between 38-50% and specificities of 86-88% in the diagnosis of PTRCTs [28]. Connor et al. [29] evaluated 20 aymptomatic overhead athletes with MRI to reveal 40% full or partial thickness tears on the dominant shoulders. None of them had subjective symptoms or required any treatment after 5 years. The researchers concluded that MRI alone should not be used as a basis for operative intervention.

TREATMENT

The choice of therapy is guided by specific patient factors such as age, pre-injury functional level and demand, and general health. As most of PTRCTs involve less than 50% of the rotator cuff and do not lead to muscle retraction, the treatment of rotator cuff tears always starts conservatively. A broad spectrum of conservative treatments for PTRCTs is available; rest, non steroidal antiinflammatory drugs, various injections, nerve blocks, physical therapy, manual therapy, prolotherapy, platelet rich plasma (PRP) injection, and intramuscular stimulation (IMS). Evidence to support the efficacy of therapeutic exercise and manual therapy is limited. Randomized controlled trials for treatments such as PRP, prolotherapy, and IMS have not yet been done to provide possible evidence for differences in outcomes.

Neural blockades, such as suprascapular nerve block with or without block of the articular branches of the circumflex nerve, produce substantial and lasting pain relief by blocking afferent C- and A delta-fibers and subsequent depletion of the neurotransmitter substance, P, and the nerve growth factor in the synovium and afferent C-fibers of the glenohumeral joint. A decrease in central sensitization of dorsal horn nociceptive neurons, or 'wind-down', with a reduction of peripheral nociceptive input following neural blockade also contributes to pain relief [30]. Various suprascapular nerve block techniques exist, such as the anterior approach developed by Wassef [31] in addition to the traditional posterior approach. The accuracy of the injection varies from 29% to 87% [32]. More accurate injections with a greater improvement of symptoms can be achieved using sono-guidance [33,34]. Subacromial injection is useful for bursal-sided PTRCTs but not for articular-sided PTRCTs. In contrast, intra-articular injection shows better pain relief for patients with articular-sided PTRCTs or PTRCTs accompanied by other conditions such as capsulitis.

Functional demands and comorbidities of individual patients should be considered before deciding on an oper-

ative treatment. In younger patients, restoring anatomy to maximize strength and function should be the focus, whereas minimizing surgical risk and relieving pain should be the focus in older patients. The onset of acute, post-traumatic weakness in young patients without pre-existing rotator cuff dysfunction is generally accepted as an absolute indication for surgical repair. Repair of tears is generally recommended only for patients with tears extending for more than 50% of the tendon thickness. Surgical intervention may be considered when conservative treatment fails. However, no evidence from high quality randomized controlled trials exists to show whether surgery provides better results than conservative treatments for these patients.

CONCLUSION

The pathogenesis of PTRCTs displays intrinsic more than extrinsic degeneration. Therefore, sufficient conservative treatment should be given, especially to older patients unlike young patients with acute, posttraumatic weakness. As abnormal findings on imaging studies are relatively common in asymptomatic patients, decisions on treatment should be based on clinical findings.

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