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Shoulder Impingement Syndrome:
MR Findings in 53 Shoulders

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The shoulder impingement syndrome refers to a condition in which the supraspinatus tendon and subacromial bursa are chronically entrapped between the humeral head inferiorly and either the anterior acromion itself, spurs of the anterior acromion or acromioclavicular joint, or the coracoacromial ligament superiorly. As a result, the space for the bursa and tendon is reduced, and repeated trauma to these structures leads to bursitis and rotator cuff injury. Although pain and limitation of motion are common early findings, the diagnosis is often delayed until a complete tear of the rotator cuff has occurred. In an attempt to determine if MR can be used to depict the abnormalities associated with impingement syndrome (subacromial bursitis, supraspinatus tendinitis, and rotator cuff tear), we reviewed 107 MR scans of painful shoulders. Changes consistent with impingement syndrome were found in 53 patients (50%), 32 of whom underwent subsequent arthrography or surgery. MR was found capable of depicting several soft-tissue and bony abnormalities that have been clinically described in impingement syndrome. In regions of inflammation, we found that the supraspinatus tendon and/or the subacromial bursa were compressed by spurs (25 shoulders), capsular hypertrophy of the acromioclavicular joint (six shoulders), and/or a low-lying acromion (14 shoulders). While T1-weighted MR imaging was highly sensitive to abnormalities of the supraspinatus tendon, tendinitis could be differentiated from a small tear of the supraspinatus tendon only with T2-weighted imaging. Large, full-thickness tears, especially if chronic, produced characteristic MR findings on both T1- and T2-weighted images.

We conclude that MR can be used to detect several abnormalities associated with the shoulder impingement syndrome.

Impingement syndrome is a painful condition that most commonly results from entrapment of the supraspinatus tendon between the humeral head and the anterior part of the acromion, coracoacromial ligament, or acromioclavicular joint [1–3]. The tendon of the long head of the biceps muscle may also be compressed [1, 3–5]. The disorder is due most often to repetitive trauma caused by vigorous overhead occupational or athletic endeavors and/or degenerative exostoses [1, 3, 5, 6]. Uncommonly, impingement results from a tumor or other abnormal mass or from the pressure of the coracoid process on the subscapularis tendon. It affects people of all ages from young adults to the elderly. Neer [5] has maintained that 95% of rotator cuff tears result from impingement. He stresses that anatomic variations in the shape and slope of the acromion can lead to a predisposition to impingement syndrome, as can prominence of the acromioclavicular joint [3, 5]. Figure 1 illustrates impingement of the supraspinatus tendon by hypertrophic spurs at the inferior aspect of the acromioclavicular joint.

MR allows detailed imaging of the shoulder [7]. To determine if MR can be used to detect the lesions associated with impingement syndrome, we devised a method of evaluating the syndrome by using MR imaging.

Fig. 1.—Cadaveric cryomicrosection shows depression of supraspinatus tendon by a spur of acromioclavicular joint. Section is oriented obliquely, along long axis of supraspinatus muscle belly.

Materials and Methods

MR scans of 107 shoulders in 96 patients were reviewed. All patients were referred for MR evaluation of a painful shoulder. Patients who had undergone an invasive procedure localized to the shoulder, such as corticosteroid injection or arthrography, within 1 week before scanning were not included.

Scans were performed with a Fonar 0.3-T permanent-magnet system. T1-weighted images were obtained in all patients by using a spin-echo (SE) sequence (echo time [TE] = 28 msec, repetition time [TR] = 300–500 msec). In addition, T2-weighted spin-echo imaging was performed in 46 patients, with a TE of 56–84 msec and a TR of 1500–2000 msec. The decision to add T2-weighted imaging to the protocol was made after data collection was already under way. Images were either 5-mm thick and obtained at 7-mm intervals or were 3-mm thick and obtained at 5-mm intervals. A 256 × 256 matrix was used for imaging and was interpolated to 512 × 512 for display. Pixel size was 0.75 mm². A planar surface coil was used for all scans.

The technique for positioning the patient and placement of the surface coil has been described [7]. In that previous study evaluating normal anatomy, we determined that positioning the subject supine with the lower arm across the abdomen (i.e., with the elbow flexed and elevated and the humeral head internally rotated) afforded maximal comfort for normal individuals. Fortuitously, this position produces impingement in susceptible persons [3, 5], thus enhancing the detection of disease.

Images were first acquired in the axial plane, followed by oblique imaging along the long axis of the belly of the supraspinatus muscle [7]. Oblique scanning allowed evaluation of the supraspinatus tendon and muscle in continuity, permitting the localization of regions of abnormal signal intensity and encroachment. Because the supraspinatus tendon is the portion of the rotator cuff that is most vulnerable to trauma in the classic impingement syndrome, evaluation of the tendinous cuff was limited to this structure.

The following soft-tissue structures were evaluated in each case: (1) the subacromial bursa was analyzed for signal intensity, thickness, and anatomic course; (2) the supraspinatus tendon was studied for signal intensity and anatomic course; (3) the musculotendinous junc-

tion of the supraspinatus muscle was analyzed for evidence of muscle retraction; and (4) the supraspinatus muscle was analyzed for signal intensity. Other abnormalities indicative of impingement syndrome were also noted, including (1) spurs from the inferior margin of the acromion or acromioclavicular joint, (2) capsular hypertrophy of the acromioclavicular joint, and (3) inferior displacement of the acromion with respect to its articulation with the distal clavicle.

The changes seen on MR were classified into three types, depending on the severity of the abnormalities. Type I, the least severe form of the disorder, implied subacromial bursitis manifested by bursal thickening; the signal intensity of the supraspinatus tendon and muscle remained normal. Type IIa was characterized by abnormally high signal intensity of the supraspinatus tendon, while the supraspinatus muscle remained normal in signal intensity and was without retraction. Type IIb featured a very bright signal within the supraspinatus tendon on T2-weighted images, but the supraspinatus muscle was not retracted; this implied disruption of the cuff, with fluid within the cuff tear. Type III changes included an abnormally high signal intensity from the supraspinatus tendon, as well as retraction of the supraspinatus muscle, indicating complete rupture of the cuff. In cases of type I or II severity, the diagnosis of impingement syndrome was not made unless the subacromial bursa and/or supraspinatus tendon were compressed by overlying structural abnormalities. This was not a requirement of type III disease.

Examples of pertinent normal anatomy are shown in Figure 2. Figures 3–5 represent the different types of impingement syndrome seen on MR.

Results

Using the aforementioned criteria, we identified 53 shoulders in 49 patients whose MR images were consistent with impingement syndrome, with or without complete tear of the rotator cuff. Scans in the remaining 54 patients were either normal or showed other shoulder abnormalities, including isolated labral tear, avascular necrosis of the humeral head, tumor, and infection.

Of the 53 shoulders, six had findings of type I disease (isolated subacromial bursitis) and 29 showed signs of type IIa disease (tendinitis). Lesions implying type IIb or III disease were present in 18 shoulders.

Fourteen of the 35 shoulders with type I and IIa lesions underwent arthrography (eight patients) or arthroscopy (six patients). These examinations yielded small or partial cuff tears in three shoulders with type IIa lesions. The other 11 did not show rotator cuff tears. All 18 complete tears (type IIb or III) were confirmed by either arthrography (12 shoulders) or surgery (six shoulders).

The coracoacromial ligament, a structure that usually is not depicted in MR images of normal shoulders, often was visualized on the oblique images of patients with subacromial bursitis.

Glenohumeral joint effusions, encountered occasionally in patients with type III lesions, were best seen within the dependent (posterior) aspect of the joint on axial images. The fluid manifested low-to-medium signal intensity on T1-weighted images and became bright with T2 weighting. High signal intensity within the supraspinatus tendon on T2-weighted imaging was pathognomonic for a rotator cuff tear (Fig. 6). Fluid was not identified within the subacromial bursa

Fig. 2.—Oblique SE 500/28 MR image of a normal shoulder. Supraspinatus musculotendinous junction lies inferior to acromion (A) or acromioclavicular joint (ACj). Undersurfaces of acromion and distal clavicle (C) approximate same level, and supraspinatus apparatus courses beneath them in a relatively straight line, without focal depression. Tendon (SSt) of supraspinatus muscle (SSm) is homogeneous and devoid of signal. Subacromial bursa (SAb) is a thin band of high signal intensity.
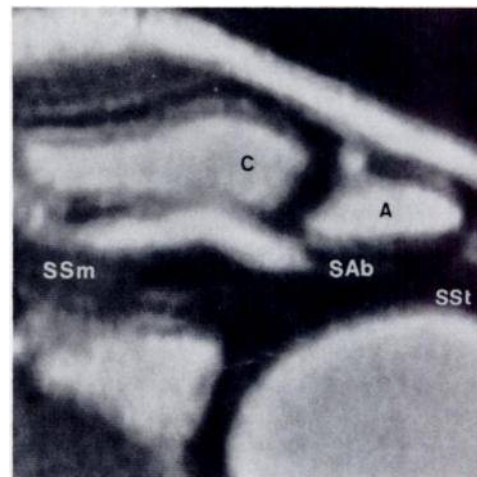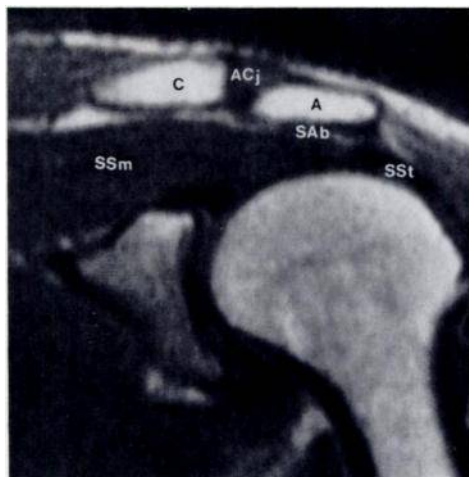
Fig. 3.—MR image (SE 500/28) of type I impingement syndrome (subacromial bursitis). SE 500/28 image. Subacromial bursa (SAb) is thickened and compressed by anterior acromion (A), which lies low with respect to distal clavicle (C). Signal intensity of supraspinatus tendon (SSt) is normal. No spurs were present radiographically or at surgery. SSm = supraspinatus muscle.

Fig. 4.—MR image (SE 500/28) of type IIa impingement syndrome (supraspinatus tendinitis). Medium signal intensity is present within supraspinatus tendon (SSt). Subacromial bursa (SAb) is depressed by hypertrophic acromioclavicular joint (ACj) capsule. Anteroposterior radiograph showed only mild degenerative changes in acromioclavicular joint. A = acromion, C = distal clavicle, SSm = supraspinatus muscle.
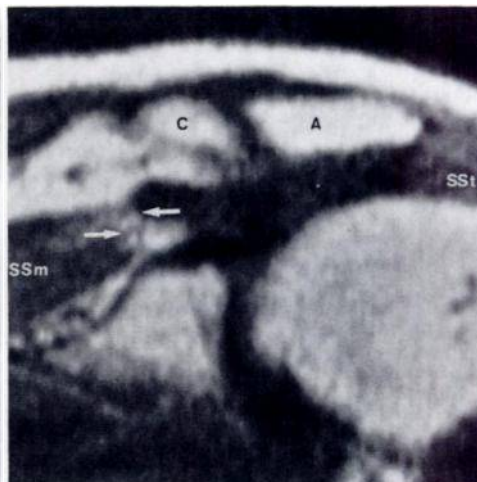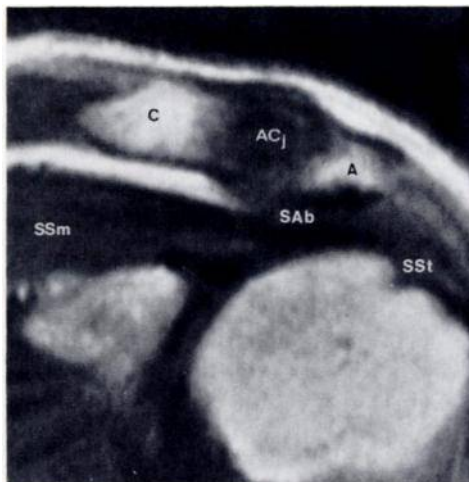
Fig. 5.—MR image (SE 500/28) of type III impingement syndrome (complete rotator cuff). SE 500/28 image. Region of supraspinatus tendon (SSt) is replaced by a region of medium signal intensity. Because musculotendinous junction is retracted (arrows), this represents a complete rotator cuff tear. A = acromion, C = distal clavicle, SSm = supraspinatus muscle.

of any shoulder, a finding consistent with the extensive surgical experience of one of our authors.

Subacromial or acromioclavicular joint spurs were found in 25 shoulders, and the capsule of the acromioclavicular joint was found to be hypertrophic in six shoulders. The anterior acromion was low-lying with respect to the distal clavicle in 14 shoulders. The subacromial space was completely obliterated in the shoulders of eight patients who had massive, long-standing tears. We assumed that any spurs that might have been present in these shoulders had been worn away by the chronic superior migration of the humeral head.

Small subacromial and acromioclavicular spurs were characterized by foci of signal void. Large spurs frequently contained marrow and were therefore imaged as extensions of bright signal from the parent bone, surrounded by a rim of cortical signal void. Tendons compressed by spurs showed abnormally high signal intensity. The exaggerated thickness of the subacromial bursa often decreased abruptly at the level of the spur, hypertrophic acromioclavicular joint capsule, or low-lying acromion, indicating significant compression (Figs. 3 and 4).

Discussion

We found MR to be an informative, noninvasive means of evaluating impingement syndrome. The impinging structures and entrapped tissues often could be imaged precisely, allowing specific details of the site and extent of bone and soft-tissue disease. Because it permitted differentiation of bursitis, tendinitis, and complete supraspinatus tear, MR could be used for classifying the extent of disease and for therapeutic planning.
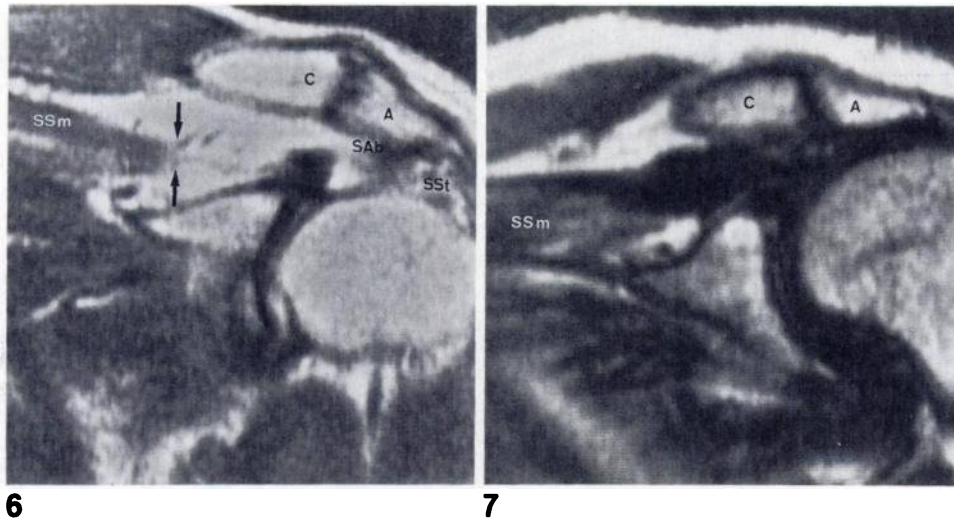
Fig. 6.—MR image (SE 2000/84) of type III impingement syndrome (complete rotator cuff tear). High signal intensity is present within supraspinatus tendon (SSt). Musculotendinous junction is retracted (arrows). A = acromion, C = distal clavicle, SAb = subacromial bursa, SSm = supraspinatus muscle.

Fig. 7.—MR image (SE 500/28) of type III impingement syndrome (complete rotator cuff tear, chronic). Muscle is retracted. Atrophy is characterized by bright bands of signal within muscle and by its diminished bulk. Signal void at expected site of supraspinatus tendon was found at surgery to represent fluid and a severely degenerated tendon. A = acromion, C = distal clavicle, SSm = supraspinatus muscle.

Neer [5] and Neer and Welsh [8] have described three progressive stages of impingement syndrome as found clinically and at surgery. Stage I consists of edema and hemorrhage and is reversible with conservative therapy. Stage II implies fibrosis and thickening of the subacromial soft tissues, and sometimes a partial tear of the rotator cuff, and is manifested clinically by recurrent pain. Although the management of stage II lesions is controversial, failure of conservative treatment is usually an indication for surgery, most often consisting of division of the coracoacromial ligament and acromioplasty, with or without bursectomy. Stage III represents complete rupture of the rotator cuff and is associated with progressive disability.

Although several clinical tests have been devised to detect impingement syndrome [3, 5, 9], the signs and symptoms are somewhat nonspecific, and the diagnosis is often delayed until the development of a full-thickness tear of the rotator cuff [5]. Similarly, radiology plays little or no role in early diagnosis. Either diagnostic plain radiographic changes are absent, or they occur only late in the course of the disease [2, 5, 10, 11]. Those that occur earliest are sclerosis of the greater tuberosity of the humerus and spurring of the inferior surface of the acromion or acromioclavicular joint. As the disease progresses, radiographs may show the typical bony changes associated with a chronic rotator cuff tear, including an acromiohumeral distance of less than 7 mm [12], superior migration of the humeral head, and a concave depression on the inferior surface of the acromion. However, even in stage III disease, radiographs are often normal. Conversely, irregularity or apparent sclerosis of the greater tuberosity may be a normal variant [6, 10], and spurs of the inferior margin of the acromion or acromioclavicular joint do not necessarily imply associated soft-tissue disease [3].

Arthrography aids little in the early diagnosis of impingement syndrome. Even when performed with conventional tomography or CT, the arthrogram is usually normal in stage I disease and may easily be misconstrued as normal in stage II disease, even in the face of an incomplete or small tear of the rotator cuff [13].

Subacromial bursography has been advocated for the early detection of impingement syndrome, and it may reveal mechanical impingement or difficulty in filling the bursa due to soft-tissue edema and thickening [14, 15]. False-positive results may arise from failure to locate and opacify the bursa, and false-negative results may occur when the arm is not put through an appropriate range of motion. In addition, bursography is invasive and requires manipulation of an already painful joint.

No conclusions can be drawn concerning false-negative MR studies in our series. Patients with normal MR scans were not included in the data analysis, and sufficient follow-up was not available in most of these patients.

In our series, MR failed to delineate three partial tears of the rotator cuff. This may have been due to one or all of the following factors: (1) these three scans were performed early in our experience, before T2-weighted imaging was routinely employed; (2) partial-volume averaging may have masked the small tears; and (3) a low-field-strength system was used for this study. However, failure to detect partial tears such as these has little impact on management, since these patients are usually managed initially without surgery. Moreover, during the treatment of advanced disease, small or partial tears can be identified and repaired at the time of acromioplasty or coracoacromial ligament release. Kneeland et al. [16] identified two partial tears in a recent series evaluating the use of MR imaging in the diagnosis of rotator cuff tears.

MR depicted the extent of muscle retraction in patients with complete rupture of the supraspinatus tendon. Muscular atrophy, also readily apparent, was characterized by bands of bright signal within the muscle belly, indicative of fatty replacement (Fig. 7). Both findings are significant in terms of surgical planning and prognosis.

The thick band of bright signal representing the thickened subacromial bursa did not manifest a central region of lower signal intensity on T1-weighted images. In fact, its appearance was homogeneous and similar to that of subcutaneous fat on both T1- and T2-weighted images. Our hypothesis is that the thickening was due to an increase in fibrofatty tissue caused

by hypertrophied synovium, a structure that may contain abundant adipose tissue and/or edematous areolar tissue [17]. Little or no research has been published on the histology of the subacromial bursa in patients with impingement syndrome, but an abnormal abundance of fibrofatty tissue is found in the subacromial space of these patients at surgery. With time, this tissue becomes less evident, much like the degeneration of the supraspinatus tendon seen with chronic rotator cuff tears as the humeral head migrates superiorly. Unfortunately, chemical-shift imaging was not available for confirmation.

MR images of several of our patients who had normal radiographs showed depression of the thickened subacromial bursa and/or the supraspinatus tendon by the anterior part of the acromion, which often appeared quite large. In all of these patients, the inferior margin of the anterior part of the acromion lay inferior to the undersurface of the distal clavicle, a finding that was not evident on routine anteroposterior radiographs. This substantiates Neer's theory that certain individuals have a predisposition to impingement syndrome by virtue of variations in the size or position of the anterior part of the acromion [5].

Although the linear signal void characterizing the coracoacromial ligament is uncommonly identified on MR scans of normal shoulders, we hypothesize that this structure is made apparent in patients with impingement syndrome by virtue of the surrounding proliferative bursitis.

No attempt was made to evaluate the ability of MR to detect tendinous calcifications. Calcific deposits are often small and lie within inflamed tissue, which already has an abnormal and often inhomogeneous signal intensity.

Striking, mottled foci of low signal intensity were often seen within the marrow of the greater tuberosity of the humerus in shoulders without a rotator cuff tear and with normal radiographs. These were generally seen most clearly on axial images and may indicate significant repetitive trauma to the rotator cuff.

Neer's classification [5] of impingement lesions has not only served to define the extent of impingement syndrome, but it has also greatly assisted in planning its therapy. There is no proof, however, that the anatomic abnormalities described by Neer do represent a sequential progression. Further studies with MR may assist in determining if this is the case, or if Neer's stages of impingement simply represent different, possibly associated abnormalities.

Although the clinical staging system for impingement syndrome has frequently proved useful for managing patients with this disorder, in the absence of a demonstrable lesion many orthopedic surgeons persist in conservative management to the detriment of cuff integrity. There is increasing evidence that timely acromioplasty will retard the progress of rotator cuff deterioration [18]. In some of our patients the detailed soft-tissue anatomy depicted by MR permitted early detection of impingement lesions. It is hoped that the earlier diagnosis of impingement syndrome with MR will facilitate more timely and appropriate treatment, long before the disorder has progressed to its ultimate debilitating and irreversible outcome.

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