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REVIEW ARTICLE

Adhesive capsulitis: review of imaging findings, pathophysiology, clinical presentation, and treatment options

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Abstract

Adhesive capsulitis, commonly referred to as "frozen shoulder," is a debilitating condition characterized by progressive pain and limited range of motion about the glenohumeral joint. It is a condition that typically affects middle-aged women, with some evidence for an association with endocrinological, rheumatological, and autoimmune disease states. Management tends to be conservative, as most cases resolve spontaneously, although a subset of patients progress to permanent disability. Conventional arthrographic findings include decreased capsular distension and volume of the axillary recess when compared with the normal glenohumeral joint, in spite of the fact that fluoroscopic visualization alone is rarely carried out today in favor of magnetic resonance imaging (MRI). MRI and MR arthrography (MRA) have, in recent years, allowed for the visualization of several characteristic signs seen with this condition, including thickening of the coracohumeral ligament, axillary pouch and rotator interval joint capsule, in addition to the obliteration of the subcoracoid fat triangle. Additional findings include T2 signal hyperintensity and post-contrast enhancement of the joint capsule. Similar changes are observable on ultrasound. However, the use of ultrasound is most clearly established for image-guided injection therapy. More aggressive therapies, including arthroscopic release and open capsulotomy, may be indicated for refractory disease, with arthroscopic procedures favored because of their less invasive nature and relatively high success rate.

Keywords Frozen shoulder · Adhesive capsulitis · Coracohumeral ligament · Magnetic resonance imaging · Subcoracoid fat triangle · Glenohumeral joint capsule

Introduction

Painful limited range of motion (ROM) of the shoulder was first described as a clinical entity in 1872, when Duplay termed the phenomenon "scapulohumeral periarthritis" [1]. This same phenomenon was termed "frozen shoulder" as early as 1934, by Codman, and is a colloquialism that is still in use today [2, 3]. Neviaser further expanded upon this concept in 1945 and introduced the term "adhesive capsulitis" to refer to a thickening of the glenohumeral joint capsule [4]. Current practice guidelines define adhesive capsulitis as a progressive pain syndrome with accompanying decreases in both active and passive ranges of motion about the glenohumeral joint [5, 6]. Management is typically conservative, although more invasive therapies may be considered in the case of refractory disease.

In spite of the fact that adhesive capsulitis has historically been considered a clinical diagnosis of exclusion, advancements in arthrography in the late twentieth century and, more recently, magnetic resonance imaging (MRI) and ultrasound, have allowed for visualization of previously unresolved confirmatory findings that may aid in the proper diagnosis and treatment of this condition [7–10]. These include thickening of the coracohumeral ligament (CHL) and joint capsule (particularly the axillary pouch and rotator interval), and effacement of the subcoracoid fat on MRI. The use of ultrasound, even though still somewhat controversial for the diagnosis of adhesive

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capsulitis, does play a role in the treatment of adhesive capsulitis through image-guided intra-articular injection [11].

Epidemiology

Adhesive capsulitis is thought to afflict between 2 and 5% of the general population, with women affected more frequently than men [12]. Individuals of middle age are most often affected, typically during the 5th to 7th decades of life [13, 14]. Diabetes mellitus is a major predisposing risk factor. In patients with diabetes mellitus, there is a prevalence of adhesive capsulitis of 13.4% (which is five times higher than that of the general population) and 30% of patients with adhesive capsulitis have diabetes mellitus [15]. Other co-morbidities include thyroid and rheumatological diseases, hypoadrenalism, Parkinson's disease, cardiopulmonary disease, cerebrovascular disease, and, in cases of secondary adhesive capsulitis, previous trauma or surgery [16–21]. There is also an association with Dupuytren's contracture and trigger finger [22], and some evidence for an association with disorders of dyslipidemia [23]. Interestingly, adhesive capsulitis tends to affect the nondominant extremity, although involvement of the dominant shoulder is seen in up to 30% of cases [24].

Clinical presentation

The diagnosis of adhesive capsulitis is often made clinically and coincides with the gradual onset of shoulder pain and limited ROM with respect to external rotation and forward flexion. At present, such a diagnosis does not require imaging evaluation; imaging is principally used to exclude other causes of shoulder pain, such as arthritis or rotator cuff or labral tears [8]. Limitations in passive and active external rotation are particularly characteristic of this condition. The clinical differential diagnosis includes rotator cuff impingement, calcific periarthritis, and capsular inflammation related to inflammatory arthritis or osteoarthrosis [25].

The four stages of adhesive capsulitis disease progression initially defined by Neviaser in 1945 have since been modified to include correlations with arthroscopic findings (Table 1) [4, 6, 10, 26]. Patients first experience pre-freezing (pre-adhesive) pain, particularly at night, followed by freezing, frozen, and thawing phases coinciding with loss and eventual return of ROM [25]. In the case of primary adhesive capsulitis, the disease is usually self-limiting, and typically lasts 18–24 months [6]. However, persistent symptoms and movement restriction beyond 3 years have been reported in up to 40% of patients, with up to 15% of patients suffering permanent disability [27]. In modern-day clinical practice, the Lundberg definition and classification of primary and secondary frozen shoulder are commonly used, with the former arising either idiopathically or in the setting of diabetes mellitus, and the latter arising secondary to previous trauma, surgery, or underlying arthritis (Table 2) [28].

Table 1 Clinical stages of adhesive capsulitis

Stage	Name	Symptoms	Arthroscopic findings
1	Pre-freezing stage	Limited range of motion and pain with symptoms occurring at 0–3 months	Erythematous/fibrinous synovium
2	Freezing stage	Severely restricted range of motion and pain occurring 3–9 months (patients usually present during this stage)	Red, thick synovium with contracted rotator interval and tight axillary recess with adhesions
3	Frozen stage	Severe stiffness, minimal pain, occurring at 9–15 months	Pink synovium with contracted axillary pouch and tight joint space
4	Thawing stage	Improving range of motion, minimal pain	Tight joint space but no evidence of synovitis

Relevant anatomy of the glenohumeral joint capsule

Rotator interval

Strictly speaking, the term rotator interval can be used to refer to one of two triangular-shaped tendinous gaps contained within capsular tissue of the rotator cuff [29]. The anterior rotator interval is the larger gap between the subscapularis and supraspinatus tendons and the posterior rotator interval is the gap between the supraspinatus and infraspinatus tendons. Convention dictates that when the term rotator interval is used alone, it refers to the anterior rotator interval. The rotator interval is the triangular space bounded by the superior border of the subscapularis inferiorly, the anterior border of the supraspinatus tendon superiorly, and the base of the coracoid process medially (Fig. 1). The rotator interval contains the long head of the biceps tendon (LHBT). The roof is formed by the CHL, superior glenohumeral ligament (SGHL), and rotator interval capsule. The floor is the articular cartilage of the humeral head. The subcoracoid fat triangle (pad) fills the intervening gap between the rotator interval joint capsule, CHL, and coracoid process [30]. Although its anatomical functionality has not been fully elucidated, it is known that the rotator interval bestows structural support during dynamic shoulder motion. Compromise of rotator interval integrity has been shown to contribute to glenohumeral contractures and joint instability, in addition to destabilization of

Table 2 Adhesive capsulitis Lundberg definition and classification

Type	Criteria
Primary	Total shoulder elevation ≤135°
	Limited range of motion only at glenohumeral articulation
	Exclusion of other etiologies known to result in decreased range of motion
Secondary	Similar restricted range of motion
	Following trauma, surgery, or with underlying arthritis or other known cause

the biceps pulley system owing to its proximate association with the intra-articular portion of the LHBT [31, 32].

Coracohumeral ligament

The CHL originates at the lateral aspect of the base of the coracoid process of the scapula and has two bands, one of which inserts onto the anterior edge of the supraspinatus tendon and greater tuberosity and another that inserts onto the superior fibers of the subscapularis, transverse humeral ligament, and lesser tuberosity (Fig. 1). The distal fibers of the CHL intimately associate with those of the SGHL to form a suspensory sling around the LHBT that is referred to as the "biceps pulley" (also: pulley sling, biceps reflection pulley; Fig. 2) [32–35]. The CHL limits medial subluxation when the arm is abducted and externally rotated, and is thus lax with adduction and internal rotation [29, 33]. Although anatomical discussions often consider both the CHL and the SGHL together as one functional unit that lends stability to the rotator interval capsule, this review focuses on imaging of the CHL, visualized abnormalities of which are more clearly implicated in the pathogenesis of adhesive capsulitis [29, 33].

Axillary pouch

The axillary pouch is the inferior glenohumeral joint capsule that lies between the anterior and posterior bands of the inferior glenohumeral ligament (Fig. 3). Of note, the inferior glenohumeral ligament provides resistance to both anterior and posterior shoulder dislocations by virtue of its attachments to the glenoid fossa of the scapula and humerus [36]. This region of the shoulder can be accessed arthroscopically during invasive manipulations in the management of refractory disease, although the anatomical complexity of the inferior glenohumeral ligament complex and the adjacent axillary neurovascular bundle poses a challenge to therapeutic intervention [37].

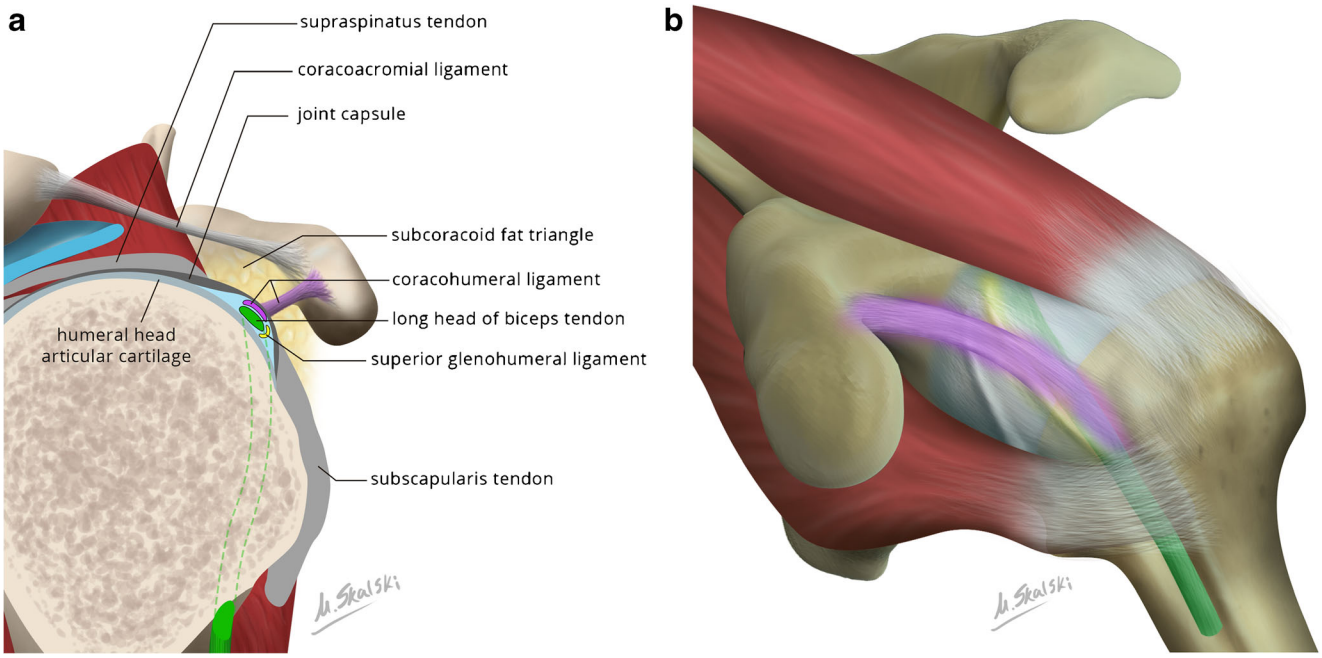


Fig. 1 Illustrations of the rotator interval and associated structures. **a** The rotator interval represents the space between the supraspinatus and subscapularis tendons and the coracoid process, containing the intra-articular portion of the long head of the biceps tendon. **b** The rotator interval capsule is reinforced by the coracohumeral ligament (*purple*) along the bursal side and the superior glenohumeral ligament (*yellow*) along the articular side, which combine to form a pulley for the long head of the biceps tendon (*green*)

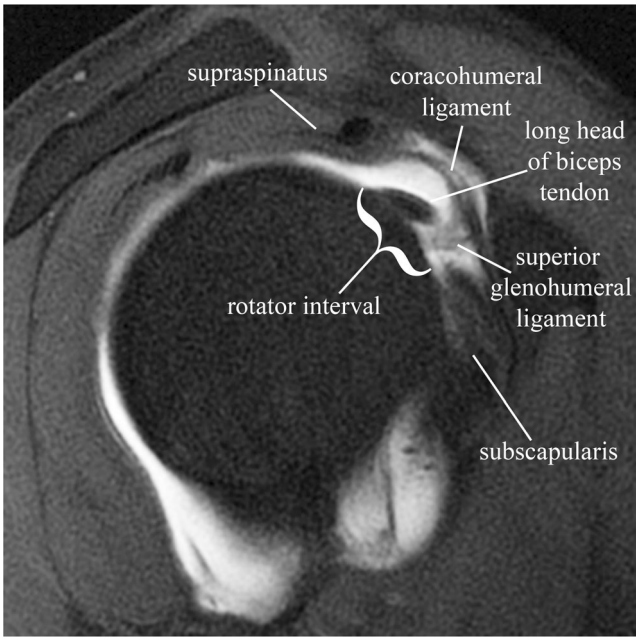


Fig. 2 Sagittal oblique MR arthrography T1 fat saturation (FS) image shows the rotator interval between the supraspinatus and subscapularis tendons containing the long head of the biceps tendon. The coracohumeral and the superior glenohumeral ligaments form a sling around the biceps tendon, referred to as the biceps pulley

Pathophysiology

In adhesive capsulitis, proliferating fibroblasts are characteristically seen on biopsy of the rotator interval, in addition to the presence of chronic inflammatory cells [25]. These fibroblasts tend to be seen among mixed type I and type III collagen, a finding that historically led to the classification of adhesive capsulitis as a fibrotic disorder akin to Dupuytren's disease. Observed transformation to the smooth muscle (myofibroblast) phenotype is thought to be associated with capsular contraction [38, 39]. New nerve growth in the capsuloligamentous complex in patients with adhesive capsulitis may explain the heightened pain response [40].

These changes have been associated with increased capsular collagen overall, in addition to elevated levels of inflammatory cytokines in the synovium, namely interleukin (IL)-6 and vascular endothelial growth factor [38, 39]. Subacromial bursal tissues in adhesive capsulitis patients were found to have substantially elevated expression of IL-1α, IL-1β, tumor necrosis factor (TNF)-α, and cyclooxygenase (COX)-1 and COX-2 when compared with controls [41]. Elevated serum levels of multiple inflammatory cytokines, including tissue transforming growth factor (TGF)-β, platelet-derived growth factor (PDGF), and hepatocyte growth factor have been implicated in early inflammatory processes. Notably, Watson et al. demonstrated that intra-articular injection of TGF-β1 into the knee joints of immunocompromised rats was capable of inducing arthrofibrosis and chondrometaplasia in the setting of adhesive capsulitis within 5 days post-injection [42].

Biceps tendinopathy and tenosynovitis is a concomitant finding in many cases of adhesive capsulitis, and can be a contributor to pain [3]. It has previously been hypothesized that tendinopathy of the LHBT might arise as a consequence of abnormal contact with bony irregularities in the region, including a supratubercular ridge of Meyer, irregularities of the floor of the intertubercular groove, or with raised margins of the humeral head or lesser tuberosity [43, 44].

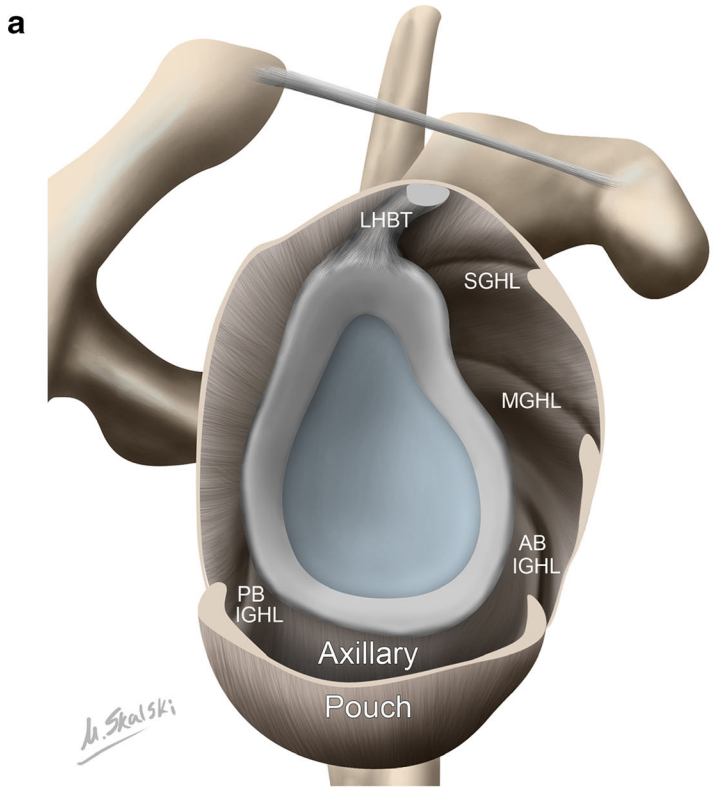
Imaging findings

Plain radiographs

The primary utility of plain radiographs lies in its ability to differentiate between primary adhesive capsulitis and glenohumeral arthritis or calcific tendinosis as alternative causes for the patient's presenting shoulder pain. Osteopenia occurring over a short period can be seen with adhesive capsulitis related to disuse and the inflammatory process [45]. Plain film images in the setting of adhesive capsulitis are often otherwise of little aid.

Arthrography

Fluoroscopy-guided glenohumeral arthrography demonstrates a number of findings characteristic of adhesive capsulitis, including decreased capsular distension, obliteration of the axillary recess, and early contrast extension to the biceps tendon sheath (Fig. 4). A glenohumeral joint volume less than 10 mL is suggestive of adhesive capsulitis, whereas a normal glenohumeral joint can be easily distended to 14 mL [46, 47]. In patients with adhesive capsulitis, these injections can be very painful compared with routine glenohumeral joint injections, both when the needle penetrates the joint capsule and during the injection itself. The volume of the axillary recess specifically was also shown to be significantly decreased compared with controls (mean 0.53 vs 0.88 mL, $p = 0.03$) as a consequence of joint capsule contraction [48]. In addition to decreased distention, there may be filling defects best seen in the axillary recess, indicating synovitis. The decreased capsular distension may also manifest as early extracapsular extension (extravasation) of contrast medium through areas of weakening in the joint capsule, such as the subscapularis recess. Conventional arthrography for the evaluation of adhesive capsulitis has largely been replaced by MRI and MRA.



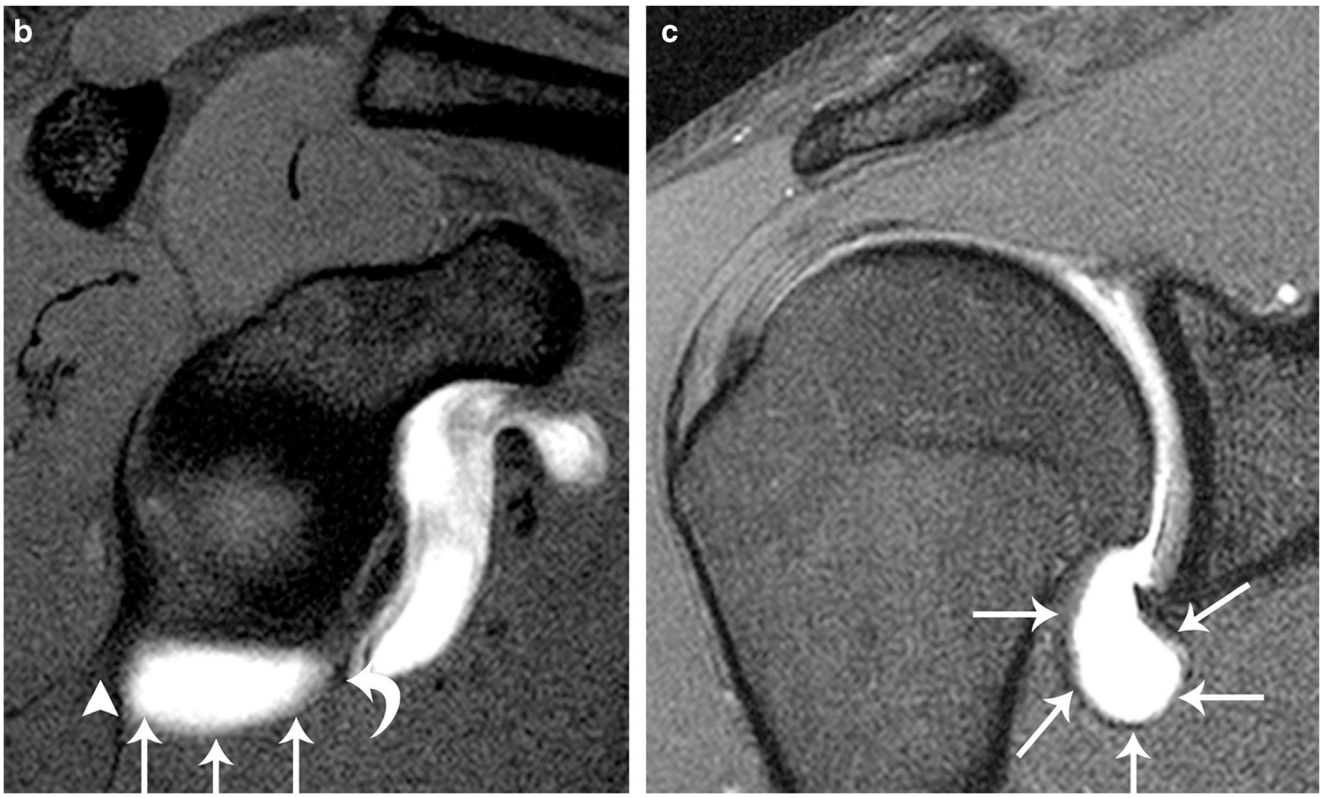


Fig. 3 **a** Illustration and **b** sagittal oblique and **c** coronal oblique MR arthrography T1 FS images show the normal axillary pouch (*arrows*, **b, c**) between the anterior band (*AB*, *curved arrow*, **b**) and the posterior band (*PB*, *arrowhead*, **b**) of the inferior glenohumeral ligament (*IGHL*). The long head of the biceps tendon (*LHBT*), superior glenohumeral ligament (*SGHL*), and middle glenohumeral ligament (*MGHL*) are also illustrated

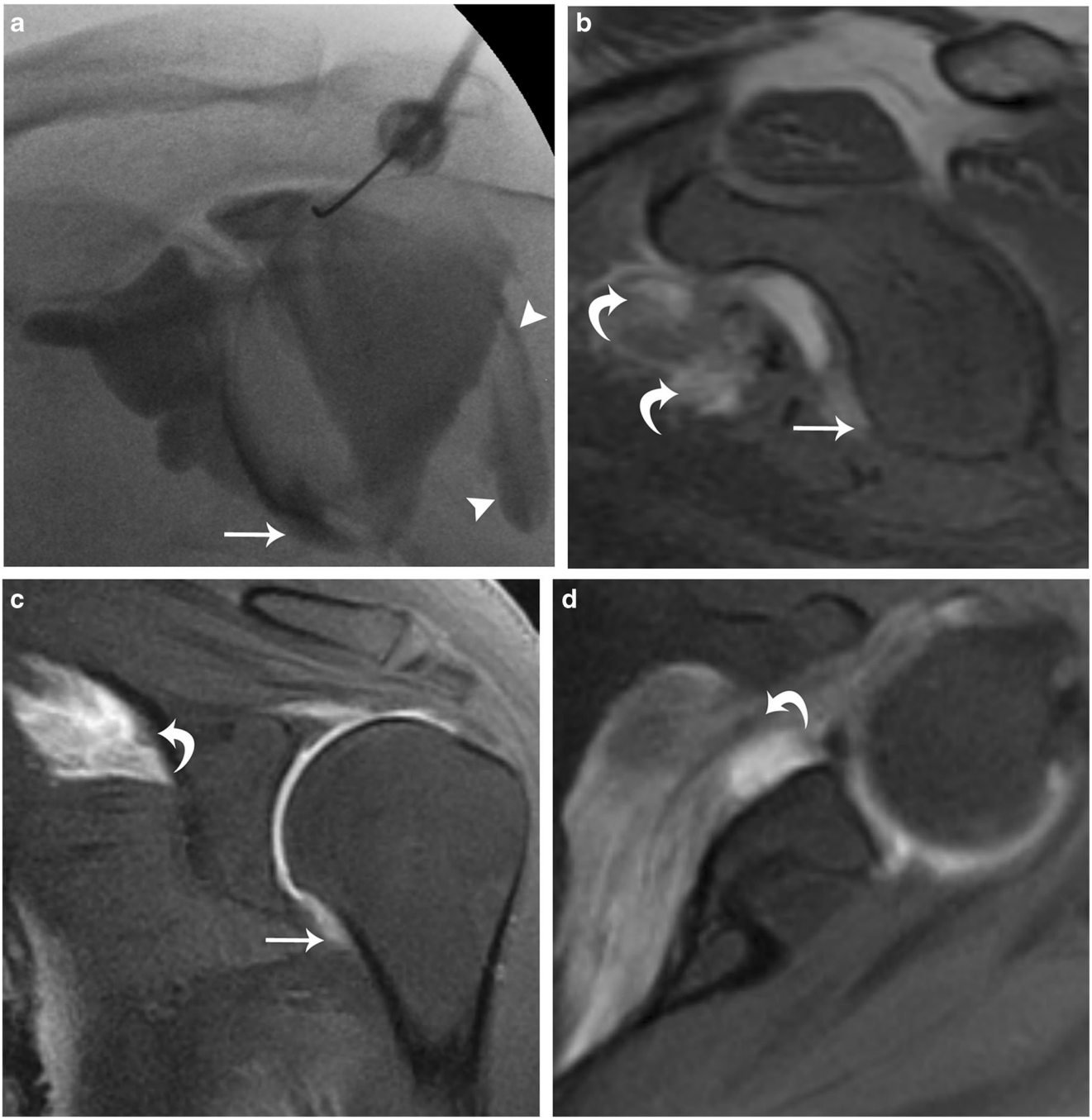


Fig. 4 A 55-year-old woman with left shoulder pain. **a** Fluoroscopically guided arthrogram demonstrates restricted joint capacity (only 8 cc of contrast medium could be injected) with diminished axillary recess (*arrows*) and contrast pooling in the biceps tendon sheath (*arrowheads*). **b** Sagittal oblique, **c** coronal oblique, **d** and axial T1 FS images from a subsequent MR arthrogram show the diminished capacity of the axillary recess and interval extravasation of contrast medium through the rotator interval along the superior subscapularis muscle belly (*curved arrows*), which was not seen during the arthrogram procedure

Magnetic resonance imaging/magnetic resonance arthrography

A number of signs of adhesive capsulitis on MRI and MRA have been reported, which we will discuss separately in sections on each anatomical region involved. Routine MRI of the shoulder without intra-articular or intravenous gadolinium can be used to diagnose all stages of adhesive capsulitis, including stage 1 when clinical findings may be subtle [6]. As a non-invasive test, MRI is considered the routine modality for adhesive capsulitis, especially in patients with less severe clinical symptoms who might be misdiagnosed with rotator cuff tears, bursitis, or other conditions. MRA and MRI with intravenous contrast medium may demonstrate a higher sensitivity

and/or specificity for some imaging findings of adhesive capsulitis [49–51]. However, these examinations are less commonly performed because of their more invasive nature.

Rotator interval/coracohumeral ligament

Thickening of the rotator interval capsule and CHL on MRA is suggestive of a diagnosis of adhesive capsulitis [48]. Both structures are best visualized on sagittal oblique images, although coronal oblique and axial images may help to confirm suspected CHL thickening (Fig. 2) [52]. Although these findings are highly specific for adhesive capsulitis, sensitivity remains low. In a study by Mengiardi et al., rotator interval capsule thickness ≥ 7 mm had a sensitivity of 64% and a specificity of 86%, and CHL thickness ≥ 4 mm had a sensitivity of 59% and a specificity of 95% for detecting adhesive capsulitis (Fig. 5) [48]. More recent data propose that a 3-mm threshold of CHL thickness provides the highest accuracy for adhesive capsulitis diagnosis by MRA [53]. Synovitis of the rotator interval may also be seen, as demonstrated by the intermediate to low T1 signal thickening, hyperintense edema on fluid-sensitive sequences, and post-contrast enhancement of the rotator interval capsule [48, 52]. On non-arthrographic MRI, CHL thickness and rotator interval enhancement retain high specificity, but sensitivity may be lower than with MRA [54].

Axillary pouch

Capsular and synovial thickening (as quantified by mean axillary pouch thickness) related well to clinical stage in patients with adhesive capsulitis, and was shown to be negatively correlated with ROM in external rotation [6, 8]. It has been proposed that signal hyperintensity on T2-weighted images may correlate with hypertrophic, hypervascular synovitis and inflammatory changes seen in stages 1 and 2 of adhesive capsulitis progression (Table 1) [6]. Enhancement after the intravenous administration of gadolinium-based contrast medium was found to positively correlate with pain intensity (OR = 0.78, $p < 0.05$) [8]. However, the utility of relying on the visualization of abnormalities of the axillary pouch alone in support of the diagnosis of suspected adhesive capsulitis is unclear, as conflicting data with respect to the sensitivity and specificity of capsular and synovial thickening on MRI and MRA in the detection of adhesive capsulitis have been reported in the literature [5, 48, 50, 55, 56].

Nevertheless, it has been proposed that a capsular and synovial thickness > 4 mm visualized on non-arthrographic coronal oblique T2-weighted images grants a sensitivity of 70% and a specificity of 95% for the detection of adhesive capsulitis (Fig. 6) [5]. Evaluation of capsular and synovial thickening is preferred on coronal oblique T2-weighted images without fat suppression, as T1-weighted images tend to overestimate capsular thickening in control subjects. With MRA, Jung et al. found that an axillary pouch thickness of

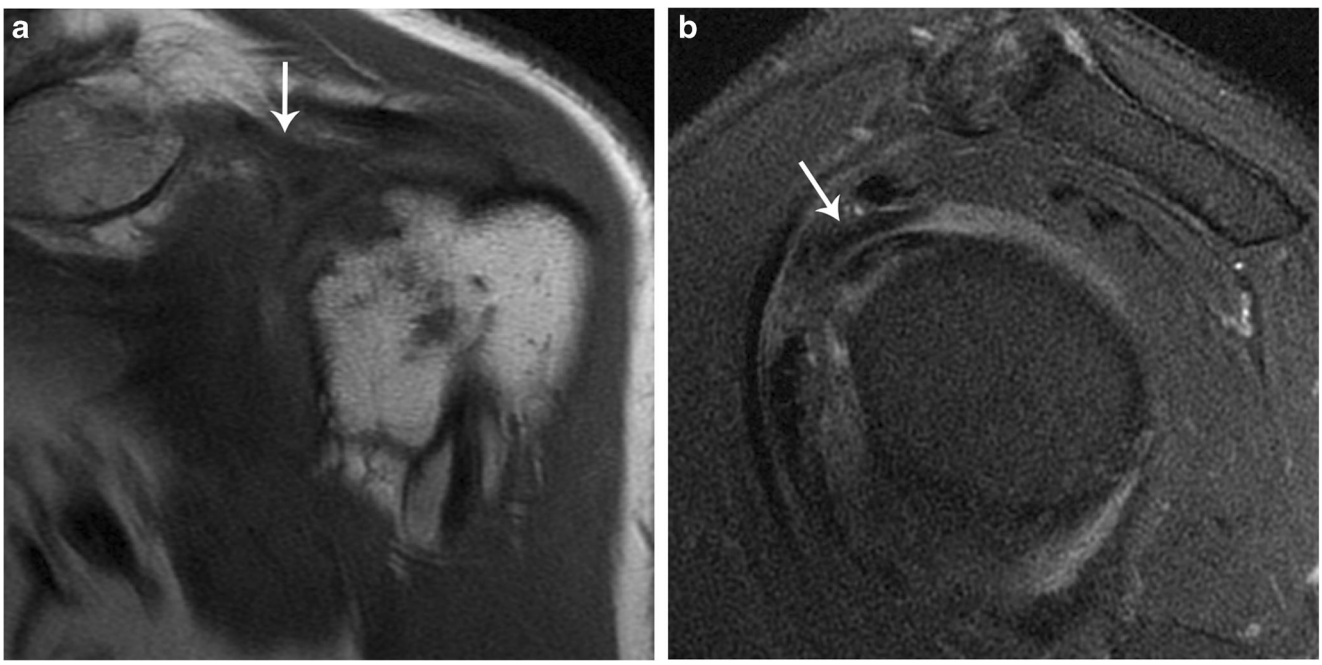


Fig. 5 A 65-year-old man with shoulder pain and a limited range of motion. **a** Coronal oblique T2 and **b** sagittal oblique T2 FS MR images demonstrate thickening (up to 4.2 mm) and intermediate signal intensity of the coracohumeral ligament (*arrows*), consistent with adhesive capsulitis

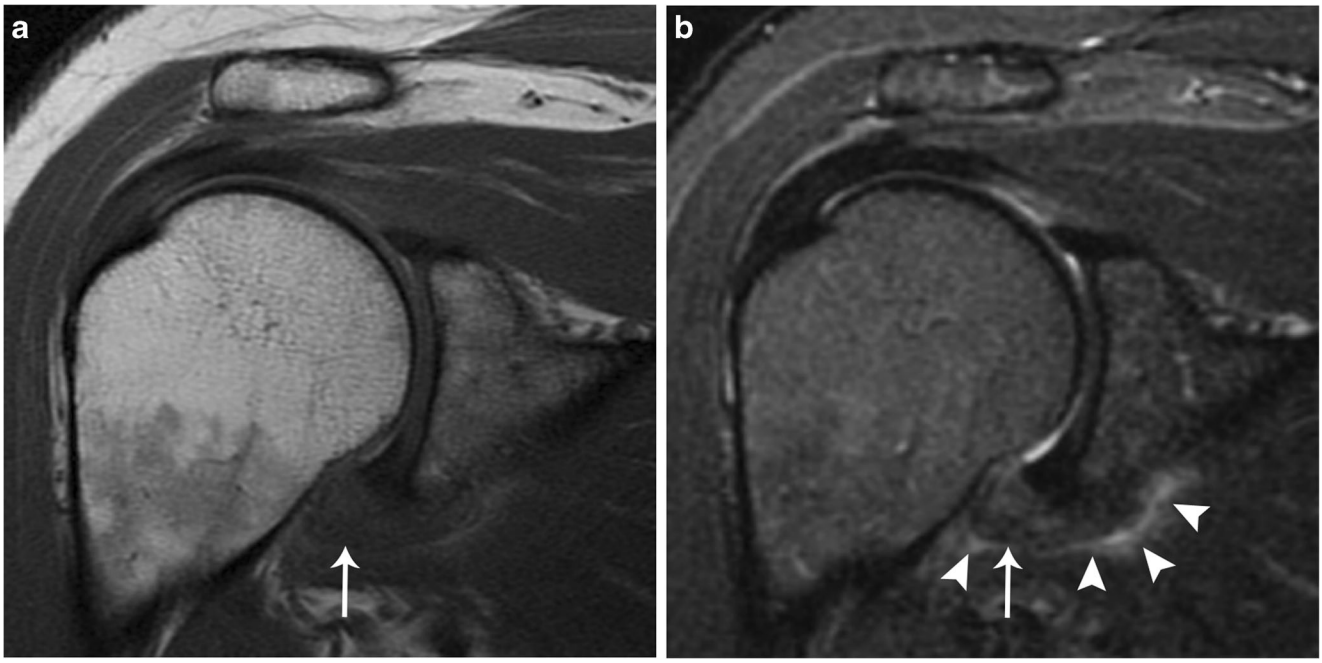


Fig. 6 A 58-year-old woman with right shoulder pain and a limited range of motion. **a** Coronal oblique T1 and **b** short tau inversion recovery MR images demonstrate thickening (up to 10 mm) and intermediate signal intensity of the axillary pouch (*arrows*) with hyperintensity/edema of the adjacent soft tissues (*arrowheads*), consistent with adhesive capsulitis

greater than 3 mm (compared with 4 mm on MRI without arthrography) generates a diagnostic accuracy of 89% for adhesive capsulitis, as measured on T2-weighted coronal oblique images [50].

Furthermore, signal hyperintensity of the axillary pouch/inferior glenohumeral ligamentous complex on MRI using non-arthrographic T2-weighted fat-suppressed sequences was both highly sensitive (85.3–88.2%) and specific (88.2%) for the presence of adhesive capsulitis, with low interobserver variability ($\kappa = 0.85$) [54]. Similarly, signal analysis for enhancement of the inferior glenohumeral ligament was also comparably sensitive (82.3–94.0%) and specific (88.2%) with excellent interobserver agreement ($\kappa = 0.82$; Fig. 7). Axillary pouch enhancement additionally correlates with pain intensity and can be graded as mild (subtle enhancement), moderate (strong enhancement involving less than half of the capsule circumference), or severe (strong enhancement involving over half the capsule circumference; Fig. 8) [8]. It should be noted that the entirety of the glenohumeral joint capsule and glenohumeral ligaments may be involved with adhesive capsulitis, but the rotator interval capsule and axillary pouch are the most commonly evaluated areas at imaging.

Subcoracoid fat triangle

The triangular space bounded by the coracoid process, joint capsule, and CHL is characterized by a well-defined region of fat signal intensity in normal shoulders [30]. Complete obliteration of the subcoracoid fat triangle on MRA was first reported by Mengiardi et al. to be a poorly sensitive (32%) yet highly specific (100%) finding for adhesive capsulitis. Dubbed the "subcoracoid triangle sign," this finding is visualized well on sagittal oblique images and easy to assess on routine scans (Fig. 9) [48]. These data were later corroborated by multiple authors with similar specificity for adhesive capsulitis [3, 53]. The subcoracoid triangle sign was not found to correlate well with clinical impairment [8, 30, 53]. Given that this finding is more commonly observed in clinical stages 1 and 2, it is thought to be related to inflammatory changes seen early in disease progression (Table 1) [30].

Comparison with computed tomography arthrography

One study has investigated the utility of computed tomographic (CT) arthrography in the diagnosis of adhesive capsulitis. Consistent with previously reported findings on MRI and MRA, thickening of the axillary pouch was observed on CT arthrography in the setting of adhesive capsulitis. Using cut-off values of 4 mm for the medial wall and 4.9 mm for the lateral wall, Cerny et al. found sensitivities of 100% and 81%, and specificities of 75% and 85% respectively [57]. Whereas other studies report abnormal width, signal intensity, and enhancement of the rotator interval on MRI, rotator interval obliteration (sensitivity = 72.2%, specificity = 75%) was observed without reduction in width on CT arthrography [50, 54, 57]. Thickening of the CHL was observed on CT arthrography with a sensitivity of 90% and 75% when using a cut-off value of 3.2 mm. However, it was noted that

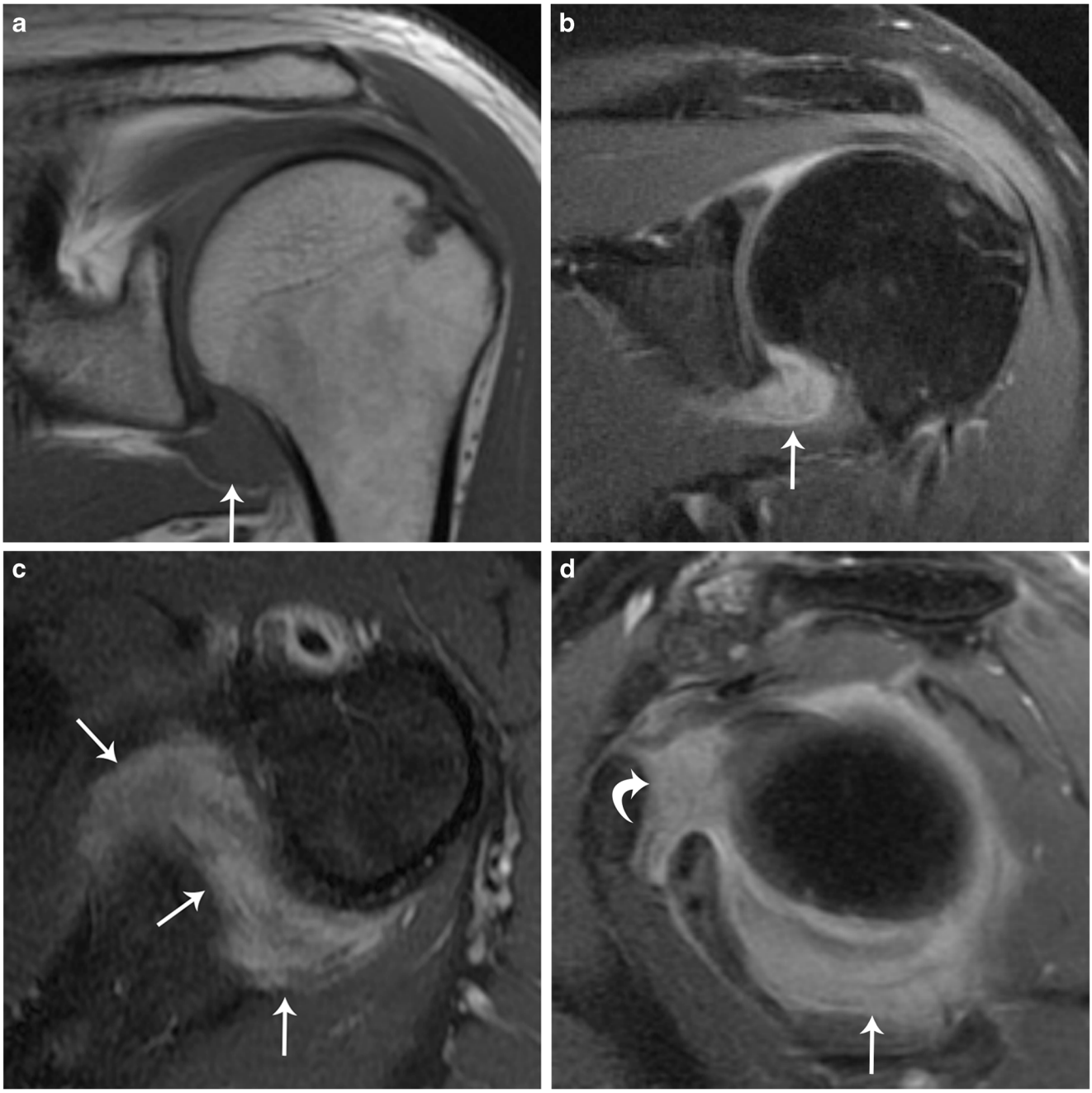


Fig. 7 A 61-year-old man with left shoulder pain and a limited range of motion. **a** Coronal oblique T1 pre-contrast and **b** coronal oblique, **c** axial, and **d** sagittal oblique T1 FS post-contrast MR images show severe thickening and enhancement of the glenohumeral joint capsule, particularly the axillary pouch (*arrows*) and the rotator interval capsule (*curved arrow*, **d**), suggestive of severe adhesive capsulitis

visualization of the CHL was difficult on CT arthrography in cases where the surrounding fat plane was infiltrated, and may have confounded the measurements obtained [57].

Ultrasound

Ultrasound is increasingly being used in the diagnosis of adhesive capsulitis, with similar findings and anatomical changes that have been detailed for MRI and MRA. In comparison to MRI, ultrasound has the notable advantages of shorter examination times, lower cost, and wider availability to patients with non-MR-compatible implants [27]. Homsi et al. found an increase in CHL thickness in patients with diagnosed adhesive capsulitis (3 mm) compared with shoulders with pain from other causes (1.4 mm) and asymptomatic shoulders (1.3 mm) [58]. Likewise, Michelin et al. found thickening of the inferior glenohumeral capsule in adhesive capsulitis patients (4.0 mm) compared with controls (1.3 mm) [59]. Kim

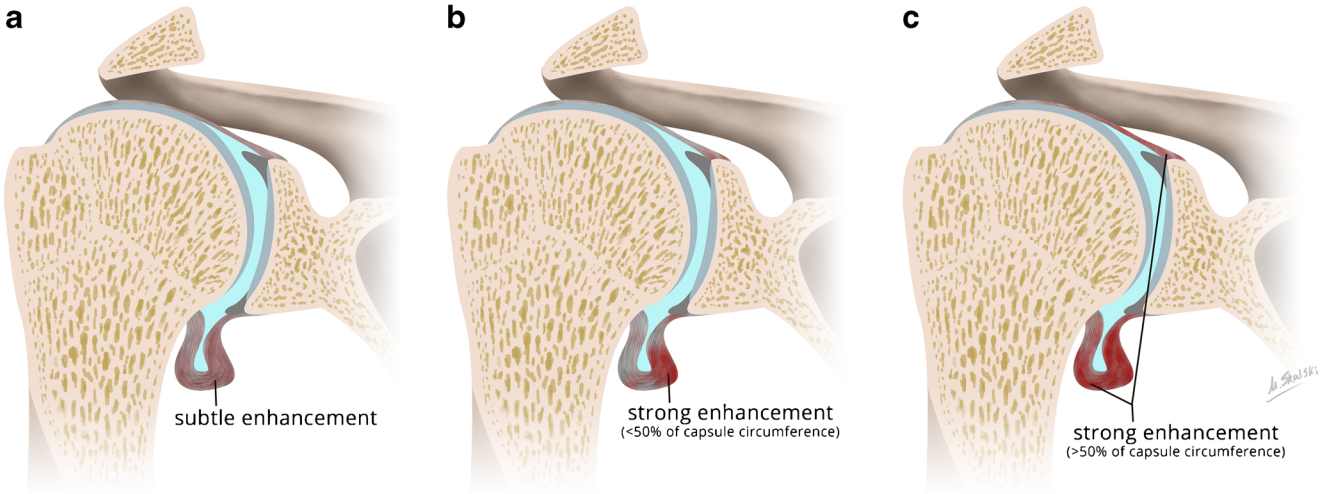


Fig. 8 Illustrations demonstrating the grading of adhesive capsulitis on contrast-enhanced MRI. **a** Axillary pouch enhancement can be mild (subtle enhancement), **b** moderate, strong enhancement involving less than half of the capsule's circumference), or **c** severe, strong enhancement involving over half the capsule's circumference)

et al. demonstrated significant differences in the ultrasound measurement of axillary recess capsule thickness (including both the humeral and the glenoid sides) between the affected and unaffected shoulders in patients with unilateral adhesive capsulitis (4.4 mm vs 2.2 mm, $p < 0.001$) and this ultrasound measurement correlated with MRI measurement ($p < 0.001$, $r = 0.83$) [60]. Lee et al. reports 100% specificity when hypoechoic soft tissue is detected within the rotator interval, with hypervascularity on power Doppler with a sensitivity of 87–97% [61]. Limitation of subacromial gliding of the supraspinatus tendon on dynamic ultrasound has been shown to be highly predictive of decreased intra-articular injection volume ($r = -0.764$, $p < 0.001$) [62].

Ultrasound arthrography using contrast-enhanced ultrasound (CEUS) and microbubble contrast agents is a relatively new technique that allows for the qualitative assessment of capsular changes. Cheng et al. describe an observation of filling defects in patients with adhesive capsulitis that are believed to be related to irregular thickening and distortion of capsular tissue and synovium, with a sensitivity of 91.1% and a specificity of 86.7% [27]. Additionally, hyperechoic microbubbles

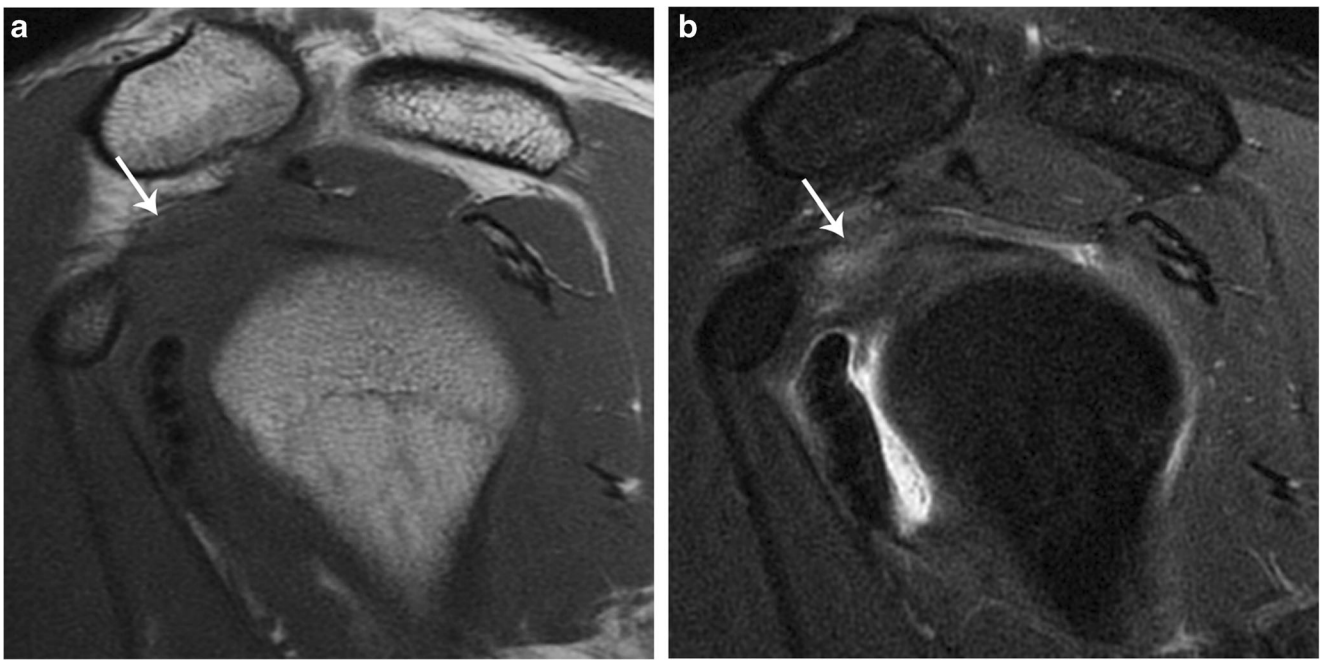


Fig. 9 A 33-year-old man with right shoulder pain and a limited range of motion. **a** Coronal oblique T1 and **b** T2 FS MR images demonstrate effacement of the subcoracoid fat triangle with intermediate signal intensity (*arrows*), consistent with adhesive capsulitis

retained on debris in the joint or on the capsule's subsynovial layer was characteristic of adhesive capsulitis, albeit with lower sensitivity (75.6%) and specificity (77.8%). Taken together, these findings were found to be more sensitive than standard ultrasound for detecting adhesive capsulitis. Extracapsular extension of injected microbubbles was also found to occur more frequently in adhesive capsulitis patients, with a concurrent decrease in the volume of the axillary recess (mean 1.1 vs 1.6 mL, $p < 0.01$).

It should be noted that in their consensus statement, the European Society of Musculoskeletal Radiology does not recommend ultrasound to evaluate patients with suspected adhesive capsulitis [63].

Management

Conservative management is preferred for adhesive capsulitis patients, even though rather effective operative treatment options exist for refractory cases in which non-invasive therapy fails [25]. Both nonsteroidal anti-inflammatory (NSAID) medications and physiotherapy have historically served as first-line treatment options for early disease stages, yet neither NSAIDs nor physiotherapy alone has definitively been shown to be of therapeutic benefit. However, in the case of NSAIDs, there is some evidence to suggest efficacy in short-term pain relief by suppression of COX-1 and COX-2 activity and thus synovitis-related inflammation in early disease stages [39, 41, 64, 65]. Current practice guidelines suggest that NSAIDs might be used for symptomatic relief in combination with other treatment modalities [64]. Physiotherapy is generally not recommended beyond stage 1 (Table 1) [39].

By contrast, intra-articular corticosteroid injections during adhesive capsulitis stages 1 and 2 have empirically demonstrated more rapid improvement in pain and ROM compared with no treatment or physiotherapy alone [39, 64, 66]. A combined regimen of physiotherapy, NSAIDs, and intra-articular corticosteroid injection may be especially helpful early on when pain is a predominant feature [39, 67]. Injection preparations commonly employ a 40-mg dose of triamcinolone or methylprednisolone [11, 68]. Practitioners may elect to co-administer a local long-acting anesthetic, such as bupivacaine, to provide immediate symptomatic relief and avoid overly concentrating the steroid suspension in a single area [69, 70]. Of note, intra-articular hyaluronate injections have also shown promise in the treatment of adhesive capsulitis, demonstrating equivalent clinical outcomes to those achieved by intra-articular corticosteroid injections [71–76].

Image guidance can additionally be used to perform intra-articular hydraulic distension (alternatively known as hydrodilatation or distension arthrography) to treat adhesive capsulitis [77, 78]. This involves the instillation of a large volume (typically 30 mL) of saline with a mixture of corticosteroid, long-acting local anesthetic, and contrast material. Although historically this was done to the point of capsular rupture, capsule preservation improves short-term outcomes [79]. Administration of hypertonic saline may also be more effective than normal saline [80]. Intra-articular hydraulic distension has been shown to improve short-term pain and function, although this benefit is only maintained up to 3 months and diabetic patients have inferior outcomes compared with nondiabetic patients [81].

More invasive management is generally reserved for patients in whom conservative management has failed over a period of at least 2–3 months, in spite of some authors advocating non-operative management for up to 6 months [25, 82]. Manipulation under anesthesia has classically been recommended, as it allows for controlled rupture of capsular contractions and is less invasive than open capsulotomy or arthroscopic release procedures [83]. Manipulation under anesthesia is well tolerated, with improvement in ROM noted within days [83, 84]. Risks of iatrogenic injury including humeral fracture and increased pain due to stretched tissue warrant consideration and can delay recovery times [25, 82]. Some authors found greater improvement in pain relief and ROM for arthroscopic release (also referred to as arthrolysis or adhesiolysis) with manipulation compared with manipulation alone, as arthroscopic release has the added benefit of allowing for concurrent evaluation of relevant anatomical structures [64, 82, 85, 86]. Open capsulotomy, although highly effective, is rarely performed and tends to be reserved for patients in whom arthroscopic release fails [39, 64].

Conclusion

Diagnosis of adhesive capsulitis may be suggested on imaging by several characteristic findings, MRI being the imaging modality of choice. Thickening of the CHL, axillary pouch, and rotator interval joint capsule have good sensitivity and very high specificity, whereas obliteration of the subcoracoid fat triangle has low sensitivity but the highest specificity. T2 signal hyperintensity and post-contrast enhancement of the inferior glenohumeral ligament on fat-suppressed images also demonstrate very high sensitivity and specificity. Similar findings are observable by ultrasound. Image-guided intra-articular injections can help to ameliorate symptoms.

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Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflicts of interest.

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