# MRI of the shoulder: Rotator cuff

J. Scott McMonagle, MD, and Emily N. Vinson, MD

Linking the axial trunk and upper extremity, the shoulder joint plays an imperative role in most daily activities, allowing us to position our hands in space. Further, the joint acts as a small fulcrum for a long lever arm, predisposing the rotator cuff to injury, especially from the rapid accelerations and decelerations inherent to most sports and even some activities of daily living.

Shoulder anatomy and biomechanics, particularly those of the rotator cuff (RC), endow the glenohumeral joint with dynamic and static stability throughout a substantial range of motion. The interconnected supraspinatus, infraspinatus, teres minor, and subscapularis musculotendinous complexes constitute the rotator cuff and act as the shoulder's primary functional unit. Because of the rotator cuff's crucial role, RC pathology may lead to considerable limitations in daily routine, work, and leisure/sporting activities.

Shoulder magnetic resonance imaging (MRI) improves the sensitivity and specificity of diagnosing RC disorders, reduces unnecessary arthroscopic procedures, and provides important clinical information to guide patient management. This review will cover recent literature regarding RC anatomy and the clinical presentation, evaluation, and management of RC disease. We will discuss new observations about the strengths, inherent blind spots, and diagnostic effectiveness of shoulder MRI, and then outline the classification of rotator cuff MRI findings and their impact on patient management. Finally, we will present an effective search pattern approach to evaluate the rotator cuff on shoulder MRI examinations.

***Dr. McMonagle*** *is an Associate Professor of Radiology, Musculoskeletal Radiology Fellow, and* ***Dr. Vinson*** *is an Assistant Professor of Radiology, Musculoskeletal Radiology Division, Department of Radiology, Duke University Medical Center, Durham, NC.*

## Normal anatomy

Knowledge of the RC tendinous insertions onto the proximal humerus, an area known as the rotator cuff footprint, makes it easier to determine the extent and location of abnormality. Much has been written recently about the anatomy of distal RC tendons as they interdigitate to insert upon the 3 facets of the greater tuberosity (superior, middle, and inferior), although their location and insertion appear somewhat more arbitrary by MR imaging. Standard landmarks and techniques used in MRI to demarcate the tendons will be elaborated upon later.

The supraspinatus muscle arises from the posterior aspect of the scapula, just above the scapular spine, and courses horizontally and anteriorly at the level of the acromioclavicular joint, a good landmark for its musculotendinous junction. The subacromial-subdeltoid bursa, which usually contains minimal fluid, if any, drapes over the supraspinatus muscle and tendon and lies just beneath the acromion. While most anatomy laboratories still teach that the supraspinatus has a broad footprint, more recent anatomic and orthopedic literature suggests that it has a relatively small triangular footprint on the superior facet of the greater tuberosity.[1,2] Importantly, the larger anterior portion of the supraspinatus muscle pulls a smaller cross-sectional area of anterior tendon,[3] predisposing the anterior articular fibers to more strain and subsequent tear propagation.[4-6]

The infraspinatus muscle arises from the posterior aspect of the scapula, below the scapular spine. It then courses laterally, with the anterior border of the infraspinatus tendon insertion overlapping the posterior border of the supraspinatus tendon, to attach to the entire middle facet interdigitating with the supraspinatus tendon at the posterior aspect of the superior facet.[2] The teres minor muscle, elongated in morphology, arises from the middle portion of the lateral scapular border and dense fascia of the infraspinatus to insert on the inferior facet of the greater tuberosity. The large, triangular subscapularis muscle arises from the anterior surface of the scapula and courses laterally under the coracoid, with its musculotendinous junction at the level of the glenoid. This muscle's attachment to the lesser tuberosity is comma shaped, with a broad proximal and tapering distal footprint.[7,8] The subscapularis fibers extend over the bicipital groove, and the superior fibers of the subscapularis tendon interdigitate with the anterior fibers of the supraspinatus tendon over the superior facet of the greater tuberosity.[9]

## Demographics of rotator cuff pathology

Shoulder pain is extremely common with reports that approximately half the U.S. population experiences at least one episode of shoulder pain annually.[10] The prevalence of shoulder pain substantially increases with age, and the most common musculoskeletal complaint in patients >65 is shoulder pain.[11] As rotator cuff tears are often asymptomatic, their true prevalence remains unknown and reports vary widely; the aptly titled "Dead Men and Radiologists Don't Lie" study reports a total prevalence of RC tears in their cadaveric data group of 30.3% (11.75% had full thickness tears; 18.49%, partial thickness tears), with the caveat that most of the cadavers, with a mean age of 70.1 years in that study, are older than the average patient.[12] Yamamoto et al screened 1366 shoulders, regardless of the presence or absence of symptoms, and found RC tears in 20.7%; their patients ranged from 22 to 85 years with a mean of 57.9 years. Their analysis also suggested risk factors for RC tears: a history of trauma, dominant arm, and older age.[13] A high correlation between the onset of RC tears and increasing age has also been reported in several other studies — in one, 50% of patients >66 years that presented with a painful RC tear also had a rotator cuff tear in their contralateral, asymptomatic shoulder.[14] Likewise, Fehringer et al found full thickness RC tears in 22% of asymptomatic patients ≥65 years.[15] In younger asymptomatic adults, the prevalence of tears is expectedly lower: 5% and 11% in the fourth and fifth decades, respectively. More surprising, however, are prevalences of 50% in the seventh decade and 80% in the 9th and 10th decades.[16] These studies confirm that RC tears are extremely common, especially in the elderly, and that it is important to remember that their presence does not always impute pain or clinically significant loss of function.[17]

## Pathogenesis of RC pathology

Although the true pathogenesis of rotator cuff tears remains unclear, mechanisms of RC degeneration are broadly divided into extrinsic and intrinsic factors. In reality, RC tears are probably a multifactorial byproduct of the interaction of intrinsic and extrinsic causes.[18] In 1934, Codman espoused the "intrinsic" theory that age-related tendon damage compounded by chronic microtrauma results in partial thickness tears, which usually then progress to full thickness tears.[19] An "extrinsic" cause was first suggested in 1972, when Neer proposed that RC tears were secondary to subacromial impingement and, therefore, best treated with anterior acromioplasty.[20] It was traditionally taught that hypovascularity within the critical zone located 10 to 15 mm proximal to the supraspinatus insertion was a key component of RC tears.[21,22] More recent research conclusively demonstrated that no significant hypovascular zone exists.[23-25] Moreover, Matthews and coworkers found that small RC tears showed increased fibroblast cellularity and blood vessel proliferation, which gave them greater potential to heal.[26] Additional studies demonstrate that as tear size increases the healing response fails, cytokines upregulate, and vascularity decreases, eventually resulting in hypoxic damage and apoptosis.[27-29] These novel findings imply the benefit of early rehabilitation and/or surgical management before tears progress.

## Clinical presentation, evaluation, and management

Patients with symptomatic rotator cuff tears usually present with shoulder pain, dysfunction, or both. Classic clinical teaching suggests that these symptoms are more significant in patients with subacromial bursitis and/or partial thickness RC tears compared to those with full thickness tears. Fukuda further reports that bursal-sided tears are more painful than articular-sided tears.[30] However, a more recent study by Brownlow et al relates that no statistical difference exists, and that neither pain nor stiffness can reliably differentiate partial and full-thickness tears.[31]

Clinical evaluation is the first step toward diagnosing RC disease. Clinicians often rely upon a battery of tests to evaluate and classify patients appropriately. A meta-analysis suggests that the diagnostic accuracy of orthopedic shoulder exams is overestimated, and that these exams are only rarely useful to differentiate RC tears. While some shoulder examination tests had high sensitivities and others had high specificities, no single test had both a high specificity and a high sensitivity.[32] Further, the lack of precise techniques and subjective interpretation of these exams leads to substantial interobserver variability.[33]

Management of partial and full thickness RC tears remains largely controversial. While nonoperative rehabilitation is successful in certain patient subgroups — predominately elderly patients with a sedentary lifestyle — early surgical repair is indicated in other patients, usually younger and more active individuals.[34] Despite good to excellent results of surgical repair in a high percentage of patients, re-rupture of the cuff is known to occur 20% to 65% of the time.[35,36] Much of the management controversy relates, in part, to diverse patient populations, varied surgical techniques, and a paucity of well-controlled comparative data. MRI plays a significant role in evaluating the stage and prognosis of RC disease: tear size, tendon retraction, and the extent of muscle atrophy, each of which negatively impacts the functional outcome. The clinical function and MRI appearance of RC tears deteriorate with time.[30,37] Partial thickness tears of the anterior supraspinatus fibers increase strain upon the remaining supraspinatus fibers and intact infraspinatus tendon, leading to tear propagation and potentially

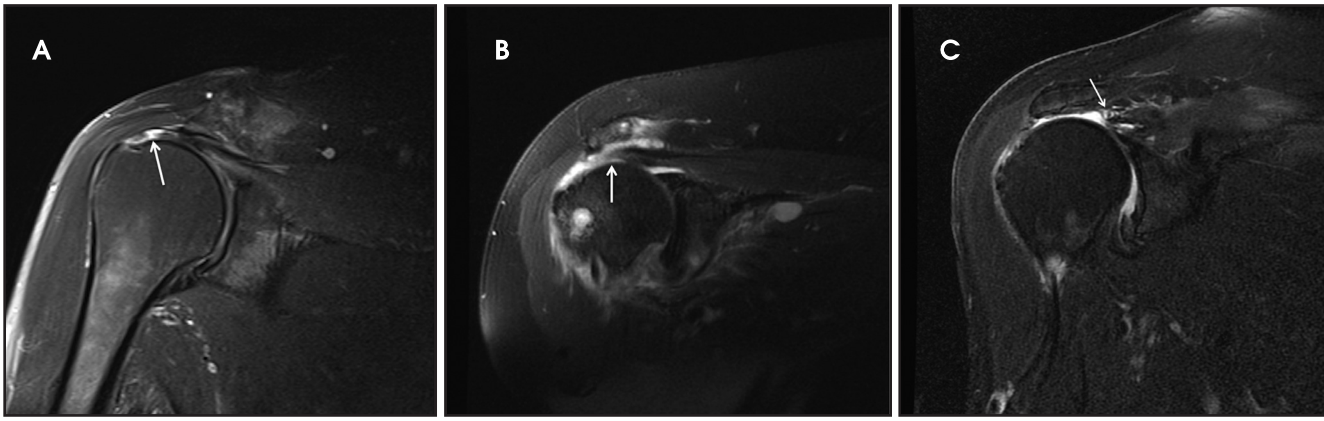


***FIGURE 1.*** *(A) Coronal oblique T2-weighted (T2W) images demonstrate full thickness tears (arrow) near the humeral insertion, (B) with tendon retraction to the level of the humeral dome (arrow), and (C) with tendon retraction to the level of the glenoid (arrow).*

impacting the decision to operate sooner as opposed to waiting.[5] The findings of Goutallier et al similarly support that increased wait times lead to increased fatty degeneration of the torn rotator cuff muscle.[38,39] Along these lines, Shen et al demonstrated a significant positive correlation between supraspinatus fatty atrophy preoperatively with postsurgical functional outcomes.[40] The article by Gladstone et al similarly confirmed that muscle atrophy and fatty infiltration played a significant role in functional outcome after repair. They added that tear size appears to have the most influential effect on repair integrity.[41] Fatty degeneration of the RC is closely associated with tear size and location. In particular, integrity of the anterior supraspinatus tendon seems to be the most important variable related to fatty degeneration.[42] A natural history of fatty infiltration relative to onset of shoulder symptoms was suggested by Melis et al, with moderate fatty infiltration at 3 years and severe fatty infiltration at 5 years.[39,43,44]

## Diagnostic imaging

Even though shoulder radiographs in acute rotator cuff tears are usually normal, they remain the appropriate first line of imaging to evaluate osseous structures and exclude common fractures and dislocations.[45-47] While multiple radiographic maneuvers and techniques have been suggested to help diagnose RC tears, and ultrasound in the hands of an experienced investigator has comparable accuracy,[47-49] MRI remains the study of choice for evaluating the shoulder. MRI can evaluate the size and shape of the tear, the amount of tendon retraction, the prominence of muscle atrophy, and the quality of remaining RC tendon. In addition, it can accurately evaluate other potential causes of shoulder pain that may mimic RC tears.[48]

Shoulder MRI can detect full thickness RC tears with high sensitivity and specificity, but MRI diagnosis of partial thickness tears is less sensitive and accurate. A large meta-analysis compiled in 2009 used a surgical reference standard and found pooled MRI sensitivity and specificity for full thickness tears of 92.1% and 92.9%; for partial thickness tears of 63.6% and 91.7%; and for full or partial thickness tears of 87.0% and 81.7%, respectively.[50] However, often cited statistics on the diagnostic effectiveness of MRI remain skewed by older protocols and outdated data that lack currently available spatial resolution; some of these even lack fat-suppression or T2-weighted images.[51] MRI of the RC at 3.0 Tesla appears promising. In 2006, Magee reported sensitivity and specificity of 98% and 96%, respectively, for full thickness supraspinatus tears and, even more impressive, of 89.5% and 90%, respectively, for partial thickness supraspinatus tears.[52] The diagnostic accuracy of shoulder MRI improves with experience and training.[53, 54]

Finally, the often-utilized gold standard of rotator cuff disease diagnosis — shoulder arthroscopy — is not without similar flaws. With the exception of distinguishing partial- from full-thickness RC tears and identifying the side of partial thickness tears (articular versus bursal), RC classification systems have little interobserver agreement even among experienced shoulder surgeons.[55]

## Classification of rotator cuff tears

Numerous rotator-cuff-tear classification systems have been proposed.[56-60] Though several are highly accurate, they are similarly complex and lack intraobserver and interobserver agreement. With an increasing emphasis on evidence-based medicine, MRI descriptions of RC tears need to be accurate, simple, and precise with high interobserver agreement.[61] Common pathology of the RC tendons includes full thickness tears, partial thickness tears, and tendinosis.

## Full thickness tears

An abnormality of the rotator cuff is considered a full thickness tear if it results in a connection between the articular and bursal surfaces of the cuff tendon. The most specific sign of a full thickness RC tear is visualization of a complete defect in the tendon, extending from the articular surface completely through to the bursal surface. This defect is usually fluid signal intensity as it is filled with fluid, organizing granulation tissue, myofibroblastic proliferation, chondroid metaplasia and/or hemorrhage.[26,62] Fortunately, this appearance of full thickness tears is also the most common, seen approximately

**FIGURE 2.** *Coronal oblique T2W image shows a partial thickness tear (arrow) of the articular-sided fibers that involves <50% of the tendon; overlying bursal-sided fibers remain intact.*

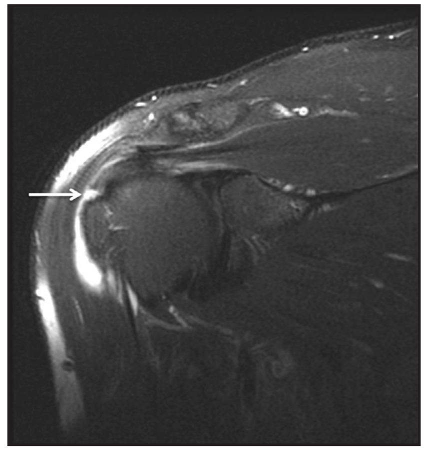

**FIGURE 3.** *Coronal oblique T2W image demonstrates a large partial thickness tear of the bursal-sided fibers (arrow) that involves >50% of the tendon; a few remaining articular-sided fibers remain intact.*

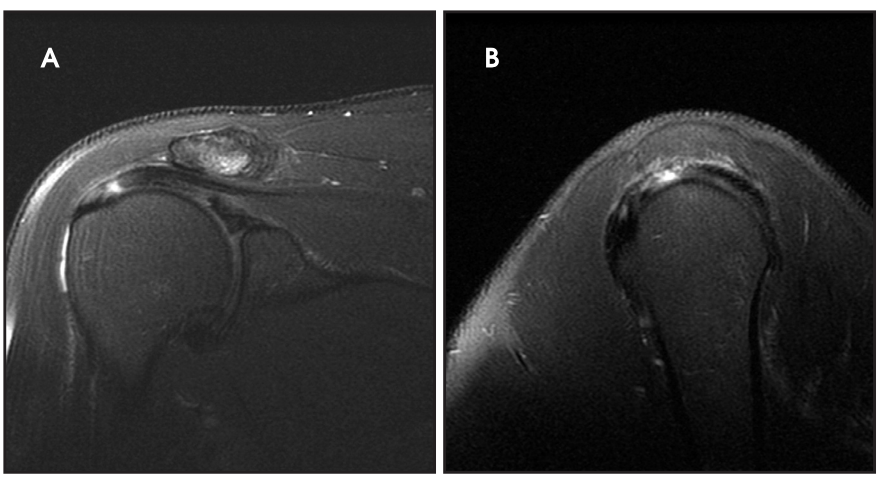


**FIGURE 4.** *(A) Coronal oblique and (B) sagittal oblique T2W images demonstrate a large partial thickness tear of the articular-sided fibers with a few remaining bursal sided fibers.*

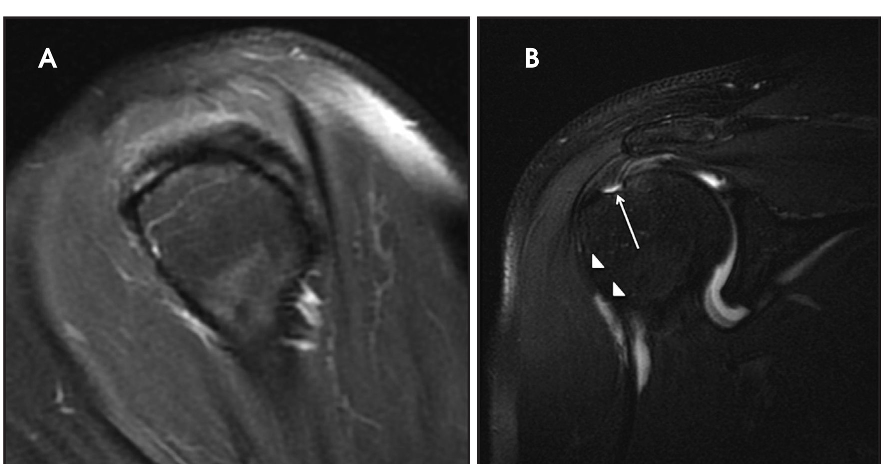


**FIGURE 5.** *(A) Sagittal oblique and (B) coronal oblique T2W images of a rim-rent (arrow), a small articular-sided tear involving the insertional fibers of the anterior most aspect of the supraspinatus. Notice the proximity to the biceps tendon (arrowheads).*

87% of the time.[63] Less commonly, full thickness defects contain intermediate T2 signal intensity, probably related to chronic scarring or volume averaging with adjacent histopathologic changes, such as scarring or mucoid degeneration.[64] Less classic findings are more frequent when the defects are small, given the increased potential for volume averaging. Secondary signs of a full thickness RC tear — fluid in the subacromial-subdeltoid bursa, muscle atrophy, intramuscular cysts, superior humeral migration, and retraction of the musculotendinous junction — used to be more heavily relied upon prior to higher-resolution MR capabilities and the routine use of fat suppression.[63,65-67] While these secondary findings in isolation never confirm a full thickness tear, they should prompt a close second look. It's worth mentioning that literature has demonstrated each of these secondary findings with partial thickness tears and even in patients without a tendon defect.[68-71] To adequately detail full thickness RC tears, two descriptors should be used: the anteroposterior extent of the tear and the amount of medial tendon retraction. Precise measurements for the anteroposterior extent of full thickness tears have been abandoned because of subjective variability and lack of reproducibility. Descriptive categories are instead used to group the anteroposterior extent of full thickness tears: small (<1 cm), medium (1-3 cm), large (3-5 cm), and massive (>5 cm). The amount of tendon retraction (Figure 1) is best evaluated on coronal oblique images by describing the location of the torn and retracted tendon in one of 3 ways: near the humeral insertion, at the level of the humeral dome, or at the level of the glenoid.[59] The final component of describing a full thickness tear is evaluating the RC muscles for volume atrophy or fatty infiltration, which, as described previously, significantly impact functional outcome after repair.[40, 41]

## Partial thickness tears

Partial thickness tears involve a spectrum of findings and are broadly classi-

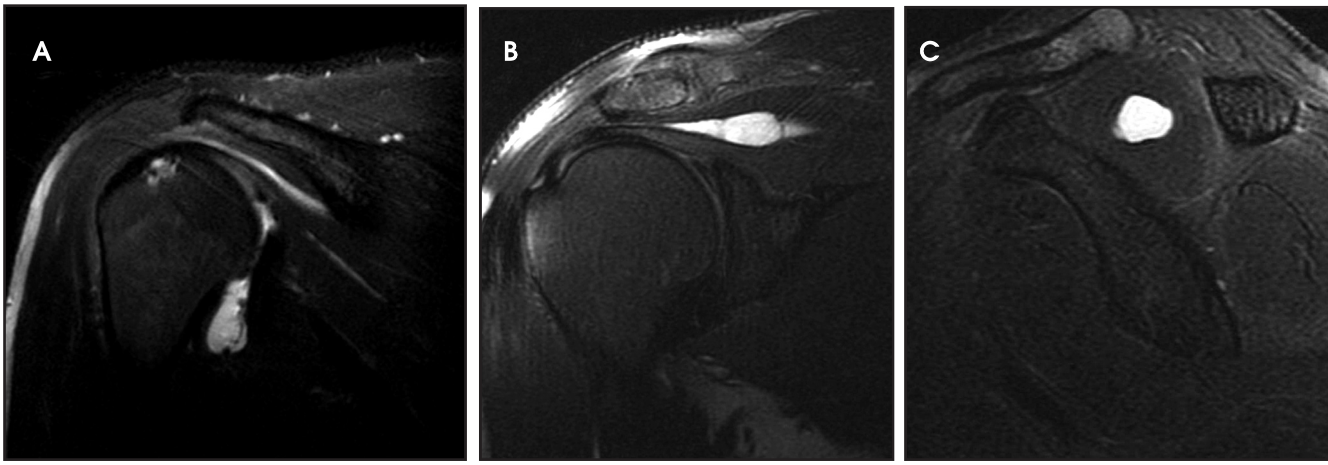


***FIGURE 6.*** *(A) Coronal oblique T2W image of an interstitial tear within the supraspinatus. (B) Coronal oblique and (C) sagittal oblique T2W images of an intramuscular cyst within the supraspinatus muscle.*

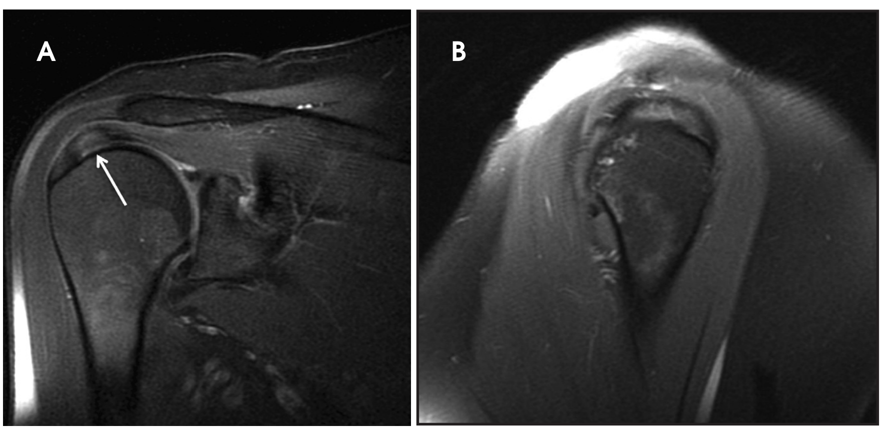


***FIGURE 7.*** *(A) Coronal oblique T2W image demonstrates focal intermediate signal intensity and tendon thickening (arrow), compatible with moderate tendinosis. (B) Cross-referenced sagittal oblique T2W image confirms moderate tendinosis with no fluid-signal intensity or tendon disruption to indicate a tear.*

fied into 3 different types according to the portion of the tendon that is abnormal: articular-sided tears, bursal-sided tears, and interstitial tears.

Articular-sided tears (Figure 2) are by far the most common, involving the tendon fibers adjacent to the humeral head. Bursal-sided tears (Figure 3) are significantly less common — 2.9% of partial thickness tears in one series[72] — and involve the more superiorly located fibers that abut the subacromial-subdeltoid bursa. The combination of fluid, granulation tissue, and blood within partial thickness tears causes their hyperintense T2 signal characteristics, similar to those of full thickness tears, but without full thickness involvement of the tendon.[73] For example, if intact bursal-sided fibers are identified overlying discontinuous articular-sided fibers, the diagnosis of an articular-sided partial thickness tear has been made. Beyond the location of the partial thickness tear, describing the extent of the tear is also important. Ruotolo et al showed that the mean thickness of the supraspinatus tendon ranges from 11.6 mm (anteriorly) to 12.1 mm (midtendon).[74] Exact measurements are difficult to recreate precisely, so descriptive terms work best, combined with an awareness of their clinical implication. Small tears (<3 mm deep) and medium tears (3-6 mm deep) involve <50% of the tendon thickness, while large partial thickness tears (>6 mm deep) involve >50% of the tendon fibers (Figure 4).[57] Treatment of articular-sided, partial thickness tendon tears involving <50% of the fibers usually involves surgical debridement. In contrast, most authors recommend surgical repair of articular-sided tears involving 50% or more of the tendon thickness.[75, 76]

Partial thickness, articular-sided RC tears involving the insertional fibers of the anterior-most aspect of the supraspinatus (Figure 5), sometimes referred to as rim-rents, are very common tears, but they are easily overlooked.[72] Becoming more aware of these tears, their location and their frequency should lead to a focused evaluation for these defects. Specific techniques to look for rim-rents will be discussed in the search pattern section below.

Interstitial tears may represent up to 33% of partial thickness tears[77] and are thought to represent shearing forces within a degenerated tendon.[78] Interstitial tears (Figure 6), also known as intrasubstance tears or intramuscular cysts, can occur in isolation within the tendon without articular- or bursal-sided extension, or they can also occur in combination with either articular- or bursal-sided partial thickness tears. Intramuscular cysts, towards the larger, more elliptical end of the interstitial tear spectrum, were seen in 0.3% of a large retrospective series of shoulder MR examinations, and are almost always (96-100%) associated with an RC tear.[66,79,80] Though management of isolated interstitial tears remains controversial, they should be reported since they are not visible to the arthroscopist.

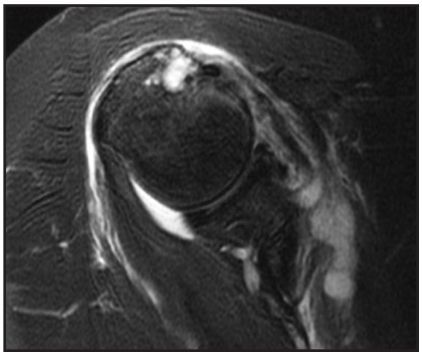

***FIGURE 8.*** *Axial T2W image demonstrates a humeral head cyst within the anterior aspect of the greater tuberosity and an overlying fluid-signal intensity defect (no supraspinatus fibers are seen) related to a full thickness tear.*

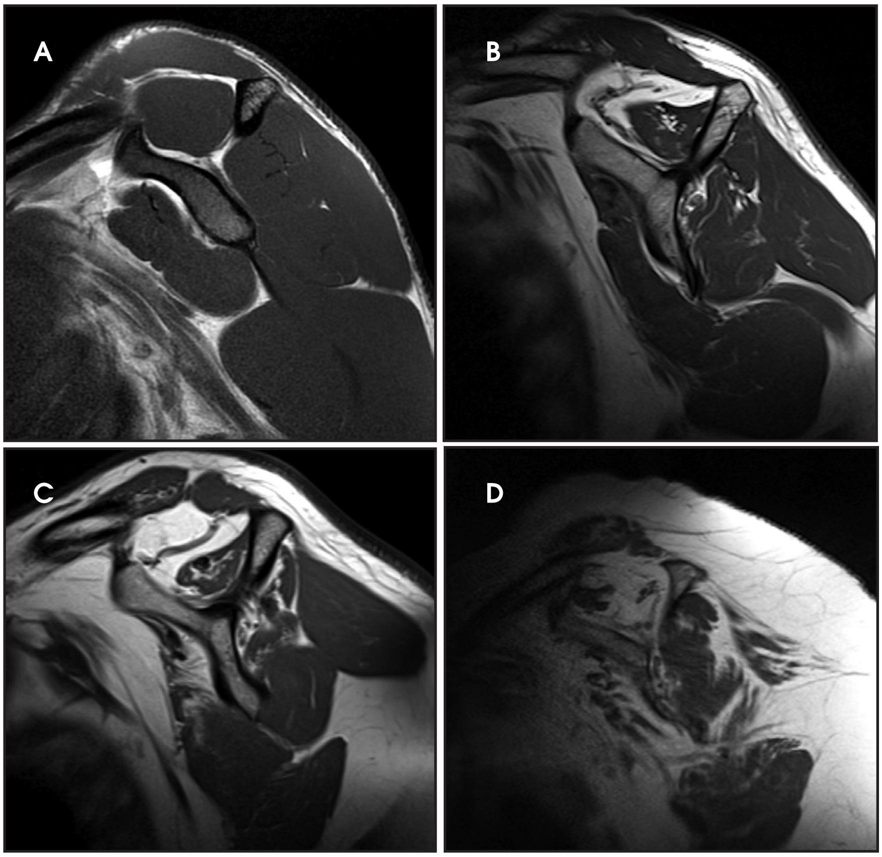


***FIGURE 9.*** *(A) Sagittal oblique T1-weighted (T1W) image demonstrates normal rotator cuff musculature. (B) Sagittal oblique T1W image shows mild supraspinatus and infraspinatus volume atrophy (each is even with its tangent line) and only minimal fatty infiltration. (C) Sagittal oblique T1W image demonstrates moderate volume atrophy and fatty infiltration of the supraspinatus and infraspinatus muscles. There is also severe atrophy and advanced fatty infiltration of the superior aspect of the subscapularis muscle. (D) Sagittal oblique T1W image shows severe atrophy and advanced fatty infiltration of the supraspinatus and subscapularis muscles.*

## Tendinosis

Normal RC tendons, composed primarily of collagen bundles, are characterized by uniform hypointense signal intensity on all pulse sequences. Though in the past the term "tendonitis" had been used to refer to a variety of shoulder problems, degenerative and traumatic changes of the RC tendons do not always include inflammation, so tendinosis or tendinopathy are the preferred terms. Histological changes of RC tendons, most commonly mucoid degeneration and fibrocartilaginous metaplasia, appear as moderately increased focal irregular or diffuse intermediate intrasubstance signal intensity on T1- and T2-weighted images.[64] This altered MRI signal intensity does not rise to the same T2 signal intensity as fluid and is usually more globular in appearance and less linear than signal abnormalities seen with RC tears. The tendon may be normal in caliber or demonstrate diffuse or focal thickening (Figure 7).[81] Sein et al showed high intraobserver reliability, with an intraclass correlation of 0.85, in grading tendinosis as mild, moderate, or severe.[82] In practice, it can occasionally be difficult to distinguish between severe tendinosis and a partial thickness tear; sometimes both possibilities must be considered.

## Humeral head cysts

Humeral head "cysts," foci of fluid signal intensity in the bone marrow of the greater tuberosity near the RC insertions, are common on shoulder MRI, seen in 70% of symptomatic patients and 24% of asymptomatic volunteers.[51, 83] They occur most frequently within the posterior aspect of the greater tuberosity (56%-90%), followed by the anterior aspect of the greater tuberosity (20%-22%), and least often within the lesser tuberosity (15%).[83, 84] Cysts within the posterior aspect of the greater tuberosity show no association to RC abnormalities, and are thought to represent normal variant pseudocysts, since they contain no synovial lining and are filled with thick connective tissue.[83-85] While one article suggested no definite association between RC tears and cysts within the anterior aspect of the greater tuberosity,[83] most studies endorse an association with RC disease. Sano et al in 1998 indicated that RC tears were always present when humeral head cysts were present in the superior facet or the anterior half of the middle facet.[86] More recently, Fritz et al reported that cysts within the anterior aspect of the greater tuberosity (Figure 8) have a high positive predictive value (94%) for RC disease; further, anterior greater tuberosity cysts were rarely, only 2.5% of the time, seen with normal rotator cuffs.[84] Cysts within the lesser tuberosity, though less common, almost always correlate with tears of the RC, particularly the overlying subscapularis or supraspinatus tendons.[86,87] Humeral head "cysts" can simulate small insertional RC tears, and should therefore be evaluated on orthogonal views, which usually better

*FIGURE 10.* *Sagittal oblique T2W image with diffusely increased signal intensity throughout the supraspinatus, infraspinatus, and teres minor muscles (arrowheads). Compare to the normal signal intensity subscapularis muscle (arrow). These MRI findings are compatible with muscular edema; the distinct combination of muscle involvement is typical of Parsonage-Turner Syndrome.*

reveal that the fluid-signal intensity is actually subcortical and not within the distal RC tendon.

## Fatty atrophy

Muscular changes after RC tears are defined by 2 separate parameters: volume atrophy and fatty infiltration. Since volume atrophy and fatty infiltration occur highly asymmetrically — especially in the supraspinatus due to the disproportionate biomechanical changes when anterior tendon fibers are torn — both qualifiers should always be evaluated and mentioned.[88] Goutallier, using CT scans, proposed the first classification scheme for volume atrophy and fatty infiltration of RC muscles.[89] Warner et al further expanded fatty atrophy evaluation using sagittal oblique T1-weighted MR images just medial to the coracoid. This volume atrophy classification scheme uses the tangent line, drawn horizontally between the superior edge of the coracoid and the scapular spine, and another line drawn vertically from the scapular spine to the inferior scapular tip. Muscle bellies that remain convex beyond these lines are graded as normal; those that are even with the lines are called mild atrophy; muscle concave below the line is moderately atrophied; muscle that is barely visible is severely atrophied (Figure 9).[90] Fuchs et al modified Goutallier's original classification scheme of fatty infiltration to MRI – describing the proportion of fatty streaks and replacement relative to muscle.[91] Their original staging has been further qualified and descriptive modifiers are now usually used to describe fatty infiltration: no fatty deposits is described as no fatty infiltration; a few fatty streaks, as minimal fatty infiltration; some fatty infiltration, though still with more muscle than fat, as mild fatty infiltration; a roughly equal proportion of muscle and fatty infiltration as moderate fatty infiltration; and a predominance of fatty infiltration, as advanced fatty infiltration.

## Search pattern

Like most radiologic exams, shoulder MR imaging is best evaluated with a routine search pattern that pays specific attention to the usual locations of pathology and relies upon the specific strengths of each obtained sequence. Our standard shoulder MRI protocol contains 5 pulse sequences: sagittal oblique T1-weighted and fast spin echo (FSE) T2-weighted with fat saturation, axial FSE proton density and FSE T2-weighted with fat saturation, and coronal oblique FSE T2-weighted with fat saturation.

## Rotator cuff muscles

The first component of MRI RC evaluation involves the muscle bellies on the T1-weighted and T2-weighted sagittal oblique sequences. As described previously, the presence of fatty infiltration or muscle volume atrophy is best evaluated on the T1-weighted sagittal oblique images. Sagittal oblique T2-weighted images are used to carefully evaluate the muscle bellies for often subtle or asymmetric edema. Muscular edema often represents an acute injury, such as muscle strain, particularly within the clinical context of an acute injury and when correlating findings of an acute RC tear are present. Neurogenic abnormalities can also cause muscle edema, if detected early, and muscle atrophy, if more chronic. They are usually identified by the distinct combination of muscle bellies involved: Suprascapular nerve entrapment at the suprascapular notch affects the supraspinatus and infraspinatus muscles; suprascapular nerve entrapment at the level of the spinoglenoid notch (distal to supraspinatus motor innervation) affects only the infraspinatus muscle; quadrilateral space syndrome (compression of the axillary nerve in the quadrilateral space) usually affects just the teres minor muscle; and Parsonage-Turner Syndrome, an acute brachial neuritis, can involve single or multiple nerve distributions (Figure 10).

## Rotator cuff tendons

Beyond knowledge of RC anatomy, MRI relies upon landmarks to delineate the location of the RC tendons. The precise transition between the supraspinatus and infraspinatus is difficult to distinguish by MRI given their interdigitating fibers. When the patient is correctly positioned with the arm neutral or slightly externally rotated, the 12 o'clock humeral head position on sagittal oblique sequences is used to distinguish where the supraspinatus and infraspinatus insert on the greater tuberosity — supraspinatus fibers are anterior (10 and 11 o'clock), supraspinatus and infraspinatus fibers blend near 12 o'clock, and infraspinatus fibers posteriorly occupy the 1 and 2 o'clock positions. Internal rotation of the arm produces overlap of the supraspinatus and infraspinatus tendons, making evaluation of the RC difficult.[92] With internal rotation or extreme external rotation, the humeral head clock positions are suboptimal for distinguishing the tendon insertions.

Having evaluated the RC muscle bellies, and with a familiarity of how to distinguish the RC footprints, the search pattern next individually evaluates each of the 4 RC tendons. Most PACS display monitors allow similarly oriented sequences to be linked and orthogonal sequences to be cross-referenced. Beginning at the medial aspect of the sagittal oblique T2-weighted images, the supraspinatus muscle is first located by its anatomic position on the scapula. Proceeding laterally, sagittal oblique images are incrementally examined. When abnormalities are seen or suspected in the supraspinatus tendon on sagittal oblique images, they are cross-referenced on coronal oblique images. The integrity of the supraspinatus tendon is again completely evaluated by the coronal oblique sequence. The extra-articular long biceps tendon is located on the anterior coronal oblique images, and proceeding posteriorly, the anterior-most fibers of the supraspinatus tendon are evaluated. Again, any signal abnormality detected within the supraspinatus tendon on coronal oblique images is cross-referenced and evaluated on the sagittal oblique images. Abnormal supraspinatus findings — tendinosis or partial or full thickness tears — are categorized and described, as mentioned earlier. A similar 2-plane approach is used to confirm and evaluate the status of each remaining tendon, usually proceeding from the supraspinatus to the infraspinatus, then to the subscapularis, and finally, to the teres minor, which is almost never torn.

Since partial thickness tears involving the far anterior supraspinatus fibers have a high prevalence, are often difficult to visualize (particularly when the arm is internally rotated), and as a result are frequently missed,[72] the last step involves an intentional search for rim-rents. Because they are often difficult to locate on sagittal oblique images, we rely instead upon coronal oblique and axial sequences. The extra-articular long head of the biceps tendon is identified on coronal oblique images; the anterior-most fibers of the supraspinatus are located just posterior to that point. These anterior fibers are also cross-referenced on axial images as a precautionary final look.

## Conclusion

Our understanding of rotator cuff pathogenesis and the optimal management of RC pathology is evolving. Shoulder MRI is a valuable tool in evaluating the patient with a painful shoulder, as it accurately depicts RC tendon pathology and any associated muscle abnormalities. In addition, shoulder MRI may reveal concurrent or alternative diagnoses, beyond the scope of this article, which can mimic RC disease clinically.

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