

Upper Extremity

The natural history of rotator cuff tears

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ABSTRACT

Rotator cuff disease is one of the most prevalent musculoskeletal disorders treated by orthopaedic surgeons. Despite its prevalence, the natural history of the disease is not completely understood, which is critical to consider when making decisions on surgical and nonsurgical treatment of rotator cuff tears. This article reviews the most recent studies on the natural history of rotator cuff disease and provides the clinician with guidelines on whether surgical or nonsurgical management is indicated.

Keywords

fatty atrophy, impingement, natural history, rotator cuff, rotator cuff arthropathy, tendon degeneration

INTRODUCTION

Knowledge of the natural history of rotator cuff disease is critical when making treatment decisions. The timing of changes that occur within the shoulder after rotator cuff tear may serve as a guideline as to if and when surgical repair may be indicated. This article reviews the most recent studies on the natural history of rotator cuff disease in an attempt to optimize clinical decision-making.

PREVALENCE

There is an age-dependent increase in rotator cuff tearing. Although the prevalence of full-thickness rotator cuff tears ranges from 7–40%, studies using either MRI or ultrasound have shown that a high percentage of these tears may be asymptomatic.^{1–4} An MRI study by Sher *et al.*¹ found a 34% rate of full-thickness tears in 96 asymptomatic volunteers. When looking only at patients over the age of 60 years, the prevalence increased to 54%. A later study, using ultrasound in 411 asymptomatic volunteers, found an age-related increase in full-thickness tears from 13% in patients between the ages of 50–59 years to a remarkable 51% in patients older than 80 years.² A more recent ultrasound study of 420 asymptomatic volunteers between the ages of 50–79 years

showed that the rate of full-thickness tears increased from 2.1% in patients during their sixth decade to 5.7% during their seventh decade. In patients between the ages of 70–79 years, the prevalence further increased to 15%.³ Although this study shows a lower prevalence than some previous work, it generally is agreed upon that there is a high rate of asymptomatic rotator cuff tears in patients over the age of 60 years. Even in the presence of a large tear, asymptomatic patients have well preserved shoulder function.^{4*}

IMPINGEMENT AND TENDON DEGENERATION

There are two major theories for the development of rotator cuff disease, the extrinsic and intrinsic theories. The extrinsic theory implies that factors external to the tendons are the primary cause of the disorder. Neer⁵ coined the term "impingement syndrome," which implicated the anterior acromion, the coracoacromial ligament, and the acromioclavicular joint. Impingement was classified into three stages: tendon inflammation, fibrosis, and cuff tear. Bigliani *et al.*⁶ later described three acromial shapes: type 1 (flat), type 2 (curved), type 3 (hooked). Of the specimens he reviewed with rotator cuff tears, 75% had type 3 morphology.

It later was realized that internal impingement affects the younger athletic population and is secondary to instability rather than the true outlet impingement described by Neer.⁵ Walch *et al.*⁷ introduced the concept of internal impingement and described it as the contact of the rotator cuff on the posterior superior glenoid when the arm is in the abducted and externally rotated position. They concluded that internal impingement can lead to articular side rotator cuff tear involving the posterior aspect of the supraspinatus and anterior aspect of the infraspinatus tendons.

The intrinsic theory implies that tendon degeneration is the result of changes in the mechanical properties of the tendons. Aging and poor vascularity have been implicated in tendon degeneration. In a study by Rudzki *et al.*,⁸ a statistically significant decrease in blood flow to the intact rotator cuff was seen in subjects older than age 40 years compared with those younger than 40 years. In particular, the medial articular side of the rotator cuff was found to be a hypovascular zone. Although not proven, this area of decreased blood flow may play a role in the degeneration and subsequent tearing of the rotator cuff often seen in this area.

An injured rotator cuff is no longer able to generate the normal compressive forces across the glenohumeral joint. This causes an imbalance to occur between the rotator cuff and deltoid, which can result in impingement with elevation

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of the arm. The loss of scapular stabilization also can lead to the loss of normal scapular rotation and subsequent impingement. Clearly tendon degeneration is likely multi-factorial and dependant on both intrinsic and extrinsic causes.

PROGRESSION OF ROTATOR CUFF TEARS AND ONSET OF SYMPTOMS

It remains unclear why asymptomatic tears become symptomatic in certain individuals. A longitudinal analysis of asymptomatic tears by Yamaguchi *et al.*⁹ was performed as a model to evaluate the natural history of rotator cuff tears. They evaluated asymptomatic tears diagnosed by routine bilateral ultrasound examinations in patients who had contralateral symptomatic rotator cuff tears. They followed 45 patients, 23 of whom returned for repeat ultrasound examination for an average of 5.5 years. During this time 51% of the patients became symptomatic, with increases in pain and decreased ability on ADL testing. Of the 23 patients who returned for ultrasound examination, nine had progression of their tears. Fifty percent of the symptomatic patients had increase in their tears, while only 22% of the asymptomatic patients showed tear progression.⁹ A second study by the same group showed that pain development and deteriorating function in initial asymptomatic patients was associated with an increase in tear size of full-thickness tears and the progression of partial tears to full-thickness tears. Patients who initially had larger asymptomatic tears were more likely to develop symptoms in the short term.^{10*} These two studies together suggest that a significant portion of tears progress with time and that the overall size of the tear may be a predictor of future pain development.

In addition to the natural history of asymptomatic rotator cuff tears, the fate of symptomatic tears has been evaluated. In a study by Maman *et al.*,¹¹ serial MRI studies were done on 59 shoulders with either full or partial rotator cuff tears treated nonsurgically. They found that full-thickness tears (52%) were more likely to progress in size compared with partial tears (17%) over time. There was a 48% rate of progression in patients followed more than 18 months compared with a 19% rate in the first 18 months. Tear progression was more common in patients over the age of 60 years. Atrophy was seen only in patients with full tears. There was a 17% rate of the development or progression of fatty infiltration during the study.¹¹

In a cadaver study by Reilly *et al.*,¹² the possible cause of tear progression was evaluated biomechanically. Significant changes in strain both anteriorly and posteriorly to a simulated full-thickness tear were seen. Partial articular-side tears were found to increase the strain on the bursal surface of the tendon. Intratendinous tears were found to increase strain on both the bursal and joint sides of the tendon. This increase in strain led to greater stress on the remaining intact tendon and could help explain the extension of tears seen clinically.¹²

STRUCTURAL AND BIOMECHANICAL CHANGES

Chronic changes that occur within nonhealing rotator cuff tendons and the risk of tear extension are important aspects

of the natural history of rotator cuff disease. Full-thickness rotator cuff tears do not heal spontaneously. With the onset of tearing and the progression of tear size, changes occur in the musculotendinous unit. These changes are important to recognize because they affect the ability of the tendon to heal and function. Chronic rotator cuff tears can potentially retract and form adhesions that lead to fatty infiltration and atrophy. Fatty infiltration is associated with a higher failure rate of rotator cuff repairs. It has been shown to increase with time in animal studies and does not improve despite successful surgical repair.¹³ A recent study by Kim *et al.*^{14*} looked at variables associated with fatty infiltration. The study involved ultrasound examination of 251 full-thickness tears. Fatty degeneration was found in 34.7% of the tears. These tears were found to be larger in both width and length compared with tears without fatty degeneration. With each increase in tear size of 5 mm in either the length or the width, the odds of fatty degeneration doubled. Size was the most important determinant of fatty degeneration of the infraspinatus muscle. Interestingly, the tear pattern also was important in the development of fatty infiltration. Superior rotator cuff tears that did not involve the most anterior aspect of the supraspinatus had less risk of developing fatty infiltration.^{14*} This may be because the rotator cable as described by Burkhart *et al.*¹⁵ is maintained in this situation. It is thought that a maintained rotator cable enables the supraspinatus muscle to continue to function effectively, avoiding disuse atrophy and fatty infiltration. The posterior aspect of the rotator cable is disrupted in larger tears and may explain the fatty infiltration of the infraspinatus muscle seen in this study.

In addition to fatty infiltration, muscle atrophy also is thought to be irreversible. Animal studies have shown a loss of muscle mass and change in muscle fiber type after the supraspinatus was surgically detached.¹⁶ Additionally, animal studies have shown that the tendon stiffens and retracts after detachment.¹⁷ These changes occur relatively quickly in the animal model, and the ability to repair these tears is affected. A sheep model showed significant differences in the quality of the repair in cuff tears fixed at 6 weeks compared with 18 weeks. The force of contraction of the repaired muscle was smaller and slower to improve in the delayed group. This loss of force was caused by both the atrophy and change in viscoelastic properties seen in the delayed group. There was a significant increase in fatty infiltration found just 6 weeks after injury, and this did not improve as quickly or as much in the delayed repair group.¹⁸

Fatty degeneration and atrophy are permanent changes that can potentially produce a weak shoulder with decreased function. Along with the changes seen in the musculotendinous unit after rotator cuff tears, changes can occur at the glenohumeral joint. As described by Neer,⁵ large, chronic tears can lead to rotator cuff arthropathy in a small subset of patients. Milder degenerative changes to the articular cartilage in shoulders with rotator cuff tears probably occur more commonly than realized. A cadaver study looked at articular cartilage changes in rotator cuff disease. Thirty-three shoulders were examined for both rotator cuff tears and degenerative changes of the glenohumeral joint. Despite

the fact that eight of the 10 specimens with rotator cuff tears were partial articular tears, the rate of degenerative changes in these specimens was almost twice as frequent as in those without tears.¹⁹

MANAGEMENT OF ROTATOR CUFF TEARS

In deciding between nonsurgical and surgical management, the risks and benefits of each approach should be considered. The benefits of nonsurgical care include avoiding surgery and its inherent complications. The risks of nonsurgical management include recurrent symptoms, tear extension, and chronic changes to the musculotendinous unit and glenohumeral joint discussed above. Although patients can have excellent function and minimal pain after going through a rehabilitation program for a rotator cuff tear, a subgroup of these patients may do better long term with early surgical intervention. The difficulty in deciding between treatments is that the timing of these irreversible changes is still largely unknown. A study by Zingg *et al.*²⁰ looked at the outcomes of nonsurgical management of massive rotator cuff tears. Despite preservation of satisfactory shoulder function over the course of 4 years, there was substantial increase in the level of osteoarthritis, size of the tear, and fatty infiltration. During this time, four of eight rotator cuff tears that were initially judged as repairable had progressed to irreparable tears.²⁰ Another study by Melis *et al.*²¹ showed an increase in fatty infiltration if the infraspinatus muscle was torn or if multiple tendons were involved. Stage 2 fatty infiltration occurred 2.5 years after onset of symptoms with severe fatty infiltration occurring after 4 years. An increase in fatty infiltration was seen with increasing patient age.²¹

Patients with rotator cuff tears generally can be divided into three categories based on the risk for development of irreversible changes with prolonged nonsurgical care. Consideration of these risks will dictate the timing of surgical repair to help prevent these changes, which include muscle atrophy, fatty degeneration, changes in tendon morphology, and degenerative joint changes. In addition, with prolonged nonsurgical treatment, further adverse chronic changes can occur, including tear extension, retraction, and the formation of adhesions. These changes make surgical treatment more difficult and less likely to lead to healing of the rotator cuff. Given these considerations, patients with rotator cuff disease can be grouped into three categories: (1) those at low risk for irreversible changes, (2) those at risk for irreversible changes, and (3) those in whom irreversible changes have already occurred.²²

Group 1 patients have intact rotator cuffs with tendinitis or have only small partial-thickness tears. These patients are at lower risk of developing irreversible changes in the short term. A longer course (3–6 months) of nonsurgical treatment can be prescribed with a good chance of successful resolution of symptoms. Group 2 patients include younger individuals (<65 years) with symptomatic full-thickness tears, acute tears of any size, or tears with a recent loss of function. These patients should have minimal or no evidence of chronic changes to the rotator cuff on initial work-up. This is the group at highest risk for tear extension,

recurrent symptoms if they do respond to initial nonsurgical management, and, most importantly, irreversible changes to the cuff and glenohumeral joint. Early surgical repair may be preferable for these patients. Although the definitions of early and late surgical repairs are not clear, a recent study suggests that a delay of up to 3 months after an acute rotator cuff tear does not affect structural or clinical outcomes of surgery.²³ Group 3 consists of patients with either large, chronic tears or those who are older than age 70 years. In the case of chronic tears, the irreversible changes of atrophy and fatty infiltration have already occurred. A trial of non-surgical treatment is warranted in this group. There is little risk in waiting 3–6 months to see if the patient improves. Despite the large tear size and altered glenohumeral kinematics, these patients can become asymptomatic. If symptoms persist, however, surgery can then be undertaken without compromising the long-term results compared with earlier surgical intervention.

CONCLUSION

An improved understanding of the natural history of rotator cuff tears is critical in optimizing the treatment of rotator cuff tears. The size of the tear, fatty degeneration, atrophy of the muscle belly, and retraction all have been shown to diminish the results of surgical fixation. As knowledge on the timing of these changes improves, the surgeon can more effectively advise the patient on the benefits of operative and nonoperative treatment and improve the results in patients who undergo surgery.

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- of special interest
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Even in patients with large tears, there was no significant loss of shoulder function compared with those with an intact cuff. Pain is the key in noticing a decrease in shoulder function. Tears in the dominant hand are more likely to become painful. This study confirms that normal function is possible even with a rotator cuff tear.

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With short-term follow-up, a significant amount of patients with known tears became asymptomatic. These patients had an increase in tear size and decrease in function whereas those that remained asymptomatic had no significant change in their tears. Those that became asymptomatic had larger tears to start. No significant change in fatty infiltration occurred during this short-term study. In the clinic, smaller asymptomatic tears can be followed with little risk of progression of tear size or fatty infiltration. Yearly imaging may be useful to follow these changes. Larger tears, even if currently asymptomatic, may benefit from early surgical repair.

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