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SCIENTIFIC ARTICLE

# Anterior capsular abnormality: another important MRI finding for the diagnosis of adhesive capsulitis of the shoulder

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## Abstract

**Objective** To evaluate the usefulness of anterior capsular abnormality, thickening, and abnormal signal intensity on MRI for the diagnosis of adhesive capsulitis of the shoulder.

**Materials and methods** This retrospective study included 29 patients with adhesive capsulitis and 20 controls. Clinical criteria with significant restricted passive motion was used for the diagnosis of adhesive capsulitis. The anterior capsular thickness and signal intensity were evaluated on the thickest portion of anterior glenohumeral joint capsule, located deep to the subscapularis muscle. In addition, the previously known MR findings of adhesive capsulitis, such as humeral and glenoid capsular thickness in axillary recess, maximal axillary capsular thickness, and coracohumeral ligament thickness, were measured. The presence of humeral and glenoid capsular abnormal hyperintensity in axillary recess, abnormal hyperintensity, and obliteration of the subcoracoid fat triangle were also evaluated.

**Results** All MRI findings significantly differed between adhesive capsulitis and controls. Among MR findings, multivariable analysis showed that anterior capsular thickness, maximal axillary capsular thickness, and anterior capsular abnormal hyperintensity were variables that could differentiate adhesive capsulitis from the control group, with odds ratios of 7.97, 17.75, and 12.41, respectively ($p < 0.05$). In ROC analysis, the anterior capsular thickness showed high diagnostic performances with an AUC of 0.897. The cut-off value of anterior capsular thickness at 3.5 mm showed excellent diagnostic accuracy, with sensitivity of 68.97% and specificity of 100%.

**Conclusions** Anterior capsular abnormality, thickening, and abnormal hyperintensity can be used for the diagnosis of adhesive capsulitis of shoulder, in addition to previously known abnormal MRI findings.

**Keywords** Anterior capsular abnormality · Adhesive capsulitis of shoulder · Anterior capsular thickness · Anterior capsular abnormal hyperintensity

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## Introduction

Adhesive capsulitis, or frozen shoulder, is a common disease characterized by shoulder pain and limited range of active and passive glenohumeral motions. The pathogenesis of this disease remains unclear. Other shoulder diseases such as rotator cuff tear, bursitis, and calcific tendinitis often have similar clinical symptoms, which may limit the validity of clinical diagnosis [1, 2]. For this reason, efforts have been made to diagnose adhesive capsulitis accurately with magnetic resonance imaging (MRI), ultrasound, nuclear medicine, and arthrography [3–8]. Of these, MRI has excellent contrast resolution, optimal soft tissue visualization, and multi-plane scanning function. Thus, MR has demonstrated high diagnostic accuracy in detecting a number of features that are suggestive of adhesive capsulitis [9].

Previous MRI studies have shown that capsular thickening and enhancement, abnormal hyperintensity in the axillary recess and rotator interval, thickening of the coracohumeral ligament (CHL), and obliteration of subcoracoid fat triangle are some of the important diagnostic findings of adhesive capsulitis [3, 6, 9–12]. These MRI findings play an important role in the diagnosis of adhesive capsulitis in the early stages of the disease or when the clinical findings are atypical. It has been demonstrated that the rapid and accurate diagnosis of this condition can reduce the morbidity of patients by shortening the duration of joint stiffness with physical therapy and intra-articular steroid injections [13, 14].

Based on histology examinations, tissue samples from patients with adhesive capsulitis have demonstrated a dense collagen matrix as well as a high population of fibroblasts with elevated levels of inflammatory cytokines limited to the anterior portion of the joint capsule [2, 15, 16]. Arthroscopic examination has frequently shown thickening of the anterior portion of the joint capsule, including glenohumeral ligament [17, 18]. The anterior portion of the capsule is also an important structure in arthroscopic capsulotomy for the restoration of range of motion [19, 20]. However, the previous reports have only described abnormal MR findings for CHL, superior glenohumeral ligament (SGHL), and inferior glenohumeral ligament (IGHL) [3, 6, 9–12]. There has been no study investigating the abnormal finding of anterior capsule including the middle glenohumeral ligament (MGHL) and spiral glenohumeral ligament (GHL), located deep to the subscapularis muscle and tendon.

Therefore, the purpose of this study was to evaluate the usefulness of abnormal thickening or hyperintensity of anterior capsule for the diagnosis of adhesive capsulitis of the shoulder.

## Materials and methods

This retrospective study was approved by our Institutional Review Board. Informed consent was waived due to the retrospective nature of this study.

### Inclusion and exclusion criteria

#### Adhesive capsulitis patients

Between January 2016 and December 2016, 219 shoulder MR examinations excluding MR arthrography were performed at our institution. The medical records of these patients were analyzed. The initial inclusion of adhesive capsulitis was based on history and clinical symptoms. Clinical criteria for the diagnosis of adhesive capsulitis included restricted passive motion of greater than 30° in two or more planes of movement compared to the normal contralateral shoulder and gradually increasing shoulder pain that was more severe at rest for at least 1 month, with normal radiographic findings [6, 21–23]. Of the 46 consecutive patients who fulfilled the criteria, using imaging study including MRI as well as clinical evaluation, 17 patients were excluded for the following reasons: bilateral shoulder restricted motion, tear of rotator cuff tendon, calcific tendinitis, rheumatoid arthritis, severe osteoarthritis, and labral lesion. The remaining 29 patients (12 males, 17 females; mean age, 51 years; age range, 30–73 years) with a final clinical diagnosis of adhesive capsulitis were identified and their images were evaluated retrospectively.

#### Control group

The control group was composed of 20 consecutive patients (ten males, ten females; mean age, 49 years; age range, 23–63 years) with normal glenohumeral joints on shoulder MRI, excluding MR arthrography. In the same time period as the adhesive capsulitis group, patients in the control group had been referred for MRI at our institution between January 2016 and December 2016. They showed no restricted shoulder motion and had no history of adhesive capsulitis. Patients in the control group were referred for the following reasons: evaluation of shoulder pain (12 patients), soft-tissue masses (lipoma, five patients; hematoma, one patient), and bone lesions (two patients).

#### Clinical assessment

All patients, including those in the control group, underwent physical examination prior to MRI examination by one physician with 22 years of experience in the shoulder clinic. Using a universal goniometer, range of motion (ROM), including external rotation, forward flexion, and abduction, was evaluated. The external rotation was assessed while the shoulder was maintained at 0° of abduction with 90° elbow flexion. ROM for the internal rotation was measured by noting the highest segment of spinal anatomy reached with the thumb. Forward flexion was measured as the maximum arm-trunk angle by the elevation of the arm forward above the head. Abduction was measured as the ability to raise both arms from the side to full abduction (180°) above the head. The mean duration of symptoms was 5.5 months (range, 1–14 months) in the adhesive capsulitis group. The mean interval between clinical assessment and MR imaging was 16 days (range, 4 ~ 48 days).

### MR image acquisition

#### MRI protocol

All patients and the control group underwent the same MRI protocol with a 3-T MRI (Intera Achieva, Philips Healthcare) unit with a dedicated shoulder coil. During imaging, patients were in the supine position with their arms externally rotated

to the maximum extent possible. The following imaging parameters were used: oblique sagittal fat-suppressed proton density (PD) VISTA sequence with spectral attenuated inversion recovery (SPAIR) imaging (TR/TE, 2000/18.6; echo-train length, 140; section thickness, 1.2 mm; matrix, 268 × 267; FOV, 160 × 160 mm), oblique coronal fat-suppressed T2-weighted imaging (TR/TE, 4700/80; echo-train length, 10; section thickness, 3 mm; matrix, 356 × 255; FOV, 160 × 160 mm), oblique coronal T1-weighted imaging (TR/TE, 530/20; echo-train length, 3; section thickness, 3 mm; matrix, 358 × 258; FOV, 160 × 160 mm), oblique sagittal T2-weighted imaging (TR/TE, 3800/80; echo-train length, 16; section thickness, 3 mm; matrix, 356 × 256; FOV, 160 × 160 mm), oblique sagittal T1-weighted imaging (TR/TE, 530/20; echo-train length, 3; section thickness, 4 mm; matrix, 356 × 258; FOV, 160 × 160 mm), axial fat-suppressed PD imaging (TR/TE, 2100/30; echo-train length, 20; section thickness, 3 mm; matrix, 356 × 240; FOV, 160 × 160 mm).

## MR image analysis

The measurements of the relevant parameters were independently performed by two musculoskeletal radiologists, each with 9 years of experience, on a picture archiving and communication system (PACS) workstation (INFINITT, Infinitt Healthcare, Seoul, Korea). Two musculoskeletal radiologists who were blinded to the clinical information independently evaluated all variables on MR images. Quantitative and qualitative MRI findings for the diagnosis of adhesive capsulitis were defined as previously described in the literature [3, 5, 11, 24, 25]. Prior to measurement, a training session was conducted for both readers using images that were different from the analysis data. After the two readers measured the parameters separately, all of the parameters were re-evaluated in order to reach a consensus, and statistical analyses were performed with these data.

### Evaluation of anterior capsule of shoulder

We defined the anterior capsule as being from the 2- to 5-o'clock anterior portion of the glenohumeral joint capsule, located deep to the subscapularis muscle and tendon. Therefore, the anterior capsule also included MGHL and spiral GHL, which demonstrated a relatively low signal intensity compared to the subscapularis muscle. SGHL and the anterior band of IGHL were not included in that evaluation of the anterior capsule. Spiral GHL or fasciculus obliquus has been described as a superficial layer of the anterior shoulder joint capsule [26–30]. Spiral GHL is also detectable as a low-signal-intensity band along its course within the anterior joint capsule (Fig. 1). Under the subscapularis muscle and tendon, the spiral GHL is originated from lesser tubercle of the humerus, then run downward to infraglenoid tubercle with spiral course in the superficial layer of the anterior shoulder joint capsule. In the middle of its course, spiral GHL make a tight connection with the MGHL, and then cross and fuse with the underlying IGHL [26–30]. The anterior capsular thickness and abnormal signal intensity were evaluated on the thickest portions of these structures. This measurement was performed on both the axial fat-suppressed PD and oblique sagittal 3D PD VISTA SPAIR images (Fig. 1c, d).

### Quantitative analysis

In quantitative analysis, the following parameters were measured on four-times magnified MRI image: anterior capsular thickness, humeral and glenoid capsular thickness in the axillary capsule, maximal axillary capsular thickness, CHL thickness, and degree of external rotation. In the axillary recess, the humeral and glenoid capsular thickness were measured on oblique coronal T2-weighted MR images of both the humeral and glenoid regions at the thickest portion. The maximal axillary capsular thickness was then determined to be the larger value of humeral and glenoid capsular thickness (Fig. 2a). The maximal CHL thickness was measured on oblique sagittal T2 images (Fig. 2b). The degree of external rotation on the axial MR image was measured by drawing a line from the center of the humeral head to the longitudinal axis of the scapular body, with a second line from the center of the humeral head to the bicipital groove of the humeral head (Fig. 2c) [6]. All measurements were recorded to two decimal places.

### Qualitative analysis

The following findings were evaluated as either being present or absent: anterior capsular abnormal hyperintensity, humeral and glenoid capsular abnormal hyperintensity in axillary recess, and abnormal hyperintensity and obliteration of the subcoracoid fat triangle. If there was abnormal hyperintensity on either side of the humeral or glenoid capsule of the axillary recess, the axillary capsular abnormal hyperintensity was considered to be present. Abnormal hyperintensity of the joint capsule and subcoracoid fat triangle was evaluated on oblique coronal fat-suppressed T2-weighted MR images (Fig. 3a and b). Obliteration of the subcoracoid fat triangle was defined as the low signal intensity of fat on T1-weighted images with respect to subcutaneous fat on oblique sagittal T1-weighted images. Both partial and complete obliteration were considered to be signs of adhesive capsulitis [11, 12, 25] (Fig. 3c).

## Statistical analysis

The demographic and various MR findings were compared between the adhesive capsulitis group and the control group. Fisher's exact test or Mann–Whitney *U* test was used to compare demographic data and imaging parameters

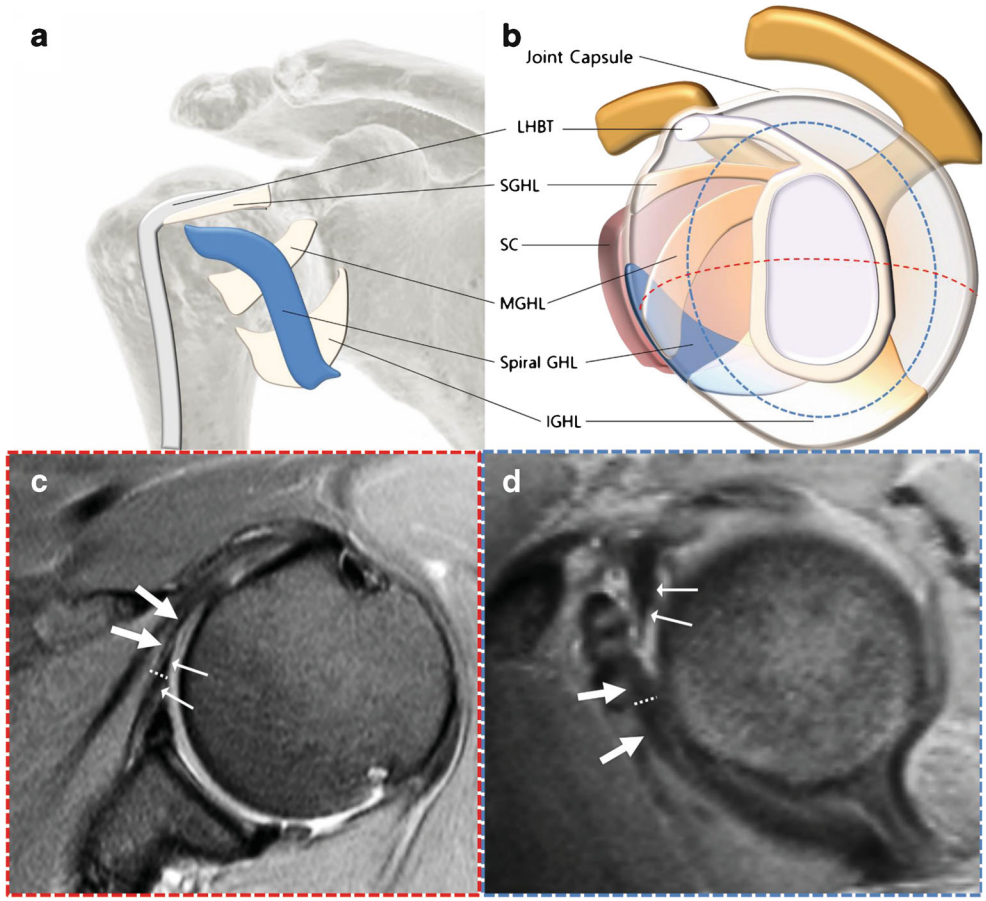


**Fig. 1** Anatomy of the capsular structure of the shoulder joint and examples of measurements of anterior capsular thickness on MRI. **a** Front view of outer side of GHL and **b** lateral view of inner side of joint capsule and GHL. The *red dashed line* indicates the axis of the axial section MR image (**c**) and the *blue dashed line* indicates the axis of the sagittal section MR image (**d**). **c** Axial fat-suppressed PD image showing measurement of anterior capsular thickness (*dashed line*) at the level of the middle glenohumeral ligament (*thin arrows*) crossing the superficial layer of the anterior joint capsule and the spiral glenohumeral ligament (*thick arrows*). **d** Oblique sagittal PD VISTA SPAIR image showing measurement of the thickest portion of the anterior joint capsule (*dashed line*). The middle glenohumeral ligament (*thin arrows*) leads to spiral glenohumeral ligament (*thick arrows*) inferiorly. *LHBT* long head of bicep tendon, *SGHL* superior glenohumeral ligament, *SC* subscapularis muscle, *MGHL* middle glenohumeral ligament, *spiral GHL* spiral glenohumeral ligament, *IGHL* inferior glenohumeral ligament

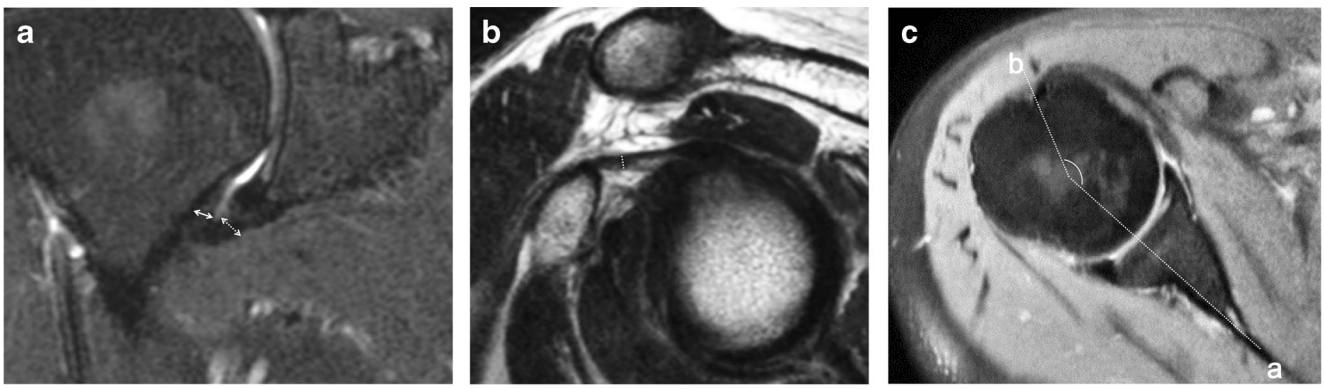


**Fig. 2** Example measurements of quantitative MR findings. **a** A 59-year-old woman with adhesive capsulitis. Oblique coronal fat-suppressed T2-weighted image showing measurement of the thickest portion of axillary joint capsule in both humeral (*arrow*) and glenoid (*dashed arrow*) attachment. **b** A 58-year-old man in the control group. Oblique sagittal T2-weighted image showing measurement of the coracohumeral ligament thickness(*dashed line*). **c** A 58-year-old man in the control group. Axial fat-suppressed PD image at the level of the center of the humeral head showing a degree of shoulder external rotation. An angle was measured between the longitudinal axis of scapula (*a*), and a line was drawn between the center of the humeral head and the bicipital groove (*b*)

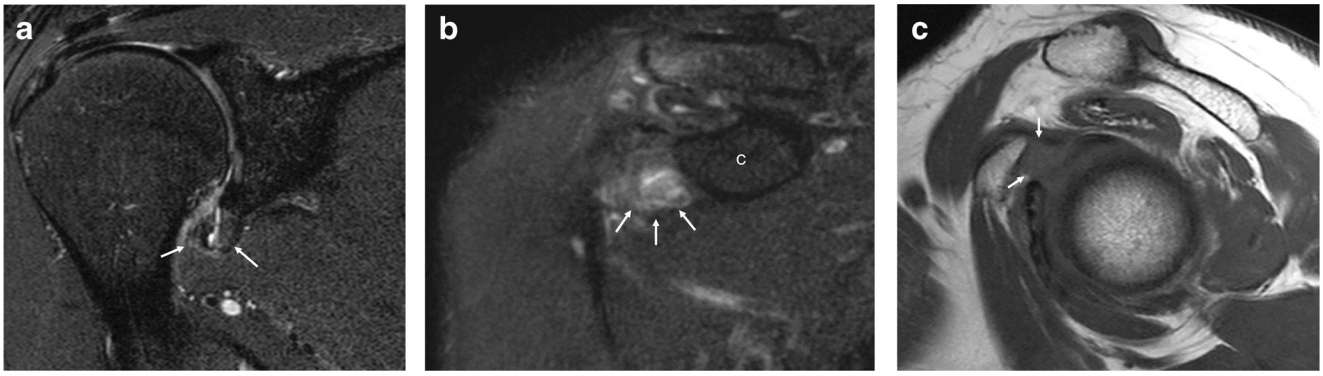


**Fig. 3** Example evaluations of qualitative MR findings. **a** A 47-year-old woman with adhesive capsulitis. Oblique coronal fat-suppressed T2-weighted image showing axillary capsular thickening and abnormal hyperintensity. Increased thickness at the glenoid (4.71 mm) and humeral (5.69 mm) portion and T2 signal intensity of axillary joint capsule (*arrows*) are present. **b** Oblique coronal fat suppressed T2-weighted image at the coracoid process (C) level showing abnormal hyperintensity at the subcoracoid fat triangle (*arrows*). **c** Oblique sagittal T1-weighted image showing the obliteration of the subcoracoid fat triangle (*arrows*)

between the two groups. A correlational study was also performed in order to evaluate the effect of the external rotation degree on MRI findings.

Binary multiple logistic regression analysis was also performed in order to determine the relative contributions of the different MR imaging findings. Characteristics with a $p$ value of less than 0.05 upon univariate analysis were used as independent input variables for multiple logistic regression analysis. With the aim of eliminating multicollinearity, multivariate analysis was conducted separately for quantitative and qualitative findings. In order to evaluate the diagnostic utilities of various parameters, we performed receiver operating characteristic (ROC) analysis so as to determine sensitivities, specificities, and cut-off values.

Inter-class correlation coefficient (ICC) was calculated in order to assess the extent of agreement between the two readers in terms of the measurements of four parameters in quantitative analysis. In order to evaluate inter-observer variability for qualitative analysis, Cohen kappa statistics were also calculated. ICC or kappa values were interpreted as follows: 0, poor agreement; 0.01–0.20, slight agreement; 0.21–0.40, fair agreement; 0.41–0.60, moderate agreement; 0.61–0.80, good agreement; and 0.81–1.00, excellent agreement.

All statistical analyses were performed using one of two computer software programs: SPSS version 20 (IBM Corp, IL, USA) or MedCalc version 16.2.1 (MedCalc Software, Ostend, Belgium).

## Results

### Comparisons of MRI findings by univariate analysis

There were no significant age or sex differences between the adhesive capsulitis group and the control group. The results of comparing the MRI findings between the two groups are summarized in Table 1. All qualitative and quantitative MRI findings were significantly different between the adhesive capsulitis and control groups. The mean anterior capsular thickness in the adhesive capsulitis group was higher than that in the control group ($3.99 \pm 1.64$ mm vs. $1.66 \pm 0.79$ mm, $p < 0.001$). Anterior capsular abnormal hyperintensity was also observed more frequently in the adhesive capsulitis group (25 vs. 4, $p < 0.001$). There was no significant relationship between the external rotation values measured on MRI and other MRI findings, including anterior capsule thickness in the adhesive capsulitis group or the control group ($p = 0.57$).

### Multivariable analysis and diagnostic performance

Among quantitative MR findings, humeral and glenoid capsular thickness were excluded from multivariate analysis because the maximal axillary capsular thickness is clinically used more often; it is also correlated with humeral and glenoid capsular thickness. Multivariate logistic regression analysis showed that anterior capsular thickness and maximal axillary capsular thickness were useful variables that could differentiate adhesive capsulitis from the control group with odds ratios of 7.97 and 17.75, respectively ($p < 0.05$). Multivariate logistic regression analysis with anterior capsular abnormal hyperintensity, axillary capsular abnormal hyperintensity, and abnormal hyperintensity at the subcoracoid fat triangle in qualitative findings showed that only anterior capsular abnormal hyperintensity was significant, with an odds ratio of 12.41 ($p = 0.01$) (Table 1).

Through ROC analysis, the anterior capsular thickness showed higher diagnostic performance than the maximal axillary capsular thickness in diagnosing adhesive capsulitis, with AUCs of 0.897 and 0.868, respectively. However, anterior capsular thickness and maximal axillary capsular thickness were not significantly different upon ROC comparison (95% confidence interval: 0.787–0.970 vs. 0.682–0.914, respectively, $p = 0.28$). The results of cut-off value and area under the ROC curve are shown in Table 2 and Fig. 4, respectively. The cut-off value of anterior capsular thickness at 3.5 mm showed excellent diagnostic accuracy, with a sensitivity of 68.97% and a specificity of

**Table 1** Demographic and MRI findings between the adhesive capsulitis group and control group

| Parameter | Adhesive capsulitis | Control | Univariate | Multivariate | |
|---|---|---|---|---|---|
| | ($n = 29$) | ($n = 20$) | $p$ value | Odd ratio | $p$ value |
| Sex M; F | 12; 17 | 10; 10 | 0.551 | | |
| Age (years) | 51.21 ± 9.18 | 49.15 ± 11.34 | 0.990 | | |
| Quantitative analysis[a] | | | | | |
| Anterior capsular thickness (mm) | 3.99 ± 1.64 | 1.66 ± 0.79 | < 0.001 | 7.97 | 0.02 |
| Maximal axillary capsular thickness (mm) | 4.61 ± 1.53 | 2.55 ± 1.03 | < 0.001 | 17.75 | 0.02 |
| Humeral capsular thickness (mm) | 3.41 ± 1.71 | 2.23 ± 0.69 | 0.015 | | |
| Glenoid capsular thickness (mm) | 4.03 ± 1.74 | 2.47 ± 0.99 | 0.002 | | |
| Coracohumeral ligament thickness (mm) | 3.13 ± 1.16 | 2.25 ± 1.04 | 0.008 | 6.75 | 0.06 |
| Degree of external rotation | 137.41 ± 9.70 | 149.67 ± 10.06 | < 0.001 | | |
| Qualitative analysis[b] | | | | | |
| Anterior capsular abnormal hyperintensity | 25 (86.2%) | 4 (20.0%) | < 0.001 | 12.41 | 0.01 |
| Axillary capsular abnormal hyperintensity | 24 (82.8%) | 2 (10%) | < 0.001 | 3.55 | 0.29 |
| Humeral capsular abnormal hyperintensity | 21 (72.4%) | 4 (21.1%) | 0.001 | | |
| Glenoid capsular abnormal hyperintensity | 19 (65.5%) | 1 (5.3%) | < 0.001 | | |
| Abnormal hyperintensity at subcoracoid fat triangle | 26 (89.7%) | 5 (26.3%) | < 0.001 | 4.33 | 0.26 |
| Obliteration of the subcoracoid fat triangle | 27 (93.1%) | 8 (40.0%) | < 0.001 | | |

*MRI* magnetic resonance imaging

[a] Data are presented as mean ± SD

[b] Data are presented as number

100% (Fig. 5). The cut-off value of maximal axillary capsular thickness at 4 mm also showed excellent diagnostic accuracy, with a sensitivity of 58.62% and a specificity of 100%.

In five patients (17.2%) in the adhesive capsulitis group, the anterior capsular thickness values were more than 3.5 mm. However, these patients did not meet the diagnostic criteria of maximal axillary capsular thickness (> 4 mm) and CHL thickness (> 3 mm). The other qualitative parameters, except for anterior capsular abnormal hyperintensity, were also negative in two (6.9%) out of these five patients (Fig. 6).

## Interobserver agreement

The results of interobserver agreement are summarized in Table 3. Good agreement was found between anterior capsular abnormal hyperintensity ($\kappa = 0.77$), abnormal hyperintensity in humeral and glenoid capsules (both $\kappa = 0.75$), humeral capsule thickness in axillary recess (ICC = 0.69), and anterior capsule thickness (ICC = 0.66). Moderate agreement was found for coracohumeral ligament thickness (ICC = 0.58), glenoid capsule thickness in axillary recess (ICC = 0.47), abnormal

**Table 2** Diagnostic performances of parameters in ROC curve analysis

| Parameter | AUC | Cut-off value (mm) | Sensitivity (%) | Specificity (%) |
|---|---|---|---|---|
| Quantitative analysis | | | | |
| Anterior capsular thickness | 0.897 (0.774–0.966) | > 3.5 | 68.97 (49.2–84.7) | 100 (83.2–100) |
| Maximal axillary capsular thickness | 0.863 (0.733–0.945) | > 4 | 58.62 (38.9–76.5) | 100 (83.2–100) |
| Humeral capsular thickness | 0.710 (0.561–0.832) | > 3.5 | 44.83 (26.4–64.3) | 100 (82.4–100) |
| Glenoid capsular thickness | 0.772 (0.628–0.881) | > 4 | 48.28 (29.4–67.5) | 100 (82.4–100) |
| Coracohumeral ligament thickness | 0.728 (0.580–0.846) | > 3 | 44.83 (26.4–64.3) | 78.95 (54.4–93.9) |
| Qualitative analysis | | | | |
| Anterior capsular abnormal hyperintensity | 0.861 (0.731–0.944) | | 82.76 (64.2–94.2) | 90.00 (68.3–98.8) |
| Axillary capsular abnormal hyperintensity | 0.831 (0.697–0.923) | | 86.21 (68.3–96.1) | 80.00 (56.3–94.3) |
| Humeral capsular abnormal hyperintensity | 0.757 (0.611–0.869) | | 72.41 (52.8–87.3) | 78.95 (54.4–93.9) |
| Glenoid capsular abnormal hyperintensity | 0.801 (0.661–0.902) | | 65.52 (45.7–82.1) | 94.74 (74.0–99.9) |
| Abnormal hyperintensity at subcoracoid fat triangle | 0.834 (0.698–0.926) | | 93.10 (77.2–99.2) | 73.68 (48.8–90.9) |

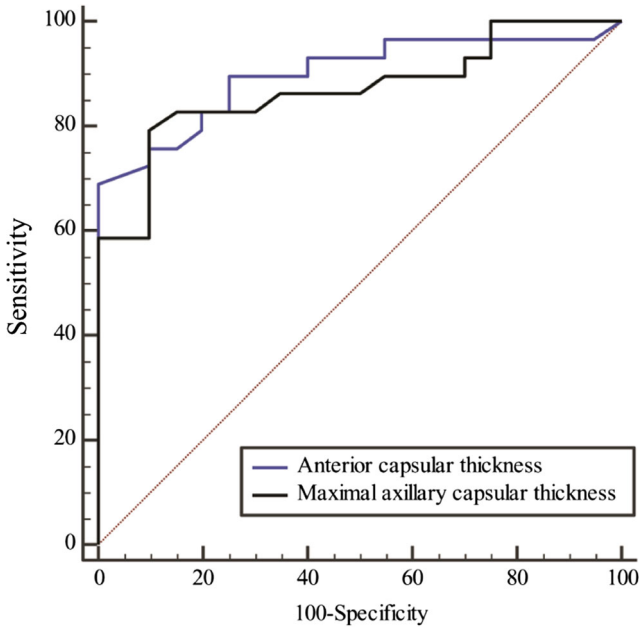


**Fig. 4** Receiver operating characteristic (ROC) analysis of anterior capsular thickness (AUC = 0.897) and maximal axillary capsular thickness (AUC = 0.863)

hyperintensity at the subcoracoid fat triangle (κ = 0.45), and obliteration of the subcoracoid fat triangle (κ = 0.43).

## Discussion

In addition to the already-known MRI findings, our results demonstrated the usefulness of the anterior capsular thickness and abnormal hyperintensity for the diagnosis of adhesive capsulitis (AUC: 0.897 and 0.861, respectively) with good interobserver reliability (ICC = 0.66 and Kappa = 0.77, respectively). The anterior capsular thickness at a cut-off of 3.5 mm afforded high diagnostic performance (sensitivity, 68.97%; specificity, 100%). Moreover, two patients among those with adhesive capsulitis had only anterior capsular thickening and abnormal hyperintensity, without any previously known adhesive capsulitis MRI findings. This means that if we had not evaluated the anterior capsule, these two patients would not have been diagnosed with adhesive capsulitis on MRI.

The main pathophysiology of the painful restriction of movement in adhesive capsulitis is an inflammatory contracture of the shoulder joint capsule [1, 2]. Therefore, abnormalities such as inferior glenohumeral ligament and CHL thickening as well as abnormal hyperintensity have been reported to be the important findings of adhesive capsulitis in imaging studies, including in conventional MRI [27–30]. Classically, the following three anterior capsular ligaments have been frequently described: SGHL, MGHL, and the anterior band of IGHL. In addition to these three well-known capsular ligaments, fasciculus obliquus or spiral GHL has been described as a superficial layer of the anterior shoulder joint capsule [27]. The spiral GHL is also detectable as a low signal intensity band along its course within the anterior joint capsule. In the present study, we could easily recognize the spiral ligament in its course, especially at the point crossing the middle glenohumeral ligament (Fig. 1). These capsular ligaments, along with the inner capsular structure of the subscapularis muscle, form the anterior joint capsule of shoulder [26–30]. Therefore, the anterior capsular thickness was measured at the thickest parts of these structures, excluding the superior glenohumeral ligament and anterior band of the inferior glenohumeral ligament complex.

Pouliart et al. [29] have simply stated that the thickness of the MGHL changes with respect to the degree of external rotation. However, in our study, there was no significant correlation found between the anterior capsule thickness and the external rotation degree measured on MR ($p = 0.57$). A previous study has reported that the length of the anterior capsular ligament is most affected by external rotation at 45° abduction of the shoulder joint [31]. Our patients might have seen less of an impact because the abduction of the shoulder joint was confirmed to be 0 degrees in each patient during MR examination.

We also evaluated abnormal hyperintensity of the anterior capsule, which also showed good diagnostic performance (AUC = 0.861). This might be due to the same pathophysiology, such as synovial proliferation and hypervascular change in the axillary capsule, as described in previous studies [1, 2, 6, 12, 15].

To the best of our knowledge, no previous study has used MRI to evaluate abnormality of the anterior capsule, located deep to the subscapularis muscle, in adhesive capsulitis patients. However, previous surgical and pathologic studies have

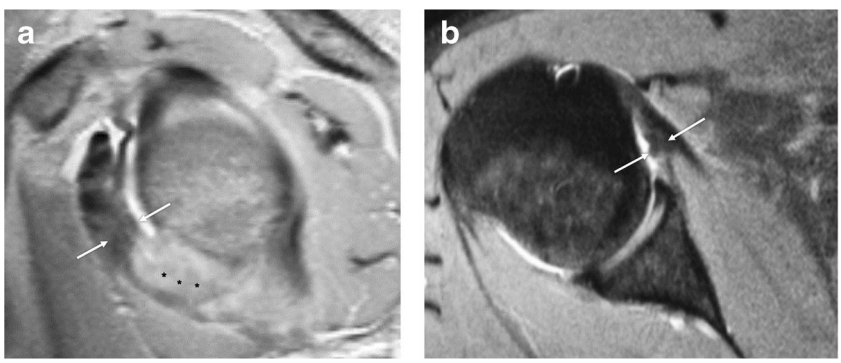


**Fig. 5** A 46-year-old man with adhesive capsulitis. **a** Oblique sagittal PD VISTA SPAIR image showing thickening (4.78 mm) of the anterior joint capsule (*arrows*). Thickening and abnormal hyperintensity of the axillary joint capsule are also noted (*asterisks*). **b** Axial fat-suppressed PD image also showing marked thickening of the anterior joint capsule (*arrows*)

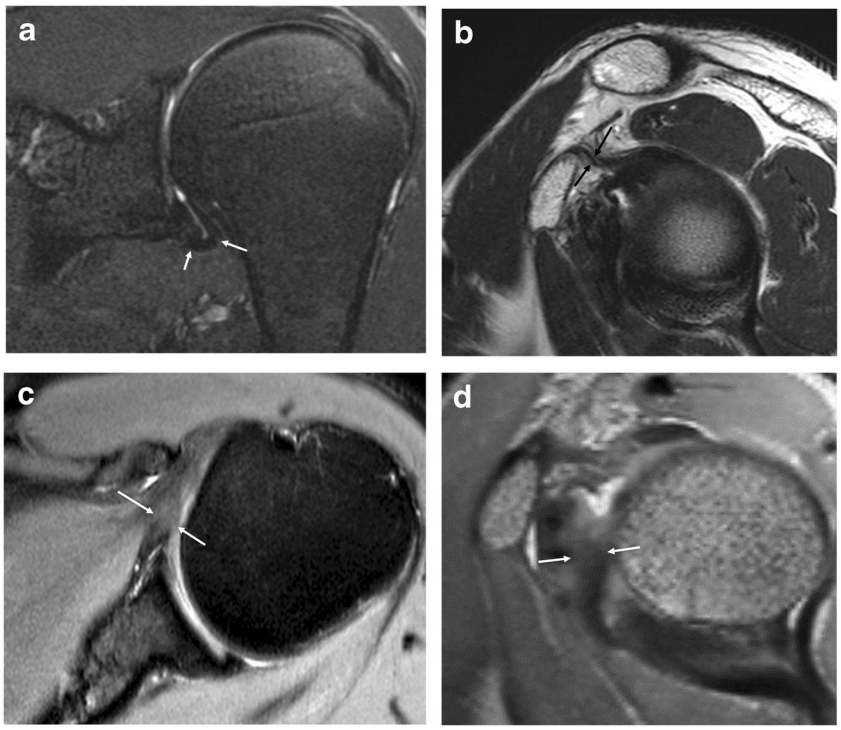


**Fig. 6** A 50-year-old man with adhesive capsulitis. **a** Oblique coronal fat-suppressed T2-weighted image showing no significant thickening (3.70 mm) of the axillary joint capsule (*arrows*). There is no significant axillary capsular abnormal hyperintensity either. **b** Oblique sagittal T2-weighted image showing no significant thickening (2.90 mm) of the coracohumeral ligament (*arrows*). **c** Axial fat-suppressed PD image showing thickening (6.94 mm) and abnormal hyperintensity of the anterior joint capsule (*arrows*). **d** Oblique sagittal PD VISTA SPAIR image showing prominent thickening of the anterior joint capsule (*arrows*)

reported abnormal findings of the anterior capsular structure. In arthroscopic observation studies, adhesive capsulitis and synovitis were mainly located on the anterior side of joint, including rotator interval and MGHL prior to manipulation. Following manipulation, hemorrhages were mainly found in the anterior-inferior capsule and IGHL [17, 18]. In addition, arthroscopic release surgery limited to the anterior capsule in patients unresponsive to conservative measures is usually enough to obtain a full range of motion. During this procedure, if external rotation remains tight following the excision of the anterosuperior ligament complex, the MGHL and anterior capsule are sequentially divided [20]. Uhthoff et al. [15] have conducted a comprehensive study to determine whether fibroplasia affects all structures equally. They found that vimentin, a cyto-contractile protein, was strongly expressed anteriorly. However, it was absent in the posterior capsule [15].

In our study, the thickness of the joint capsule in axillary recess and CHL showed a significant difference between the adhesive capsulitis and control groups, corresponding to the results of previous studies which found similar cut-off values [3, 5, 11, 24, 25]. Axillar capsular abnormal hyperintensity and obliteration of subcoracoid fat triangle were also found to be significant parameters for the diagnosis of adhesive capsulitis, the same as in previous studies [6, 12, 25].

Our study had several limitations. First, our study included a small number of subjects who underwent MRI, compared with the total number of adhesive capsulitis patients in our clinic. This is because MRI was not always needed to diagnosis adhesive

**Table 3** Interobserver agreement

| Parameters | ICC | κ | Agreement |
|---|---|---|---|
| Quantitative analysis | | | |
| Anterior capsular thickness | 0.66 | | Good |
| Humeral capsular thickness in axillary recess | 0.69 | | Good |
| Glenoid capsular thickness in axillary recess | 0.47 | | Moderate |
| Coracohumeral ligament thickness | 0.58 | | Moderate |
| Qualitative analysis | | | |
| Anterior capsular abnormal hyperintensity | | 0.77 | Good |
| Humeral capsular abnormal hyperintensity in axillary recess | | 0.75 | Good |
| Glenoid capsular abnormal hyperintensity in the axillary recess | | 0.75 | Good |
| Abnormal hyperintensity at the subcoracoid fat triangle | | 0.45 | Moderate |
| Obliteration of the subcoracoid fat triangle | | 0.43 | Moderate |

*ICC* Intraclass Correlation Coefficient, *κ* kappa

capsulitis, as it is largely a clinical diagnosis. Patients are also reluctant to undergo MRI because of its high cost. These might have biased our results in our retrospective study design. However, 29 patients was not too small of a number to draw statistical conclusions compared to previous studies. Second, our study was a case-control design, therefore, we did not demonstrate whether the anterior capsular abnormality is a specific finding associated with adhesive capsulitis. Further study with larger numbers of subjects with various diseases are needed in order to evaluate the associations between various MRI findings and adhesive capsulitis. Third, the anterior capsular abnormality was not confirmed through arthroscopy or histologic analysis. Further arthroscopic or pathologic study of patients with anterior capsular abnormalities is also needed in the future. Fourth, we did not perform a subgroup analysis regarding the clinical stage of adhesive capsulitis because the number of patients was too small. Thus, there may have been bias toward subgroup analysis. Fifth, we used the clinical criteria for the diagnosis of adhesive capsulitis and did not confirm the diagnosis through arthroscopy or histology. A definitive diagnosis of adhesive capsulitis can only be achieved through direct surgical observation. However, arthroscopic treatment is not recommended as the primary treatment for adhesive capsulitis, thus making histologic correlation with clinical findings difficult. However, in routine practice, the initial suspicion of adhesive capsulitis is often noted in clinical examination, and MRI and other imaging methods are taken only to exclude other diseases such as cuff tear and to identify suggestive findings of adhesive capsulitis. In order to overcome this limitation and reflect the actual clinical situation, we used the same process in our inclusion process as the one used in clinical practice. First, we used the clinical criteria that have been suggested in previous studies [6, 21, 23], and then used imaging modality, including MRI, in order to exclude other pathology.

In conclusion, anterior capsular abnormality, such as thickness and abnormal hyperintensity, can be used for the diagnosis of adhesive capsulitis of the shoulder. Among various MR findings of adhesive capsulitis, anterior capsular thickness and abnormal hyperintensity, as well as maximal axillary capsular thickness, had strong diagnostic value in adhesive capsulitis. In addition to the previously known abnormal MRI findings at the rotator interval, subcoracoid fat triangle, and axillary recess, the evaluation of the anterior capsule of shoulder joint could be used for diagnosis of adhesive capsulitis.

## Compliance with ethical standards

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