How to discriminate between acute traumatic and chronic degenerative rotator cuff lesions: an analysis of specific criteria on radiography and magnetic resonance imaging

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\textbf{Background:} Discrimination between acute traumatic and chronic degenerative rotator cuff lesions (RCLs) is an important aid to decision making in therapeutic management. To date, no clinical signs or radiologic findings that enable confident differentiation between these distinct etiologic entities have been identified. The purpose of this investigation was to perform a systematic analysis of known radiographic and magnetic resonance imaging (MRI) features of RCLs and of further, not yet accurately described parameters. The hypothesis was that there are specific radiologic features that allow reliable discrimination between traumatic and nontraumatic RCLs.

\textbf{Methods:} Fifty consecutive patients with RCLs confirmed by MRI were enrolled in this study. Group A was made up of 25 patients with a history of trauma within the previous 6 weeks and no pre-existing shoulder pain, whereas group B comprised 25 patients with shoulder pain for not more than 12 months and no history of relevant trauma. Radiographs and magnetic resonance images were analyzed in a standardized protocol.

\textbf{Results:} No radiographic features were found to differ significantly between the 2 groups. On MRI, edema in the injured muscle was more common in group A (37.5\% vs 4\%, \(P = .04\)). A characteristic feature in traumatic RCLs was a wavelike appearance (kinking) of the central tendon (64\% vs 32\%, \(P = .03\)). In group B, more muscular atrophy was found (29.2\% vs 60\%, \(P = .02\)). Thinning and retraction did not differ between the groups.

\textbf{Conclusion:} MRI, but not radiography, can be used to help discriminate between traumatic and nontraumatic RCLs. Although no absolute distinguishing feature was found, edema, kinking, and muscular atrophy are positive criteria for differentiation.

\textbf{Level of evidence:} Level II, Diagnostic Study.
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\textbf{Keywords:} Rotator cuff tear; trauma; MRI; kinking; atrophy

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Rotator cuff lesions (RCLs) can impair patients’ quality of life and lead to secondary cuff tear arthropathy. In younger patients in particular, RCLs interfere with the function and loading capacity of the shoulder and thus with the ability to work.29,33,35 In several investigations, autopsy, magnetic resonance imaging (MRI), and ultrasound analyses have shown an increasing incidence of RCLs with age.31,46 There is consensus that most RCLs are the result of degenerative causes. Various extrinsic (outlet impingement) and intrinsic (blood supply) factors have been identified as causes.35,50 Especially in elderly patients with chronic and nontraumatic RCLs, initial nonsurgical treatment is recommended.20,21

In contrast, it remains controversial whether any rotator cuff tears are caused exclusively by trauma without any pre-existing degenerative impairment of the tendons. In patients aged younger than 40 years, normally only a high-force injury (e.g., causing a shoulder dislocation) suffices to tear the tendons of the rotator cuff.79 In elderly persons, however, trauma is frequently held responsible for the tendon defect. In those cases, the question of whether the trauma was truly responsible for the tendon rupture or whether the patient already had an asymptomatic RCL regularly arises. In addition, there is the possibility that trauma worsens or reveals a pre-existing asymptomatic RCL (acute-on-chronic tear). Conservative treatment should also be considered in such cases.

Early surgical treatment is recommended in most cases of traumatic RCLs.3,17,38 The outcome of rotator cuff repair is better in traumatic than in degenerative RCLs.2,22 In many cases, a complete traumatic RCL tends to retract rapidly because of the high elasticity of the tendon tissue, which disappears with time because of remodeling and fibrosis.13,17 Therefore, reliable differentiation between acute traumatic RCLs and chronic degenerative RCLs has a high impact on therapeutic decision making. A careful survey of the patient’s medical history is able to distinguish between a solely traumatic and a purely degenerative RCL. However, for the common situation in which an acute-on-chronic RCL mimics a pure traumatic RCL, the patient history alone is insufficient for differentiation. Defining parameters that characterize traumatic and degenerative RCLs could be of assistance.

Surprisingly, there are almost no evidence-based data that facilitate discrimination between traumatic and nontraumatic RCLs. A previous study by our group, focusing on the analysis of clinical diagnostics, showed that physical examination, together with established clinical tests, can indicate the cause and likely time of occurrence of an RCL.26 Certain radiologic parameters may also discriminate between the 2 etiologic entities. The purpose of this study was to analyze radiographs and MRI scans of patients regarding established or new radiologic parameters enabling differentiation between acute traumatic RCLs and chronic degenerative RCLs. The hypothesis was that there are features that allow reliable discrimination.

**Methods**

Between 2011 and 2013, 50 patients with MRI-proven RCLs were enrolled prospectively. All patients underwent surgery because of their symptoms and disabilities. All patients gave their written consent to the analysis and publication of their anonymized data.

Group A comprised 25 consecutive patients who underwent a shoulder injury with no history of problems with the involved joint. Trauma was defined as a sudden, unexpected external event determined by date and place. Only falls from standing height or greater onto the abducted outstretched arm were classified as trauma. Cases of simple contusions, a direct force on the shoulder (e.g., falling on the adducted arm), and distortion during active weight lifting were excluded. Of the 25 patients in group A, 16 had fallen onto the outstretched arm from standing position; 3 reported a fall from greater than standing height; and 6 had been involved in high-velocity bicycle, motorcycle, or skiing accidents. In 2 patients, glenohumeral dislocation resulted.

Group B consisted of 25 consecutive patients with shoulder symptoms for no more than 12 months who were not aware of any trauma on the involved side (Table I). All patients had undergone MRI no more than 6 weeks before enrollment. The exclusion criteria were as follows: age older than 65 years, neurologic disease involving the arm, previous surgery on the affected shoulder, known RCL of the contralateral shoulder, any radiologic signs of shoulder arthritis exceeding grade 1 according to Samilson and Prieto,66 and cuff tear arthropathy of grade I or more according to Hamada et al.15 on either side.

In all patients, standardized radiographs were obtained in the true anteroposterior view in neutral rotation and in the axillary and supraspinatus (SSP) outlet views. The radiographs were analyzed for the following: presentation of degenerative features such as cortical thickening, subcortical sclerosis, and cyst-like lesions at the major tuberosity; acromiohumeral distance; and sclerosis and cortical thickening at the undersurface of the acromion. Furthermore, the form of the acromion was documented using the classification of Bigliani et al.4 and any signs of advanced arthritis of the acromioclavicular joint were recorded.

All patients had undergone MRI of the involved shoulder no more than 6 weeks before enrollment in the study. In most cases, MRI had been performed elsewhere. The minimum requirement for MRI was the availability of images in all 3 planes (coronal oblique, sagittal oblique, and axial) with T1, T2, and fast spin echo fat-saturated sequences. In the trauma group, most patients underwent MRI soon after trauma (median, 7 days after injury; range, 1-36 days).

Analysis of the images included the following: number and topography of injured tendons, degree of retraction according to the classification of Patte,36 configuration of the proximal tendon stump, atrophy or fatty infiltration of the involved muscles,45,51 distinctive effusion in the glenohumeral joint or in the subacromial bursa (or both), and presence of subcortical signal enhancement in the humeral head (bone bruise) (Table II). All radiographs and magnetic resonance images were analyzed by 2 blinded authors with expertise in shoulder surgery (M.L. and F.P.).

Descriptive statistics and invariant data analysis were performed using IBM SPSS Statistics, version 20.0.0 (IBM, Armonk, NY, USA). Categorical variables were analyzed using contingency tables and the chi-square test, and metrical data were analyzed with the i
Results

Radiographs

There were no significant differences in radiographic findings between the 2 groups (Table III). In group B, there were trends toward a higher incidence of degenerative features such as subcortical sclerosis and cyst-like lesions at the greater tuberosity (57.9% in group A vs 78.9% in group B, \( P = .163 \)) and cortical thickening at the undersurface of the acromion (sourcil sign) (47.4% in group A vs 68.4% in group B, \( P = .319 \)) (Fig. 1).

A type III acromion was found in 9.5% of the traumatic cases and 15.8% of the nontraumatic cases (\( P = .451 \)). The acromiohumeral distance differed slightly (8.5 mm in group A vs 7.8 mm in group B, \( P = .211 \)). Acromioclavicular joint arthritis was a frequent finding (52% in group A vs 76% in group B, \( P = .077 \)).

Magnetic resonance imaging

The SSP tendon was torn in 96% of group A patients (24 of 25) and in all patients in group B. A partial- or full-thickness tear of the infraspinatus (ISP) tendon was found in 68% of cases in group A and in 50% of group B (\( P = .392 \)). In contrast, the incidence of subscapularis (SSC) tendon tears was 48% (8% complete and 40% partial) in group A and 12.5% (all partial) in group B (\( P = .021 \)).

In contrast to conventional radiographs, MRI identified some characteristic features with significant differences between the 2 groups (Table IV). Two examiners who were unaware of any specific features assigned the patients to the correct etiologic group by means of MRI with a sensitivity of 72% and specificity of 64%. The positive predictive value of MRI for acute traumatic RCLs was therefore 67%; the negative predictive value, 70%.

Edema (signal enhancement on T2 sequences) in the musculotendinous transition zone of the SSP or ISP (Fig. 2) was found significantly more frequently in group A (37.5% vs 4%, \( P = .004 \)). The specificity of this phenomenon was 96%; the sensitivity, 62.5%.

The incidence of a crinkly, undulating appearance of the peripheral end of the torn muscle, described as “kinking” (Fig. 3), was 64% in group A versus 32% in group B (\( P = .024 \)). The specificity of this feature for traumatic lesions was 68%; the sensitivity, 64%.

Atrophy of the SSP muscle was found significantly more frequently in the nontraumatic group (29.2% in group A vs 60% in group B, \( P = .021 \)). A positive tangent sign was found in 8.7% of patients in group A compared with 32% in group B (\( P = .047 \)). Regarding advanced atrophy (grades II and III), there was a trend toward a higher incidence in the nontraumatic group.

The degree of retraction of the SSP tendon did not differ significantly between traumatic and nontraumatic RCLs (grade I in 37%, grade II in 46%, and grade III in 17% of patients in group A vs grade I in 36%, grade II in 48%, and grade III in 16% of patients in group B; \( P = .989 \)). In both groups, MRI showed a residual tendon stump at the greater tuberosity in about half of the cases (52% in group A vs 56% in group B, \( P = .777 \)). Degenerative features such as cysts or cortical sclerosis were observed in 42% (group A) versus 48% (group B) (\( P = .656 \)). Extensive effusion in the subacromial bursa occurred with similar incidences (84% in group A vs 79% in group B, \( P = .663 \)), whereas glenohumeral joint effusion tended to be more frequent in group A (72%) than in group B (50%) (\( P = .114 \)).

Bone bruising in the humeral head was rare in both groups (8% in group A and 4% in group B, \( P = .552 \)). The 2 patients who sustained glenohumeral dislocations showed no bone bruising.

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**Table I**  Demographic data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A (traumatic)</th>
<th>Group B (degenerative)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, n</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>4</td>
</tr>
<tr>
<td>Age, mean ± SD, y</td>
<td>57.08 ± 7.97</td>
<td>60.4 ± 8.18</td>
</tr>
<tr>
<td>Injury time, mean ± SD, wk</td>
<td>2.14 ± 1.53</td>
<td>42.56 ± 56.31</td>
</tr>
</tbody>
</table>

**Table II**  Radiologic parameters

<table>
<thead>
<tr>
<th>Radiography</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphology of acromion according to Bigliani et al⁴</td>
<td>Intra-articular effusion</td>
</tr>
<tr>
<td>Sclerosis of greater tuberosity</td>
<td>Bursa effusion</td>
</tr>
<tr>
<td>Sourcil (eyebrow) sign</td>
<td>Edema in SSP/ISP</td>
</tr>
<tr>
<td>AC joint arthritis</td>
<td>Bone bruise</td>
</tr>
<tr>
<td>Acromiohumeral distance (in millimeters)</td>
<td>Atrophy of SSP according to Thomazeau et al⁵⁵</td>
</tr>
<tr>
<td></td>
<td>Tangent sign according to Zanetti et al⁵¹</td>
</tr>
<tr>
<td></td>
<td>Retraction of SSP according to Patte⁶⁶</td>
</tr>
<tr>
<td></td>
<td>Degenerative features at greater tuberosity</td>
</tr>
<tr>
<td></td>
<td>Tendon stump at greater tuberosity</td>
</tr>
<tr>
<td></td>
<td>Kinking of tendon</td>
</tr>
</tbody>
</table>

AC, acromioclavicular; ISP, infraspinatus; MRI, magnetic resonance imaging; SSP, supraspinatus.

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\( P < .05 \).
Discussion

The discrimination between traumatic and nontraumatic RCLs represents a complex but nevertheless important medical and socioeconomic challenge. Both for decisions regarding treatment and for compensation by statutory or personal accident insurance providers, reliable differentiation plays a significant role. It is known that most RCLs are the result of degenerative changes. Many cadaveric, MRI, and ultrasound studies have shown the increasing prevalence of RCLs with age. Moreover, the fact that the incidence of RCLs is higher in persons who perform manual labor permits the conclusion that strain and degeneration frequently lead to RCLs.

Traumatic tears are obviously less frequent. In a large series, Bassett and Cofield reported that 8% of RCLs occurred after a fall or another form of trauma. Other articles have estimated the proportion of traumatic RCLs at between 2.3% and 17.7%, but the authors did not analyze these cases in detail. The reason for this wide range is certainly the lack of an explicit definition of traumatic tendon ruptures. Various factors complicate the assessment. About half of the patients with RCLs relate this lesion to a trauma or specific instance of excessive load. Moreover, it is known that about 60% of nontraumatic RCLs remain asymptomatic for years, and many first become symptomatic after an insignificant trauma. On the other hand, Sørensen et al. found that the prevalence of RCLs after trauma was significantly higher than that in a control group without trauma.

In many cases, classification of an individual RCL as traumatic or otherwise depends on the perspective of the observer. Shoulder surgeons tend toward assigning trauma as the cause, whereas in the assessment in which there is a potential relation to an accident, a degenerative nontraumatic reason is preferentially assumed.

There are few evidence-based studies that might help classify an RCL as traumatic or nontraumatic in origin. Therefore, it is recommended to analyze the history, the course of injury, and the primary clinical and imaging findings in every individual case in the attempt to prove causality. Our study aimed to identify features on radiographs or magnetic resonance images that could be characteristic of acute traumatic or chronic degenerative RCLs.

Radiographs

Obviously degenerative changes on radiographs, such as narrowing of the acromiohumeral distance, sclerosis, osteophytes, subchondral cysts, osteolysis, and acromion spurs, have been proposed as criteria for a nontraumatic RCL. This study provides proof that these parameters are not reliable. The 2 groups exhibited no differences in degenerative changes at the major tuberosity. These findings confirm the results of previous studies. Huang et al. analyzed the association between changes at the major tuberosity and RCLs and found a very low positive predictive value (14% to 48%). Even in MRI studies, the presence of

Table III

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A (traumatic) (n = 25)</th>
<th>Group B (degenerative) (n = 25)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type III acromion</td>
<td>9.5%</td>
<td>15.8%</td>
<td>P = .451</td>
</tr>
<tr>
<td>Sclerosis of greater tuberosity</td>
<td>57.9%</td>
<td>78.9%</td>
<td>P = .163</td>
</tr>
<tr>
<td>Sourcil (eyebrow) sign</td>
<td>47.4%</td>
<td>68.4%</td>
<td>P = .319</td>
</tr>
<tr>
<td>AC joint arthritis</td>
<td>52%</td>
<td>76%</td>
<td>P = .077</td>
</tr>
<tr>
<td>Acromiohumeral distance, mean ± SD, mm</td>
<td>8.5 ± 1.4</td>
<td>7.8 ± 2.1</td>
<td>P = .211</td>
</tr>
</tbody>
</table>

AC, acromioclavicular.
humeral cysts did not correlate with RCLs. Subcortical sclerosis of the acromion was not found to be a specific feature in either group. These observations suggest that these radiographic changes represent an unspecific aging process, not allowing any conclusion as to the condition of the rotator cuff.

A decrease in the acromiohumeral distance has been frequently described in RCLs. The size of rotator cuff tendon tears and the degree of fatty degeneration in all rotator cuff muscles have been shown to have a significant negative correlation with the acromiohumeral distance. Our study showed only a tendency toward a decrease in the acromiohumeral distance in nontraumatic lesions. In fact, there was a correlation between the reduction of the subacromial space, the size of the RCL, and the number of ruptured tendons. In group A, there was a higher incidence of 2- and 3-tendon lesions, suggesting that a large traumatic RCL can lead to superior migration within a small number of weeks, thus mimicking a chronic degenerative lesion.

A frequently discussed factor for RCLs is the morphology of the acromion. The first study by Bigliani et al proved a positive correlation between RCLs and acromial spurs. Several subsequent studies have confirmed this observation whereas others have disagreed. In a univariate stratification regarding age, Gill et al calculated that there was no significant correlation between RCLs and acromial morphology. Balke et al confirmed the hypothesis that the morphology of the acromion in patients with degenerative tears of the SSP tendon differed from that in patients with traumatic tears. Shoulders with degenerative tears showed greater lateral extension and steeper angulation of the acromion than those with traumatic tears. In contrast to these observations, our study showed no significant differences in acromial morphology between the 2

### Table IV MRI findings

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A (traumatic) (n = 25)</th>
<th>Group B (degenerative) (n = 25)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intra-articular effusion</td>
<td>72%</td>
<td>50%</td>
<td>( P = .114 )</td>
</tr>
<tr>
<td>Bursa effusion</td>
<td>84%</td>
<td>79%</td>
<td>( P = .663 )</td>
</tr>
<tr>
<td>Edema in SSP/ISP</td>
<td>37.5%</td>
<td>4%</td>
<td>( P = .004^* )</td>
</tr>
<tr>
<td>Bone bruise</td>
<td>8%</td>
<td>4%</td>
<td>( P = .552 )</td>
</tr>
<tr>
<td>Atrophy grade I-III</td>
<td>29.2%</td>
<td>60%</td>
<td>( P = .021^1 )</td>
</tr>
<tr>
<td>Atrophy grade II + III</td>
<td>8%</td>
<td>28%</td>
<td>( P = .078 )</td>
</tr>
<tr>
<td>Tangent sign</td>
<td>8.7%</td>
<td>32%</td>
<td>( P = .047^* )</td>
</tr>
<tr>
<td>Degenerative features at greater tuberosity</td>
<td>42%</td>
<td>48%</td>
<td>( P = .656 )</td>
</tr>
<tr>
<td>Tendon stump at greater tuberosity</td>
<td>52%</td>
<td>56%</td>
<td>( P = .777 )</td>
</tr>
<tr>
<td>Kinking of SSP/ISP</td>
<td>64%</td>
<td>32%</td>
<td>( P = .024^* )</td>
</tr>
</tbody>
</table>

ISP, infraspinatus; MRI, magnetic resonance imaging; SSP, supraspinatus.

* Statistically significant according to \( \chi^2 \) test.

1 Statistically significant according to score test.
groups. In conclusion, the link between a type III acromion and an RCL seems coincidental rather than causal.

**Magnetic resonance imaging**

MRI allows a reliable topographic identification of the injured tendons. Both in traumatic lesions and in nontraumatic lesions, the SSP tendon was involved in almost every patient. In contrast, the SSC tendon was injured significantly more frequently in traumatic lesions than in degenerative lesions. This finding confirms the observations of Gerber et al., Moor et al., and Walch and colleagues, who described a traumatic causation in most of the patients with SSC lesions.

An important finding of our study is that the extent of tendon retraction did not permit differentiation between the 2 etiologic entities. Patte, the first author to describe the degree of tendon retraction, found a correlation with muscle atrophy. Braun et al. found no greater extent of retraction in nontraumatic RCLs than in traumatic RCLs. In a clinical study, Hantes et al. reported that especially traumatic lesions tend toward a rapid retraction of the tendons. Even in experiments on rats, retraction of the muscle-tendon complex was observed to commence a few days after tenotomy.

Tendon retraction is relevant because worse results after surgical rotator cuff reconstruction have been described. Butler et al., however, found no correlation of surgical outcome with the extent of retraction in acute RCLs.

Atrophy and fatty infiltration of the muscles turned out to be a relatively reliable indicator for assessment. In our study, atrophy and especially a positive tangent sign appeared significantly more frequently in the nontraumatic group. Melis et al. described that moderate fatty infiltration of the SSP muscle appeared, on average, 3 years after the onset of symptoms in chronic RCLs and severe fatty infiltration appeared, on average, 5 years after symptom onset. Because atrophy and fatty infiltration are known to be negative prognostic factors for postoperative outcome, Melis et al postulated that rotator cuff repair should be performed before the appearance of fatty infiltration (stage 2) and atrophy, especially when the tear involves multiple tendons.

Some other MRI findings are thought to be trauma-specific features. Hematoma or effusion in the subacromial bursa or inside the glenohumeral joint is believed to indicate an acute tendon rupture. However, hemorrhage and bursal hematoma are difficult to discriminate from an inflammatory effusion. In our study, no differences were found between the 2 groups concerning effusions in the bursa or the joint.

In some publications, bone marrow edema at the greater tuberosity, so-called bone bruising, manifesting as enhancement on fast spin echo fat-saturated sequences, has been described as characteristic for acute RCLs. In our study, however, this phenomenon was observed very rarely, without any difference between the groups. Surprisingly, even the 2 recently dislocated shoulders showed no bone marrow edema on MRI. The small number of studies analyzing the incidence of bone bruising confirms these observations. Mason et al. found a bone bruise in 12 of 712 patients with shoulder problems. Eustace et al. described bone bruising as indicating bone marrow edema of either traumatic hemorrhagic or reactive inflammatory origin.

The most frequent MRI features in the traumatic group were signal enhancement on T2 sequences and the tendon-muscular transition that was interpreted as a traumatic hemorrhage or edema of the injured muscle. This phenomenon was observed in only 1 of the 25 patients in the nontraumatic group. The positive predictive value was 93.7%; the negative predictive value, 72.7%.

In connection with tendon retraction, a phenomenon was observed that we have already described in a clinical study. Kinking of the injured rotator cuff was found significantly more frequently in the patients with assumed acute traumatic RCLs. This term describes a wavelike appearance of the proximal SSP and ISP tendons, also found in some patients with SSC tears. A potential explanation is the well-preserved structural elasticity of the recently torn tendon. In addition, this theory would explain why the degree of retraction was slightly greater in the group with traumatic RCLs than in those with nontraumatic RCLs. Kinking was also found in a third of the cases in group B. Hypothetically, in those cases, a pre-existing degenerative partial lesion had transformed to a complete tear of the tendon (acute-on-chronic rupture). In an as yet unpublished study, we showed a positive correlation between kinking on MRI and structural mobility and elasticity of the torn tendon during surgery. Elasticity (and thus kinking) of the torn tendon is of particular importance for surgical treatment. Davidson and Rivenburgh showed in a clinical study that the outcome of rotator cuff repairs is better in circumstances of low tension load on the reconstructed tendon. Although the validity of the tendon kinking sign on MRI for acute RCLs was not convincing (positive predictive value of 66% and negative predictive value of 65%), the fact remains that in two-thirds of the patients with a positive kinking sign, there was relevant preceding trauma, and in two-thirds of the patients with obviously nontraumatic RCLs, no kinking was found on MRI.

**Limitations**

Our study has several limitations. First, the definition of trauma was challenging. Some mechanisms are accepted as adequate to cause an RCL whereas others are not, but there are no experimental studies proving these hypotheses. Being aware of this, we used strict inclusion and exclusion criteria regarding trauma. A detailed analysis of trauma
mechanisms or the forces acting on the shoulder is not possible here.

An interesting approach to trauma definition was taken by Mackey et al., who proposed a classification for osteoporotic fractures. They distinguished “high trauma,” defined as high-energy trauma such as motor vehicle or bicycle accidents or falls from greater than standing height, from “low trauma,” including all injuries due to falls from standing height or less. When we classified the RCLs of our trauma group in this manner, the injuries in 9 patients were rated as high trauma whereas 16 sustained low trauma. We found no significant differences concerning radiographic findings between the low-trauma and high-trauma subgroups.

These findings are in concordance with a study by Moor et al., who—among other things—calculated the predictive value of trauma for rotator cuff tears using a different but similar grading system. They differentiated traumas due to a relevant documented injury (eg, glenohumeral joint dislocation or fracture at the level of the shoulder girdle) (trauma 1) from symptoms triggered “by an accident (shoulder contusions or strains) without data about severity” (trauma 2). They reported no relevant differences regarding the predictive power for RCLs between their 2 groups.

The sample size of 25 patients per group in our study is quite low. A larger sample would have conferred greater statistical power, but because of the strict inclusion and exclusion criteria, the enrollment—especially of the trauma cases—took almost 2 years.

Furthermore, the duration of symptoms in the non-traumatic group was relatively short. Within 1 year of chronic RCLs, no advanced secondary changes can be expected on either radiography or MRI. However, the aim of this study was to discriminate acute traumatic RCLs from short-term degenerative RCLs.

Finally, no standard protocol for MRI was available. Given the quality standards and the high sensitivity and specificity of MRI, however, no misinterpretations should be expected.

Conclusion

In this study, we systematically analyzed radiologic factors for discrimination between acute traumatic and chronic nontraumatic RCLs. It was shown that radiographs in standard projections are not appropriate for differentiation because assumed degenerative changes occurred in both groups of patients with the same frequency. On MRI, however, specific features can be identified as arguments for or against traumatic causation of RCLs. Indicators of traumatic origin are muscle edema and the kinking sign of the injured proximal tendon. Factors arguing against traumatic causation are advanced atrophy and fatty degeneration of the muscle. Bone bruising and the extent of tendon retraction are not significant for the determination of etiology. The described radiologic features can be used as pointers to the traumatic or nontraumatic etiology of RCLs, especially for the situation in which an acute-on-chronic RCL mimics a true traumatic RCL, and therefore influence the therapeutic management.

Disclaimer

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