Shoulder pain is a common musculoskeletal medical condition affecting 7% to 26% of individuals and is the third most common musculoskeletal-related complaint in the primary care setting.\(^1,2\) Rotator cuff pathology is a common etiology for shoulder pain, with impingement of the rotator cuff often playing an important role. Rotator cuff impingement was first described by Neer et al when he stated that 95% of rotator cuff tears were attributed to impingement and generally occur in patients over age 40.\(^3,4\) Two predominant types of shoulder impingement have been described: intrinsic and extrinsic.

Intrinsic shoulder impingement is relatively uncommon and seen almost exclusively with overhead throwing athletes.\(^5\) It is a result of extreme abduction and external rotation, which can lead to entrapment of the supraspinatus and/or the infraspinatus tendons between the glenoid.\(^6\) MRI appearance of intrinsic impingement is varied and includes labral and rotator cuff pathology. The infraspinatus tendon is commonly injured, especially in patients under age 30, with MRI findings ranging from undersurface tears to complete tears.\(^6,7\)

Extrinsic, or external, impingement is one of the more common causes of shoulder pain and a frequent source for an orthopedic evaluation.\(^8\) Extrinsic shoulder impingement is most commonly related to mechanical compression from the acromion, acromioclavicular joint, and the coracoacromial ligament.\(^3,9\) Numerous operative and nonoperative treatment options have been described for extrinsic impingement, ranging from physical therapy and injections to acromioplasty and Mumford procedures.

The purpose of this article is to help understand the relevant anatomy with regard to the subacromial region, review the imaging findings, and discuss the differing etiologies of extrinsic impingement. This knowledge will allow radiologists to effectively communicate with clinicians and help guide appropriate treatment.

**Anatomy**

The coracoacromial arch provides a safeguard for the shoulder, limiting superior migration of the humeral head. The coracoacromial arch is composed of (from anterior to posterior) the coracoacromial ligament, and the acromion process. Inferior to these structures, and coursing through the arch, are the subacromial/subdeltoid bursa, supraspinatus tendon, and biceps tendon. The humeral head provides the posterior/inferior border of the arch (Figure 1). Processes that decrease the space within the coracoacromial arch presumably may lead to impingement-like symptoms.

The coracoid process is a hook-like osseous structure arising from the superolateral edge of the scapula and extends in an anterolateral orientation. The coracoid is an anchor point for several ligaments and tendons, including the pectoralis minor, coracobrachialis, and short head of the biceps brachii tendons. Also, the coracoid is an attachment site for the coracoacromial, coracoacromial, coracoclavicular, and superior transverse ligaments.\(^10\) The coracoid process can be palpated below and at the lateral edge of the clavicle. Known as the “Surgeon’s Lighthouse,” the coracoid process serves as a landmark to avoid neurovascular injury, as major neurovascular structures traverse medially to the coracoid process.\(^11\)

The coracoacromial ligament is a thick triangular ligamentous structure extending inferomedially from the anterolateral undersurface of the acromion to the lateral border of the coracoid process. This structure provides an...
osseo-ligamentous static restraint to superior humeral head displacement. Ligamentous connection of the coracoacromial ligament and the rotator interval capsule is thought to prevent inferior migration of the glenohumeral joint. Coracoacromial ligament thickness is normally 2 to 5.6 mm. Implicated as a pain generator in impingement syndrome, treatment of the coracoacromial ligament has been controversial. There is uncertainty regarding whether or not to perform a release of the coracoacromial ligament during acromioplasty, as this could increase the risk of superior and anterior glenohumeral translation.

The acromion process is a triangular-like extension of the scapular spine, and anteriorly articulates with the clavicle. The acromioclavicular articulation is a synovial joint, connecting the scapula and clavicle, allowing the scapula to have multidirectional motion with relation to the rest of the body. The coracoacromial ligament forms the ligamentous attachment between the acromion and coracoid process (Figure 2). The deltoid and trapezius muscle groups have attachments on the acromion. The
deltoid muscle abducts the arm at the shoulder and contraction of the trapezius rotates the scapula, providing stability to the scapular body. At birth, the distal acromion and clavicle are cartilaginous structures, but maintain the shape of the fully matured ossified bone. With maturation, the primary ossification center ossifies and proceeds to fuse with smaller secondary ossification centers to form a single fused plate. Most literature suggests these ossification centers normally ossify between 18 to 25 years of age. Unfused secondary ossification centers may be mistaken for avulsion fractures, and primary and secondary fusion scars can mimic an incompletely healed fracture on radiographs and MRI, respectively (Figure 3). The presence of marrow edema on MR images should portend a diagnosis of fracture.

As stated earlier, Neer proposed that tears of the cuff tendons (specifically the supraspinatus) are often a result of impingement by structures forming the coracoacromial arch. Extrinsic impingement syndrome is clinically characterized as acute or chronic pain induced by abduction and external rotation or elevation with internal rotation of the shoulder. Impingement typically occurs in young athletes who participate in sports involving repetitive movements of elevation and abduction of the shoulder, or in the elderly population with degenerative joint disease. Numerous anatomical etiologies have
Shoulder Impingement and Associated MRI

been suggested as a contributor of impingement syndrome including variant acromion shape, slope of the acromion, acromioclavicular arthropathy, acromion positioning, coracoacromial ligament thickening, and os acromiale. However, it is worth noting that many of the aforementioned anatomical factors can be seen in the asymptomatic patient, and ultimately the diagnosis is one of a clinical nature.\(^{17}\)

Three predominant shapes of the lateral acromion have been described by Bigliani et al and are based on the scapular-Y view radiograph.\(^{18}\) Type I acromion has a flat undersurface (Figure 4). Type II acromion is more concave along the undersurface with the inferior acromial cortex parallel to the cortex of the humeral head (Figure 5). Type III acromion has an inferiorly projecting anterior hook, narrowing the space between the acromion and humeral head (Figure 6). Some theories suggest type II and III acromions have an increased incidence of cuff disease, but this remains controversial among most surgeons.\(^{17}\) Type IV acromion has also recently been described as having a convex undersurface, although correlation with extrinsic impingement has not been shown.\(^{19,20}\)

Normal orientation of the lateral aspect of the acromion is horizontal or slightly downsloping posteriorly on the sagittal images. An abnormally sloped acromion occurs in an anteriorly downsloped and inferolateral sloped orientation (Figure 7). These anomalous acromial sloping positions can impinge on the subacromial space and cause mechanical trauma to the subjacent supraspinatus tendon.\(^{17}\)

Normal positioning of the acromion has an inferior cortex running parallel with the inferior cortex of the clavicle. When the acromion is low-lying, the inferior cortex of the acromion lies below the inferior cortex of the clavicle (Figure 8). This low position causes narrowing of the subacromial space.\(^{17}\)

Os acromiale represents an accessory ossification center that has not fused by age 25 years. This normal variant occurs in 15% of the population. Failure of fusion occurs between one of the 3 ossification centers: pre-acromion (Figure 9A), meso-acromion (Figure 9B), and meta-acromion. A classification scheme was proposed by Park et al with subtypes A-G. Type A, also known as the meso-acromial or meso-type, is the most common and is a failure of fusion between the meso-acromion and meta-acromion.\(^{21}\) The presence of an os acromiale has been associated with increased incidence of impingement and rotator cuff disease,
Shoulder Impingement and Associated MRI

**FIGURE 11.** Coronal STIR image showing supraspinatus tendinosis/tendinopathy. There is intermediate intrasubstance signal intensity (asterisk) within the thickened supraspinatus tendon. This signal is hypointense compared to intra-articular fluid signal. The bursal and articular surface fibers are intact (solid arrows). There is also incidentally mild subacromial/subdeltoid bursitis with fluid-like signal in the bursa (dashed arrow).

**FIGURE 12.** Bursal surface partial thickness rotator cuff tear. Coronal T2-weighted fat-suppressed image with bursal surface hyperintense signal involving >50% of the supraspinatus tendon footprint depth (solid arrow) with intact articular surface fibers (dashed arrow). This is consistent with a grade III partial bursal surface supraspinatus tear.

**FIGURE 13.** Intrasubstance partial thickness rotator cuff tear. Coronal PD fat-suppressed image showing an intrasubstance tear of the supraspinatus tendon at the footprint (solid arrow) measuring <3mm, consistent with a grade I partial tear. Linear longitudinal signal propagates from the footprint into the substance of the tendon (dashed arrow), which represents an interstitial/delaminating component. Intact bursal and articular surface fibers are at the tendon footprint (asterisk).

**FIGURE 14.** Articular surface partial thickness rotator cuff tear. Coronal T2-weighted fat-suppressed image showing focal articular surface hyperintense signal (solid arrow) involving <50% of the tendon depth. An incidental superior labral tear is also seen (dashed arrow).
Shoulder Impingement and Associated MRI

although is controversial in the literature.\textsuperscript{21,22} There is suggestion that the os acromiale is mobile, and thus reduces the size of the coracoacromial arch during motion. Os acromiale can cause impingement in 2 separate manners: Contraction of the deltoid muscle will force the os acromiale inferiorly, leading to narrowing of the rotator cuff outlet. The other mechanism occurs when osteophytes develop at the margin of the acromial gap, often directly impinging on the rotator cuff.

**Imaging Appearance of Extrinsic Impingement**

The imaging findings of external impingement are varied, ranging from subacromial/subdeltoid bursitis to full-thickness rotator cuff tears. In

**FIGURE 15.** Full thickness rotator cuff tear. Coronal T2-weighted fat-suppressed image with intra-articular contrast displaying a full-thickness defect extending through the bursal and articular surfaces of the supraspinatus tendon (solid arrow). There is minimal retraction of the tendon from the footprint (dashed arrow). Contrast is extending through the tear into the subacromial/subdeltoid space.

**FIGURE 16.** Full thickness rotator cuff tear with retraction. Coronal PD fat-suppressed image showing a full-thickness tear (solid arrow) of the supraspinatus tendon. Retraction of the tendon is > 5 cm from the footprint, medial to the glenoid (dashed arrow). Retraction medial to the glenoid is an indication of a tear not usually amenable to repair. Incidentally, there is cephalization of the humeral head nearly abutting the undersurface of the acromion secondary to complete rotator cuff tear.

**FIGURE 17.** Subacromial spurs. Coronal PD fat-suppressed image showing a keel osteophyte (solid arrow) along the anterolateral undersurface of the acromion. The keel osteophyte is an aggressive spur often resulting in severe damage to the bursal cuff.

**FIGURE 18.** Lateral acromial angle. Coronal PD fat-suppressed image with a normal lateral acromion angle. This angle is formed by a vertical line lateral to the glenoid and a line parallel to the undersurface of the acromion. Normal angle is > 70 degrees.
Shoulder Impingement and Associated MRI

1972, Charles Neer classified impingement into 3 distinct stages. Stage I impingement is characterized by edema and/or hemorrhage in the subacromial/ subdeltoid bursa and is generally seen in patients less than 25 years-old. Stage II demonstrates chronic changes such as fibrosis and tendinitis of the rotator cuff and is generally seen in those 25 to 40 years. Stage III is characterized by partial or complete rotator cuff tears, and is usually seen in patients > 40 years.

Subacromial/subdeltoid bursitis is a finding seen with considerable frequency on MRI examinations of the shoulder (Figure 10). This diagnosis is characterized by fluid-like signal in the bursa that extends 2 cm medial to the acromioclavicular joint with distension of the bursa > than 3 mm thick. Fluid also commonly extends into the anterior aspect of the bursa.

Rotator cuff tendinopathy (or tendinosis) is a finding seen with considerable frequency on MRI examinations of the shoulder (Figure 10). This diagnosis is characterized by fluid-like signal in the bursa that extends 2 cm medial to the acromioclavicular joint with distension of the bursa > than 3 mm thick. Fluid also commonly extends into the anterior aspect of the bursa. Rotator cuff tendinopathy is hyperintense on fluid-sensitive sequences, but is hypointense compared to nearby intra-articular fluid signal (Figure 11). Fluid-like hyperintense signal within the tendon suggests an intrasubstance partial tear.

Partial thickness tears of the rotator cuff are characterized by fluid-like signal in the tendons that does not involve the entire tendon bulk. These tears can be further characterized by whether they involve the bursal surface, intrasubstance portion of the tendon, or articular surface of the tendon fibers (Figures 12-14). Partial thickness tears occur more frequently along the articular surface of the tendon. Ellman developed a classification system for partial rotator cuff tears based on the tendon tear depth. This system classifies partial thickness tears into grade I (< 3 mm), grade II (3 to 6 mm), or grade III (> 6 mm). As grade III tears comprise > 50% of the rotator cuff, these are generally considered significant and are typically repaired surgically.

Full-thickness rotator cuff tears are manifested by fluid-like signal within the tendon, which extends throughout the entire substance of the tendon from the articular to the bursal surface (Figure 15). These can be further classified according to size. A widely used system created by DeOrio and Cofield classifies full thickness tears based on their greatest dimension. These are classified as small (< 1 cm), medium (1 to 3 cm), large (3 to 5 cm), or massive (> 5 cm). Other important features of full-thickness tears are the degree of tendon retraction and presence or absence of muscle atrophy. A tendon retracted medial to the glenoid has historically indicated that the tendon tear is not amenable to repair (Figure 16). Muscle atrophy in association with a full-thickness tear of the supraspinatus or infraspinatus tendons suggests that the muscle has lost its ability to contract and may not be successfully treated with surgery. This information is of clinical value to the surgeon when deciding whether to repair the tear.

MR characteristics of impingement have been subsequently grouped into subtypes by Seeger et al. Type I impingement is noted as the least severe and includes subacromial bursitis and bursal thickening with normal signal intensity of the supraspinatus tendon. Type II impingement is classified as abnormally high-signal in the supraspinatus tendon without abnormal intramuscular signal or tendon retraction.
Type III impingement findings include full-thickness abnormal signal intensity with muscle retraction, indicating a full-thickness tear.

Acromion morphology and acromion-related pathology is easily identified on MR. Acromial spurs have been described with numerous morphologic subtypes including acromial hook and keel osteophytes. The acromial hook lies within the origin of the coracoacromial ligament and projects toward the coracoid. This is located at the anterior inferior acromion. A subacromial keel osteophyte is an acromial spur that causes severe damage to the bursal cuff and is shaped like the keel of a sailboat. The spur is found along the anterior lateral edge of the acromion, between the lateral border of the acromion and the acromioclavicular joint, and extends posteriorly to the middle of the acromion undersurface.

Location of the acromial spur is also important, occurring along the medial, lateral, and anterior aspects of the acromion with anterolateral spurs having a closer association with cuff pathology.

**FIGURE 21.** Coracoacromial ligament thickening. Sagittal T1-weighted image with thickening of the coracoacromial ligament at the coracoid attachment (solid arrows) measuring > 6 mm.

**FIGURE 22.** Subacromial decompression. Coronal PD fat-saturated image (A) showing flattening of the undersurface of the acromion (asterisk) consistent with acromioplasty. Also visualized is partial thickness articular surface intermediate signal (solid arrows) consistent with chronic partial tear of the supraspinatus tendon. Hyperintense bursal signal (dashed arrows) is consistent with mild subacromial/subdeltoid bursitis. Sagittal T1-weighted image (B) of the same patient showing thinning of the coracoacromial ligament near the coracoid attachment (solid arrow), which is consistent with a prior ligamentous release.
Subacromial spurs are contiguous with the cortex of the acromion undersurface with signal isointense to adjacent bone marrow on MRI (Figure 17). Acromion shape has been implicated in impingement with a classification scheme devised by Bigliani et al, as described previously. Only type III has shown a common association with impingement and rotator cuff tears, by most authors. Impingement and rotator cuff tears attributed to a type III acromion are best appreciated on the coronal and sagittal sequences.\textsuperscript{20,35} The slope and angle of the acromion is also associated with extrinsic impingement. Studies have indicated that low lateral acromial angle was seen with a higher incidence of impingement. Lateral acromial angle is calculated by a vertical line lateral to the glenoid and a horizontal line parallel to the acromion surface (Figure 18). Lateral acromial angle can be seen effectively on the coronal MR image with an angle < 70 degrees associated with cuff pathology.\textsuperscript{20,36}

Evaluating an os acromiale and its contribution to impingement can be challenging. On coronal and sagittal planar imaging, an os acromiale may be mistaken for a normal acromioclavicular joint. Axial imaging, usually the most cephalad image, is ideal for identifying an os acromiale. High-signal intensity on T2-weighted images may be appreciated at the synchondrosis, which may represent degenerative changes and/or instability of the os acromiale (Figure 19).\textsuperscript{21,37}

Acromioclavicular joint osteoarthritis may result in inferior projecting osteophytes or fibrosis around the joint capsule, potentially causing impingement. Inferior spurring of the distal clavicle and acromion can also cause impingement, with an inferior acromial spur associated more often with clinical symptoms. MRI appearance varies and acromioclavicular arthritis may demonstrate marrow edema (which will have high signal intensity on T2-weighted sequences), subchondral cysts, sclerosis, and/or erosions.\textsuperscript{38}

Coracoacromial ligament (CAL) pathology has been extensively studied with regard to impingement syndrome.\textsuperscript{39} The CAL is susceptible to pathological degeneration, which can thicken the ligament, greater than its normal thickness of 2 to 5.6 mm. This thickening, and associated subacromial osteophytes at the ligamentous attachment, can cause impingement on the supraspinatus tendon (Figures 20, 21).\textsuperscript{40}

**Treatment**

Of the nonoperative treatment options, studies have shown that exercise therapy combined with other noninvasive treatments have higher efficacy with regard to pain score.\textsuperscript{31} Exercise therapy and localized drug injections showed better positive effects on pain score than any other nonoperative treatment options.\textsuperscript{41,42}

Operative treatment options for extrinsic shoulder impingement are usually related to the underlying etiological cause, and are usually reserved for cases of failed conservative treatments. One of the more common surgical procedures is subacromial decompression, which involves a subacromial bursectomy, release or retraction of the coracoacromial ligament, and removal of the subacromial spurs (Figure 22). This can be performed either as open surgery or arthroscopically. Some studies have shown that an arthroscopic approach has a better efficacy, while others show no significant difference in long-term outcome. However, it is generally accepted that arthroscopy has less of an economic burden and should be the preferred method.\textsuperscript{43,44}

CAL release is a controversial topic and resection can lead to biomechanical alteration with regard to humeral head movement. Therefore, partial release has been advocated with good long-term outcomes.\textsuperscript{39,45} Limited studies have evaluated bursectomy alone vs. standard subacromial decompression, with no significant difference in clinical results between the procedures.\textsuperscript{46} Acromioclavicular joint arthritis alone can be a source of pain, but has also been implicated as a cause of impingement. Distal clavicular resection, or the Mumford procedure, has been shown to improve symptomatic impingement with related acromioclavicular arthropathy as an adjunct to subacromial decompression.\textsuperscript{47,48} Os acromiale treatment includes conservative measures, although patients with persistent symptomatic or functional deficits can benefit from surgical options. One of the more common procedures is an osseous fusion of the os acromiale combined with internal fixation and tension banding. This is usually followed by acromioplasty of the new acromio-acromion joint, ie, a 2-stage fusion.\textsuperscript{49}

**Conclusion**

Extrinsic shoulder impingement is a common cause of shoulder pain and frequently seen in clinical practice. Imaging plays an important role in identifying the cause of impingement and also in guiding clinicians with treatment planning. Understanding the anatomy, underlying mechanics, and MRI characteristics of rotator cuff impingement will allow for prompt recognition and ultimately improve outcomes.

**References**

15. Flecker H. Roentgenographic observations of the times of appearance of epiphyses and their fusion with the diaphyses. J Anat 1932;67(Pt 1):118-164.3.