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Can magnetic resonance imaging distinguish clinical stages of frozen shoulder? A state-of-the-art review

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Background: Frozen shoulder (FS) is a common disorder causing shoulder pain and limited motion. Magnetic resonance imaging (MRI) is expected to help diagnose FS and realize the disease stage if stage-specific features are present. We aimed to survey stage-related MRI findings of FS in the literature.
Methods: MEDLINE, SCOPUS, and Google Scholar databases were searched with search terms "frozen shoulder" or "adhesive capsulitis" combined with "magnetic resonance imaging." Studies that discussed MRI findings in relation to FS stages were retrieved. The course of FS was divided into stages 1 to 4 according to Hannafin and Chiaia.
Results: Two of the noncontrast-enhanced MRI findings were stage-related. T2 signal hyperintensity of the joint capsule was more frequent in stages 1 and 2. The axillary capsule thickness was greater in stages 1 and 2. However, these findings were also seen in the later stages to a lesser degree. Effusion around the long head of biceps, subcoracoid fat obliteration, and coracohumeral ligament thickening were common in FS but their relation to the stages was not evident. Signal enhancement on contrast-enhanced MRI was not consistently linked to stages.
Conclusion: T2 signal hyperintensity and axillary capsule thickening are characteristic of the early stages of FS, although MRI alone cannot completely define the disease stage.
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Frozen shoulder (FS), or adhesive capsulitis, is a common musculoskeletal disorder, characterized by spontaneous onset of shoulder pain accompanied by progressive loss of active and passive ranges of motion.[42] The lifetime prevalence of FS is estimated to be 2 to 5 percent of the general population.[19] Severe pain, sleep disturbance, and difficulties in performing activities of daily living decrease the quality of life and increase depression and anxiety in patients with FS.[3]

Although the etiology of FS is still unclear, the primary pathology is thought to be inflammation and subsequent fibrosis of the shoulder joint synovium and capsule.[21,40] Briefly, patients in the freezing stage show inflammation in the synovium of the shoulder joint, while those in the frozen stage show fibrotic processes in the subsynovial layer and the joint capsule.[34] Inflammatory cytokines such as tumor necrosis factor-alpha, interleukin-1 and -6, and immune cells such as mast cells, macrophages, and T and B lymphocytes are noted in the synovium of the FS.[34] There are fibrotic growth factors such as transforming growth factor-beta and type-III collagen in the synovium and the joint capsule of FS.[34] Thus, a complex cascade of growth factors and cytokines leads to the activation of fibroblasts and the deposition of collagen.[21,40]

FS has historically been a clinical diagnosis of exclusion. Conditions that should be ruled out include a history of definite trauma, fractures, rotator cuff tearing, calcific tendinitis, rheumatoid arthritis, osteoarthritis, septic arthritis, labral lesion, neoplastic condition, neurologic deficit, or cervical spine disease. Imaging has been used for the exclusion of these conditions when

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they present with clinical symptoms similar to those of FS. Imaging is also helpful for the identification of FS when the clinical symptoms are atypical. Recent studies have demonstrated several findings of magnetic resonance imaging (MRI) that are characteristic of FS. They included effusion around the long head of biceps (LHB), obliteration of the subcoracoid fat triangle, T2 signal hyperintensity of the joint capsule, thickening of the joint capsule, thickening of the coracohumeral ligament (CHL), and postcontrast enhancement of the rotator interval (RI) and axillary joint capsule.[12,35,39]

In the light of pathologic processes in FS that inflammation precedes fibrosis, manifestations of inflammation such as T2 signal hyperintensity of the joint capsule are expected to be seen in the early stage of the disease, while manifestations of fibrosis such as thickening of the CHL and the joint capsule are expected to be seen in the late stage. If MRI can reveal which stage a patient is in, the diagnostic value of MRI will be even greater than it is now and can be useful in developing an appropriate treatment plan based on the stage. With the aim of identifying the MRI findings specific to stages of FS, we surveyed the literature to see at which stage each MRI finding is observed.

Materials and methods

An online search of the MEDLINE, SCOPUS, and Google Scholar databases was performed between 15 May 2023 and 31 August 2023 to identify relevant original articles presenting the MRI findings of FS. Search terms of “frozen shoulder” or “adhesive capsulitis” were combined with “magnetic resonance imaging”. The search was limited to studies written in English. The references cited by the retrieved articles were inspected to explore additional relevant studies. Studies that investigated MRI findings of FS but did not describe their association with the stage of FS were not included in the review of stage-related changes, while studies on the patients at specific stages of FS were included. The MRI findings were obtained from noncontrast-enhanced (non-CE) or contrast-enhanced (CE) MRI. The CE MRI included direct magnetic resonance arthrography (direct MRA), ie, imaging after intra-articular administration of the contrast material, and indirect MRA, ie, imaging after intravenous administration of the contrast material. We investigated both non-CE and CE MRI findings, although the use of non-CE MRI was recommended to diagnose FS due to non-inferiority to CE MRI.[39]

As for the clinical stages of FS, Reeves described three stages, namely, pain, stiffness, and recovery stages, in patients with a total duration of 30 months.[33] On the other hand, Hannafin and Chiaia claimed four stages with a total duration of 24 months, based on the clinical, arthroscopic, and histologic findings.[18] Briefly, Stage 1 refers to the prefreezing stage of the first three months, presenting with pain at rest and the end of shoulder motion resulting from hypervascular synovitis. Stage 2 refers to the freezing stage from 3 to 9 months after the onset, presenting with persistent pain and progressive loss of motion resulting from hypervascular synovitis with scar formation and capsular fibroplasia. Stage 3 refers to the frozen stage from 9 to 15 months after the onset, presenting with relatively pain-free but significant loss of motion resulting from dense scar formation of the capsule. Stage 4 refers to the thawing stage from 15 to 24 months after the onset, presenting with minimal pain and progressive improvement of the range of motions (Table I). In the MRI reports on FS, the four-stage categorization of Hanaffin and Chiaia has most often appeared, so we employed this classification in the current review.

Review results

Non-CE MRI findings of FS and their association with clinical stages

Effusion around the LHB

It is known that effusion around the LHB is more frequent in patients with FS than in control subjects.[45] Park et al considered effusion around the LHB clinically significant when the depth of fluid surrounding the LHB was greater than 2 mm on axial fat-suppressed T2-weighted magnetic resonance images at the level of the humeral neck.[30] This effusion was observed in 53% of the patients with stage 1, 65% with stage 2, and 57% with stages 3 and 4, with no significant differences between stages.[30] Chellathurai et al, with similar criteria of the depth of effusion >2 mm on axial fat-suppressed proton density images at the level of the humeral neck, found effusion around the LHB in 100% of the patients with stage 1, 96% with stage 2, 81% with stage 3, and 62% with stage 4.[5] Thus, effusion around the LHB is a common finding throughout the four stages of FS.

Obliteration of the subcoracoid fat triangle

The subcoracoid fat triangle is the triangular space bounded by the coracoid process, joint capsule, and the CHL seen as a well-defined region of fat signal intensity in normal shoulders.[12] Complete obliteration of this triangle was first reported on direct MRA to be a poorly sensitive (32%) yet highly specific (100%) finding for FS[27] (Fig. 1, *A* and *B*)

This finding can be observed in non-CE MRI as well. Park et al defined obliteration of the subcoracoid fat triangle as the low signal intensity of fat with respect to subcutaneous fat on oblique sagittal T1-weighted images.[30] They found obliteration of the subcoracoid fat triangle in 74% of the patients with stage 1, 56% with stage 2, and 21% with stages 3 and 4, with a significantly higher incidence in stages 1 and 2. Thus, obliteration of the subcoracoid fat triangle was thought to be related to inflammatory changes seen early in disease progression.[12,30]

On the other hand, Chellathurai et al found obliteration of the subcoracoid fat triangle on sagittal T1-weighted images in 44% of the patients with stage 1, 46% with stage 2, 91% with stage 3, and 85% with stage 4, with a significantly higher incidence in later stages.[5] The authors also found obliteration of fat outside the subcoracoid triangle to be more frequent in stages 3 and 4. They claimed that these findings reflect ongoing fibrosis and scar formation. Thus, the association between subcoracoid fat obliteration and the stage of the disease is controversial, and no conclusions can be drawn as to whether this finding suggests inflammation or fibrosis.

T2 signal hyperintensity of the joint capsule

T2 signal hyperintensity of the inferior glenohumeral ligament, or axillary joint capsule, on fat-suppressed T2-weighted images has been known as a highly sensitive and highly specific finding for the diagnosis of FS.[15] Choi and Kim examined FS patients with stages 1 and 2 using oblique coronal fat-suppressed T2-weighted images.[7] They found hyperintensity of the axillary recess in 83%, hyperintensity of the RI in 76%, and hyperintensity of the anterior capsule in 90% of the patients. This indicates that the hyperintensity of the joint capsule is common in the earlier stages of FS, but this study did not investigate for later stages. Park et al investigated joint capsule edema in the axillary recess on oblique coronal fat-suppressed T2-weighted magnetic resonance images, which is equivalent to T2 signal hyperintensity.[30] The authors found capsule edema of the axillary recess in 97 % of the patients with stage 1, 83 % with stage 2, and 64 % with stages 3 and 4, with a significantly

Table I
Four stages of FS.

Stage	Duration	Symptoms	Pathology
1 (Prefreezing)	0-3 mo	Pain at rest and the end of motion	Hypervascular synovitis
2 (Freezing)	3 to 9 mo	Persistent pain, progressive loss of motion	Hypervascular synovitis, scar formation, capsular fibroplasia
3 (Frozen)	9 to 15 mo	Less pain, significant loss of motion	Dense scar formation of the capsule
4 (Thawing)	15 to 24 mo	Minimal pain, recovery of motion	

(Prepared by the authors based on Hanaffin and Chiaia[18]).
FS, frozen shoulder.

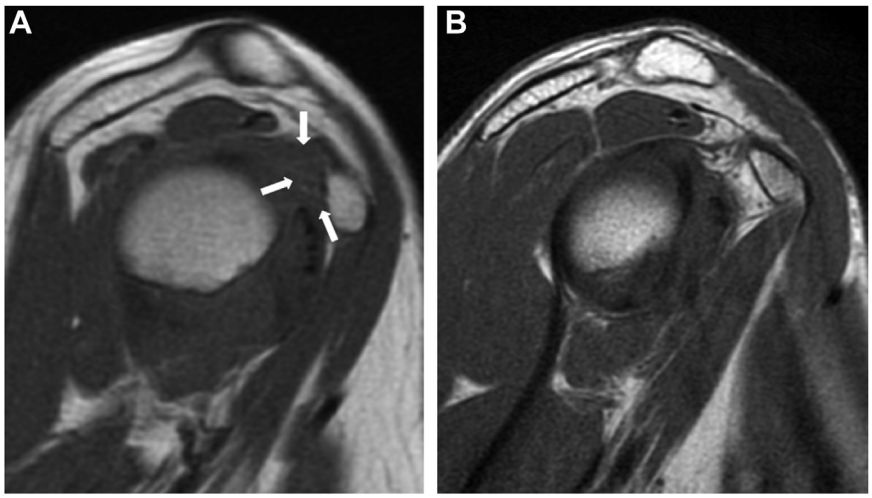

Figure 1 An oblique sagittal T1-weighted image (TR 508msec, TE 8msec) of the left shoulder of a 60-year-old woman with the stage 1 FS (**A**). *Arrows* indicate complete obliteration of the subcoracoid fat triangle. An oblique sagittal T1-weighted image (TR 508msec, TE 8msec) of a 54-year-old man with subacromial impingement is shown for comparison (**B**). *FS*, frozen shoulder.

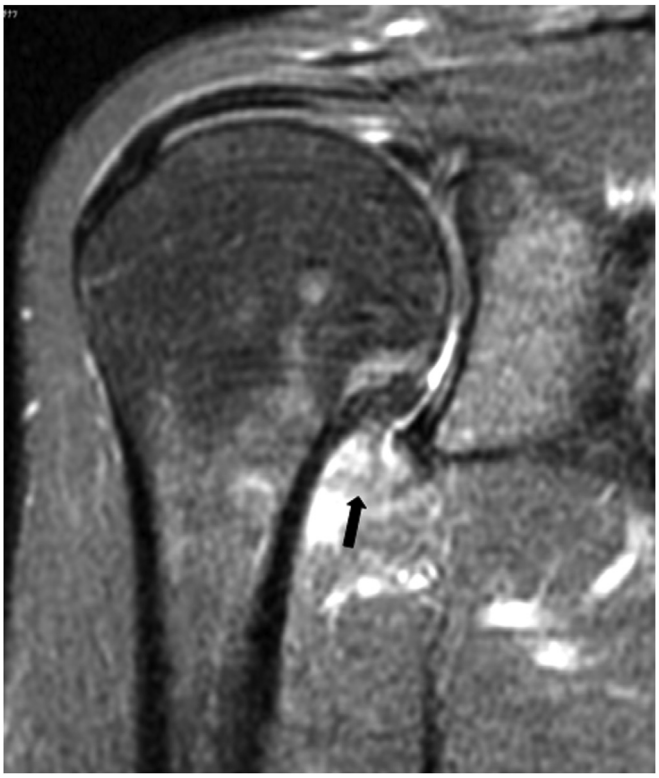

Figure 2 An oblique coronal fat-suppressed T2-weighted image (TR 4900msec, TE 60msec) of the right shoulder of a 55-year-old woman with the stage 2 FS. The *arrow* indicates the hyperintense axillary joint capsule. *FS*, frozen shoulder.

higher incidence in stage 1 than in other stages.[30] Similarly, using oblique coronal fat-suppressed proton density images, Chellathurai et al reported that joint capsule edema of the humeral portion of the axillary recess was observed in 100 % of the patients with stage 1, 89% with stage 2, and 24 % with stages 3, 0 % with stage 4, with a significantly higher incidence in stages 1 and 2[5] (Fig. 2).

Gillet et al investigated the signal intensity of the inferior glenohumeral ligament on oblique coronal fat-suppressed T2-weighted images.[13] The signal intensity was graded into four categories, while the pain duration of the patients was graded into five categories. The authors showed that the patients with higher signal intensity tended to have shorter pain duration, with the pain duration of 3 to 6 months being most frequent among the patients with higher signal intensity.[13] This suggests that patients with stage 2 FS have high signal intensity of the axillary capsule. Signal hyperintensity in stage 2 was also shown in the anterior capsule. Sofka et al, using proton density fast spin-echo images, graded the signal intensity of the anterior capsule as hypointense, hyperintense, or isointense relative to the low signal intensity of the normal capsule.[37] They found hyperintense signal was significantly correlated with stage 2, and speculated that this reflects hypervascular synovitis.[37]

These reports suggest that T2 signal hyperintensity is more frequently observed in stages 1 and 2, but may not be uncommon in stages 3 and 4.

Thickening of the joint capsule

Thickening of the joint capsule has been known as a characteristic finding of FS, most commonly evaluated on oblique coronal T2-weighted images. Differences in thickness of the joint capsule

between FS patients and normal controls have been documented; 5.2 mm vs. 2.9 mm for axillary recess,[10] 7.1 mm vs. 4.5 mm[27] or 7.2 mm vs. 4.4 mm[45] for RI capsule (Fig. 3).

Jung et al investigated joint capsule thickness in FS patients with stage 2 compared to the control group.[22] The mean thickness of the axillary capsule was 5.9 mm vs. 3.6 mm on oblique coronal T2-weighted images, and the mean thickness of the RI capsule was 7.2 mm vs. 4.8 mm on oblique sagittal proton density images. Choi and Kim investigated joint capsule thickness in FS patients with stages 1 and 2 on oblique coronal fat-suppressed T2-weighted images.[7] The mean thickness of the humeral portion of the axillary capsule was 6.0 mm, which is thought to be greater than normal considering the normal value in a cadaveric study (2.8 mm).[8] Thus, the thickness of the axillary or RI capsule is increased in the early stage of FS.

Sofka et al measured combined capsular and synovial thickness at the level of the midaxillary pouch on oblique coronal proton density images.[37] The mean thickness was 4.1 mm, 7.5 mm, 5.5 mm, and 4.1mm in stages 1, 2, 3, and 4, respectively. Thus, the axillary capsule was significantly thicker in stage 2. Chellathurai et al measured the maximal thickness of the humeral and glenoid portions of the axillary capsule on oblique coronal proton density fat-suppressed images.[5] The mean thickness of the humeral portion was 4.1 mm, 6.1 mm, 5.2 mm, and 4.6 mm for stages 1, 2, 3, and 4, respectively. Thus, similarly, the axillary capsule was significantly thicker in stage 2. Park et al reported that the thickness of the humeral portion of the axillary capsule measured on oblique coronal intermediate-weighted images was significantly greater in stage 1 (mean, 4.7 mm) than in the later stages (3.7 mm in stage 2; 3.7 mm in stages 3 and 4).[30] Taken together, it is certain that the thickness of the axillary capsule is greater in stage 1 or 2 than in later stages.

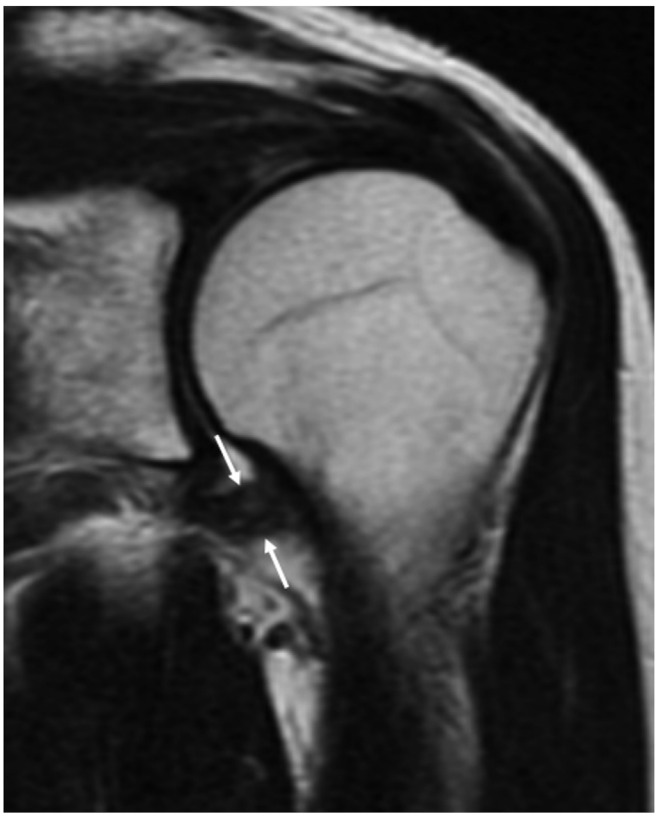

Figure 3 An oblique coronal T2-weighted image (TR 3896msec, TE 90msec) of the left shoulder of a 62-year-old woman with the stage 1 FS. *Arrows* indicate the thickened axillary joint capsule. *FS*, frozen shoulder.

Thickening of the CHL

In its normal state, the CHL is a loose connective tissue that courses from the coracoid base down to the superior aspect of the intertubercular groove, thus lying over the RI.[9] It covers the supraspinatus and infraspinatus posteriorly and envelops the cranial part of the subscapularis inferiorly.[2] In FS, the CHL restricts shoulder abduction, external rotation, and internal rotation[16] (Fig. 4).

Using non-CE MRI, significantly thicker CHL has been noted in patients with FS compared to the control (4.0 mm vs. 3.1 mm,[25] 3.4 mm vs. 2.6 mm,[6] although the difference was denied in another study (1.6 mm vs. 1.6 mm).[10] Difficulties in visualizing the CHL on non-CE MRI may be encountered.[13,25] Choi and Kim reported the thickest part of the CHL to be 3.0 ± 0.86 mm on oblique sagittal T2-weighted images in FS patients with stages 1 and 2, but they did not investigate those with later stages.[7] Chellathurai showed the mean CHL thickness to be 1.6 mm, 2.0 mm, 2.1 mm, and 1.8 mm in FS patients with stages 1, 2, 3, and 4, respectively, indicating no significant variation between the stages.[5] Thus, the difference in CHL thickness among FS stages is indefinite, although non-CE MRI mostly shows an increase in thickness in FS.

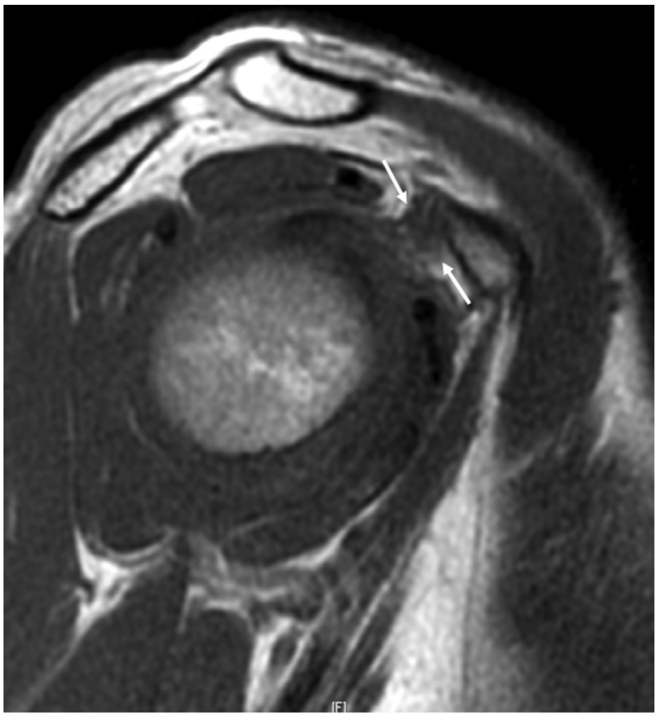

Figure 4 An oblique sagittal T1-weighted image (TR 508msec, TE 8msec) of the left shoulder of a 49-year-old woman with the stage 2 FS. *Arrows* indicate the thickened coracohumeral ligament (CHL). *FS*, frozen shoulder.

CE MRI findings of FS and their association with clinical stages

Postcontrast enhancement of the RI and axillary joint capsule

Generally, the CE MRI provides a higher sensitivity and/or specificity for diagnosing FS.[1,14,32,38] Song et al, in the comparison of FS with the control, showed the difference in thickness of the enhancing portion of the axillary recess and of the RI on indirect MRA was greater than the difference in the thicknesses of the joint capsule on non-CE MRI, thus indicating increased sensitivity of CE MRI.[38] Pessis et al noted a greater RI capsule enhancement on indirect MRA in the earlier stage of FS.[32] The authors also showed that

the sensitivity and specificity of axillary capsule signal enhancement for diagnosing FS were significantly superior to hyperintense signals on T2-weighted fat-suppressed images of non-CE MRI. Erber et al showed that indirect MRA increased sensitivity but not specificity in detecting signs relevant to FS.[11] One report, however, indicated that the indirect MRA did not increase the performance of the inferior glenohumeral ligament signal analysis on T2-weighted fat-suppressed images.[15] Thus, CE MRI is likely to improve sensitivity for estimating joint capsule changes on most occasions, but differences between stages have not been documented.

Some studies evaluated postcontrast enhancement of the joint cavity by dynamic MRI enhanced with intravenous contrast administration. Tamai and Yamato showed a greater enhancement of the axillary pouch in FS patients with stages 1 and 2, although enhancement of less degree remains in those with later stages.[41] Studies using dynamic three-dimensional MRI disclosed an abnormal cluster of blood flow, “burning sign”, in the RI and the axillary pouch in patients with stages 3 as well as in those with stage 2.[20,36] Thus, the enhancement of the joint is seen in both earlier and later stages.

Thickening of the joint capsule

Lee et al showed a greater thickness of the axillary joint capsule (mean, 4.0 mm) in patients with FS compared to the control (mean, 2.3 mm) on oblique coronal T2-weighted images of the direct MRA.[24] Also, in indirect MRA, thickened joint capsule of FS patients has been documented.[38,44] Park GY et al reported the thickness of the axillary recess on oblique coronal T1-weighted images of indirect MRA was 10 mm for stage 2 and 8.8 mm for stage 3, although the difference between stages was not significant.[31] Thus, no reports of CE MRI showed differences in capsular thickness by stage.

Thickening of the CHL

A significantly greater CHL thickness in FS patients compared to that of the control has been reported on oblique sagittal direct MRA images; 4.1 mm vs. 2.7 mm,[27] or 4.1 mm vs. 2.5 mm.[24] Mengiardi et al claimed that a thickness greater than 4 mm was associated with FS.[27] Park GY et al showed the thickness of the CHL on oblique sagittal T1-weighted images of indirect MRA was 4.1 mm for stage 2 and 4.6 mm for stage 3.[31] Because of the paucity of data, it is uncertain at which stage the CHL becomes thickened in patients of FS.

Discussion

To summarize the results of the review, effusion around the LHB is a common finding in FS and is not stage-specific. The subcoracoid fat obliteration is seen in either earlier or later stages of FS, so the association with the stage is controversial. T2 signal hyperintensity of the joint capsule is more frequent in earlier stages, although not uncommon in later stages of FS. The thickness of the axillary capsule is greater in stage 1 or 2 than in later stages. The thickness of the CHL is greater in FS, but the stage-related difference is not definitely shown. The CE MRI increases the sensitivity to detect the capsular changes and denotes enhancement of the joint structures in either early or late stages. Hence, what can be said stage-related is that the T2 signal hyperintensity of the joint capsule is more frequent in stages 1 and 2, and that the axillary capsule is thicker in stages 1 and 2.

The high incidence of T2 signal hyperintensity of the joint capsule in stages 1 and 2 is consistent with the concept of FS that inflammation precedes fibrosis. Previous biopsy studies show that inflammatory cytokines appear in the synovium in the earlier stage of FS.[34] Immune cells such as mast cells, macrophages, and T and B lymphocytes are also observed.[23] In addition, angiogenesis and neurogenesis occur in the subsynovial layer.[43] These synovial and subsynovial events are accompanied by an increase in water, then an increase in the signal on fat-suppressed T2-weighted magnetic resonance images. T2 signal hyperintensity, however, is also seen in stages 3 and 4. This may be due to continued inflammation in the late stages of FS. Chronic inflammatory cell infiltrates,[17] expression of mRNA for inflammatory cytokines,[4] or nonspecific inflammation with synovial hyperplasia[29] have been shown in patients with disease duration over nine months.

Before this study, we thought that the thickening of the axillary capsule was due to fibrosis and is seen in the late stage of FS. Contrary to our expectations, the MRI showed that the axillary capsule was thicker in stages 1 and 2. Early capsule thickening may be, in part, due to inflammation or edema of the capsule, as evidenced by T2 signal hyperintensity of the capsule. Another reason may be the onset of fibrosis in the early stage of FS. Increased fibroblast proliferation,[28] as well as upregulation of genes for collagen, matrix metalloproteinases, and transforming growth factor-beta,[26] were known to occur in Stage 2 FS. However, it is difficult to determine the true cause of the early capsular thickening seen on MRI. The cause for the reduction of the capsular thickness in later stages is also unknown, but may be a sign of quiescence of the disease.

Prior to this study, we also believed that the CHL thickens at a later stage of FS. Contrary to our expectations, we did not find any reports showing a later onset of CHL thickening. It is not clear what mechanism causes the thickening of the CHL that lies extra-articular. Anatomical studies show that the CHL is not a true ligamentous tissue but a reflection of the articular capsule.[9] Under normal conditions, it consists of sparse fibers with high type III collagen content.[2] Accordingly, some stimuli, such as inflammation of the adjacent RI, may easily cause fibrosis and scar tissue formation in the CHL at the early stage of FS.

Because of the paucity of stage-specific MRI findings, MRI alone could not be used to identify the stage of FS. However, the pathologies observed on MRI can provide an appropriate treatment plan. If T2 signal hyperintensity of the joint capsule is noted in a patient with the early stage of FS, intra-articular corticosteroid injection may be indicated. In this setting, the axillary capsule, and probably the CHL, would be already thickened, so vigorous stretching of the capsule may be needed shortly after injection. Yet, whether an MRI-based treatment plan will have better outcomes must be proven in future clinical trials.

There are several limitations to this study. First, it uses only the disease duration as a basis for staging. Almost all retrieved articles determined the stage based on disease duration, not on arthroscopic or pathologic findings. This may be unavoidable since most patients with FS do not undergo arthroscopic or histological examination. In addition, because the course of FS varies greatly from person to person, the disease status can be variable even during the same stage of the disease. Second, inflammatory thickening of the capsule was not distinguished from fibrous thickening on MRI. Third, the thickening of the axillary capsule was not strictly distinguished from the shortening of the capsule.

Despite these limitations, this study revealed that T2 signal hyperintensity of the joint capsule and the thickening of the axillary capsule typify the earlier stages of FS, whereas effusion around the LHB, subcoracoid fat obliteration, and thickened CHL do not differ in frequency by stage and are not helpful in differentiating the stage of the disease.

Conclusion

T2 signal hyperintensity and the axillary capsule thickening are characteristic of the early stages of FS, although MRI alone cannot completely define the disease stage.

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