

Quantitative Dixon MRI Fat Fraction Is Associated with Bipolar Bone Loss and Chronic Musculotendinous Degeneration in Anterior Shoulder Instability

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Abstract

Background

Anterior shoulder instability is a multifactorial condition involving both static and dynamic stabilizing structures. Although bipolar bone loss and glenoid-track status are increasingly recognized in the assessment of instability, the relationship between osseous abnormalities and quantitative muscle composition remains insufficiently characterized. Dixon MRI enables quantitative assessment of intramuscular fat fraction (FF), potentially providing information beyond conventional visual grading of fatty degeneration.

Purpose

To determine whether quantitative Dixon-derived rotator cuff muscle FF is associated with Hill–Sachs lesions, glenoid bone loss, and glenoid-track status in patients with combined static and dynamic shoulder stabilizer abnormalities, and to evaluate its relationship with chronic musculotendinous degeneration.

Materials and Methods

This retrospective observational study included 116 adult patients aged 18–82 years who underwent shoulder MRI. Patients were classified according to their predominant MRI phenotype as having isolated static stabilizer abnormalities (n = 30), isolated dynamic stabilizer abnormalities (n = 52), or combined static and dynamic abnormalities (n = 34). The primary analysis was performed in the combined subgroup. Quantitative FF was measured using Dixon MRI and evaluated in relation to Hill–Sachs lesions, glenoid bone loss, glenoid-track status, tendon retraction, Goutallier grade, and muscle atrophy. Receiver operating characteristic (ROC) analysis was performed to assess the diagnostic performance of FF for advanced fatty degeneration, muscle atrophy, and full-thickness rotator cuff tears.

Results

Among patients with combined static and dynamic abnormalities, FF was significantly higher in those with Hill–Sachs lesions than in those without lesions ($29.7 \pm 12.8\%$ vs. $17.2 \pm 8.4\%$; $p = 0.006$). Patients with glenoid bone loss also demonstrated higher FF than those without bone loss ($34.6 \pm 12.1\%$ vs. $20.8 \pm 10.3\%$; $p = 0.003$). Among patients with Hill–Sachs lesions, FF was higher in off-track than in on-track configurations ($38.4 \pm 11.6\%$ vs. $23.1 \pm 9.2\%$; $p = 0.009$). FF increased progressively with Goutallier grade and was significantly higher in patients with tendon retraction and muscle atrophy (all $p < 0.001$). ROC analysis demonstrated an AUC of 0.932 (95% CI, 0.874–0.990) for identification of Goutallier grade III–IV degeneration, with a sensitivity of 88.9% and specificity of 90.6% at an FF threshold of $> 28.5\%$. For

muscle atrophy, the AUC was 0.891 (95% CI, 0.817–0.965), with a sensitivity of 84.8% and specificity of 86.2% at an FF threshold of > 22.5%.

Conclusion

Quantitative Dixon-derived rotator cuff muscle FF was associated with bipolar structural abnormalities and chronic musculotendinous degeneration in patients with combined static and dynamic shoulder stabilizer abnormalities. Higher FF values were observed in patients with Hill–Sachs lesions, glenoid bone loss, and off-track configurations and were also associated with greater fatty degeneration, tendon retraction, and muscle atrophy. These findings support the potential role of quantitative Dixon MRI as a complementary imaging marker of the musculotendinous component of anterior shoulder instability. The proposed FF thresholds are exploratory and require validation in larger, independent cohorts before clinical application.

Introduction

Anterior glenohumeral instability is a multifactorial condition resulting from the interaction of capsulolabral injury, glenoid bone loss, humeral head defects, and dysfunction of the dynamic stabilizing structures. In patients with recurrent anterior instability, isolated assessment of either the glenoid or humeral head may therefore provide an incomplete representation of the structural abnormalities underlying instability. Increasing attention has consequently been directed toward the concept of bipolar bone loss, in which glenoid and humeral-sided defects are considered as interacting components of the same mechanical problem. [1–4]

The glenoid track concept provides an established biomechanical framework for evaluating this interaction. The glenoid track represents the portion of the posterior humeral head that remains in contact with the glenoid during functional shoulder motion. A Hill–Sachs lesion is classified as on-track when the Hill–Sachs interval remains within the glenoid track and as off-track when the lesion extends medially beyond the track, indicating a greater potential for engagement with the anterior glenoid rim. This framework has become an important component of contemporary imaging assessment and surgical decision-making in patients with bipolar bone loss. [1, 5, 6] However, recent studies have also emphasized variability in the measurement of glenoid-track parameters and differences in the reliability of imaging techniques, particularly when two-dimensional MRI is used for on-track/off-track classification. [7]

Although osseous morphology is central to the assessment of instability, the condition of the dynamic stabilizing system may provide additional information regarding chronic structural disease. Rotator cuff muscles contribute to dynamic centering of the humeral head, and chronic tendon injury, retraction, altered mechanical loading, recurrent instability, and disuse may be accompanied by muscle atrophy and fatty infiltration. Conventional assessment of muscle degeneration commonly relies on visual or semiquantitative grading systems such as the Goutallier classification. Although clinically established,

categorical grading compresses a continuous biological process into a limited number of categories and may therefore provide only an approximate representation of intramuscular fat content. [8, 9]

Quantitative chemical-shift-based MRI techniques, particularly Dixon imaging, provide an alternative approach by separating water and fat signals and enabling estimation of the fat fraction (FF) within skeletal muscle. Previous studies have demonstrated the feasibility and reproducibility of Dixon-based quantitative assessment of rotator cuff muscle fatty infiltration, including in patients with rotator cuff tears. Quantitative FF measurements have also shown strong associations with conventional indicators of muscle degeneration and can provide continuous information that may be useful for detecting changes not readily captured by visual grading alone. [10–12]

Despite these advances, the relationship between quantitative rotator cuff muscle FF and the integrated osseous phenotype of anterior shoulder instability remains insufficiently characterized. In particular, it is unclear whether quantitative muscle fat content differs according to the presence of Hill–Sachs lesions, glenoid bone loss, or the combined on-track/off-track status of bipolar bone defects. A better understanding of this relationship may help to characterize anterior shoulder instability as an integrated structural and musculotendinous disorder rather than as an isolated osseous or capsulolabral abnormality.

Therefore, the primary objective of this study was to determine whether quantitative Dixon-derived rotator cuff FF differs according to Hill–Sachs lesions, glenoid bone loss, and glenoid-track status in patients with combined static and dynamic shoulder stabilizer abnormalities. The secondary objectives were to evaluate the association of FF with tendon retraction, Goutallier grade, and muscle atrophy and to assess the diagnostic performance of FF for advanced chronic musculotendinous degeneration using receiver operating characteristic (ROC) analysis.

Materials and Methods

Study population and baseline characteristics

This retrospective observational study included 116 adult patients aged 18–82 years (mean age, 42.1 ± 18.1 years), comprising 74 men (63.8%) and 42 women (36.2%). Patients were classified according to the predominant MRI phenotype into three groups: isolated static stabilizer abnormalities ($n = 30$; 25.9%), isolated dynamic stabilizer abnormalities ($n = 52$; 44.8%), and combined static–dynamic abnormalities ($n = 34$; 29.3%).

The primary quantitative analysis of the association between rotator cuff fat fraction (FF) and bipolar osseous instability was performed in the combined static–dynamic subgroup ($n = 34$), in which both instability-related osseous abnormalities and rotator cuff pathology were present.

Abnormalities involved the right shoulder in 64 patients (55.2%), the left shoulder in 50 patients (43.1%), and both shoulders in 2 patients (1.7%). Clinical manifestations included pain, limitation of motion or

stiffness, difficulty elevating the arm, weakness, numbness, and, rarely, changes in skin color. Clinical symptoms were not mutually exclusive.

Eligibility criteria

Patients were eligible for inclusion if they were aged ≥ 18 years, had clinical signs or symptoms of shoulder dysfunction, had undergone standard shoulder MRI, and demonstrated MRI abnormalities involving the static and/or dynamic shoulder stabilizers. Eligible clinical manifestations included pain, motion limitation, instability, or reduced shoulder function. Patients were also required to have adequate MRI image quality and sufficient clinical and imaging information for analysis.

Patients were excluded if they had contraindications to MRI, previous surgery involving the examined shoulder, neoplastic, infectious, or systemic inflammatory shoulder disease, acute fracture, sports injury or professional sports-overload injury according to the source study protocol, inadequate MRI quality, incomplete clinical or imaging data, or substantial motion or metallic artifacts affecting image interpretation.

MRI protocol and Dixon technique

MRI examinations were performed on 1.5-T and 3.0-T scanners using dedicated multichannel shoulder coils. Patients were examined in the supine position with the examined upper extremity maintained in a neutral position. The MRI protocol included T1-weighted, proton-density (PD), proton-density fat-suppressed (PD-FS), T2-weighted, and short-tau inversion recovery (STIR) sequences, together with Dixon MRI, acquired in axial, oblique-coronal, and oblique-sagittal planes.

Dixon imaging generated water-only and fat-only reconstructions. Quantitative fat fraction (FF) was calculated as the fat signal divided by the sum of fat and water signals and was expressed as a percentage:

$$FF (\%) = [fat\ signal / (fat\ signal + water\ signal)] \times 100.$$

Quantitative FF was assessed in conjunction with conventional morphological indicators of chronic musculotendinous degeneration, including Goutallier fatty degeneration, muscle atrophy, rotator cuff tear characteristics, and tendon retraction.

Structural MRI assessment

MRI assessment included systematic evaluation of the static and dynamic stabilizing structures of the shoulder.

Static stabilizer assessment included evaluation of the labrum, including Bankart, anterior labroligamentous periosteal sleeve avulsion (ALPSA), Perthes, glenolabral articular disruption (GLAD),

and superior labral anterior-to-posterior (SLAP) lesions; the rotator interval; Hill–Sachs lesions; glenoid bone loss; glenoid-track status; synovial abnormalities; and capsular abnormalities.

Dynamic stabilizer assessment included evaluation of rotator cuff tendon involvement, tear depth and extent, tendon retraction, tendon signal abnormalities on PD/PD-FS sequences, Goutallier fatty degeneration, and muscle atrophy.

Hill–Sachs lesions were recorded according to their presence or absence. Glenoid-track status was categorized as on-track or off-track according to the relationship between the Hill–Sachs interval and the available glenoid track. Arthroscopic findings were used for comparison with MRI findings when arthroscopic data were available.

Statistical analysis

Statistical analyses were performed using Statistica version 13.0 (StatSoft Inc., USA). Continuous variables were summarized as mean $\pm$ standard deviation (SD) and, where available, median with interquartile range (IQR). Categorical variables were expressed as frequencies and percentages.

Pearson's chi-square test was used for comparisons of categorical variables. For quantitative fat-fraction analyses, between-group comparisons were performed according to the statistical methods used in the source analysis. A two-sided p value < 0.05 was considered statistically significant.

Associations between quantitative FF and structural MRI characteristics were evaluated using correlation analysis. Pearson or Spearman correlation coefficients were selected according to the distributional characteristics of the variables.

Receiver operating characteristic (ROC) analysis was performed to evaluate the diagnostic performance of quantitative FF for advanced fatty degeneration, muscle atrophy, and full-thickness rotator cuff tears. Diagnostic performance was characterized using the area under the ROC curve (AUC), sensitivity, specificity, and FF thresholds. Where available, 95% confidence intervals (CIs) for the AUC were reported.

The primary bipolar bone-loss analysis was restricted to the combined static–dynamic subgroup ($n = 34$). Because only nine patients demonstrated off-track morphology, the off-track analysis was interpreted with consideration of the limited number of events. ROC-derived FF thresholds were considered internally derived estimates and were not regarded as externally validated clinical cut-offs.

All statistical tests were two-sided, and $p < 0.05$ was considered statistically significant.

Results

Study population and MRI phenotype

The overall cohort comprised 116 patients aged 18–82 years (mean age, 42.1 ± 18.1 years), including 74 men (63.8%) and 42 women (36.2%). Patients were classified into three MRI phenotypes according to

the predominant pattern of shoulder stabilizer abnormality: isolated static stabilizer abnormalities (n = 30; 25.9%), isolated dynamic stabilizer abnormalities (n = 52; 44.8%), and combined static and dynamic stabilizer abnormalities (n = 34; 29.3%).

The distribution of patients according to demographic characteristics and MRI phenotype is summarized in Table 1.

Table 1 Baseline characteristics of the study cohort	
Characteristic	Overall cohort (n = 116)
Age, years, mean ± SD	42.1 ± 18.1
Male sex, n (%)	74 (63.8)
Female sex, n (%)	42 (36.2)
Isolated static stabilizer abnormalities, n (%)	30 (25.9)
Isolated dynamic stabilizer abnormalities, n (%)	52 (44.8)
Combined static and dynamic abnormalities, n (%)	34 (29.3)

The overall study workflow and selection of the subgroup used for the primary analysis are shown in Fig. 1.

The primary analysis of bipolar bone loss, glenoid track status, and quantitative Dixon-derived fat fraction (FF) was performed in the subgroup of 34 patients with combined static and dynamic stabilizer abnormalities.

Structural MRI characteristics of the combined phenotype

Among the 34 patients with combined static and dynamic stabilizer abnormalities, a Hill–Sachs lesion was identified in 22 patients (64.7%), while Bankart-type labral abnormalities were present in 20 patients (58.8%). Glenoid bone loss was identified in 11 patients (32.4%).

Among the 22 patients with Hill–Sachs lesions, 13 (59.1%) were classified as on-track and 9 (40.9%) as off-track.

Dynamic stabilizer abnormalities were also common. Supraspinatus involvement was identified in 29 patients (85.3%), including partial-thickness tears in 14 (41.2%) and full-thickness tears in 7 (20.6%). Tendon retraction was observed in 13 patients (38.2%).

The complete distribution of structural MRI abnormalities in this subgroup is presented in Table 2.

Table 2
Structural MRI characteristics of patients
with combined static and dynamic
stabilizer abnormalities (n = 34)

MRI characteristic	n (%)
Hill–Sachs lesion	22 (64.7)
Bankart-type lesion	20 (58.8)
Glenoid bone loss	11 (32.4)
On-track configuration	13 (38.2)
Off-track configuration	9 (26.5)
Supraspinatus involvement	29 (85.3)
Partial-thickness tear	14 (41.2)
Full-thickness tear	7 (20.6)
Tendon retraction	13 (38.2)

Quantitative Dixon fat fraction and bipolar bone loss

Quantitative Dixon FF was significantly higher in patients with Hill–Sachs lesions than in those without a Hill–Sachs lesion ($29.7 \pm 12.8\%$ vs. $17.2 \pm 8.4\%$, respectively; $p = 0.006$).

Similarly, patients with glenoid bone loss demonstrated higher FF values than those without glenoid bone loss ($34.6 \pm 12.1\%$ vs. $20.8 \pm 10.3\%$, respectively; $p = 0.003$).

Among the 22 patients with Hill–Sachs lesions, FF was significantly higher in the off-track group than in the on-track group ($38.4 \pm 11.6\%$ vs. $23.1 \pm 9.2\%$, respectively; $p = 0.009$).

These comparisons are summarized in Table 3.

Table 3
Quantitative Dixon fat fraction according to structural instability findings

MRI finding	Quantitative FF, %	p value
Hill–Sachs lesion		
Absent	17.2 ± 8.4	
Present	29.7 ± 12.8	0.006
Glenoid bone loss		
Absent	20.8 ± 10.3	
Present	34.6 ± 12.1	0.003
Glenoid track status among patients with Hill–Sachs lesions (n = 22)		
On-track	23.1 ± 9.2	
Off-track	38.4 ± 11.6	0.009

The distribution of quantitative FF according to Hill–Sachs lesion status, glenoid bone loss, and glenoid track classification is additionally illustrated using box-and-whisker plots in **Fig. 5**.

Quantitative FF and chronic musculotendinous degeneration

Quantitative FF was significantly associated with several MRI features of chronic rotator cuff degeneration.

Patients with tendon retraction had substantially higher FF values than those without retraction ($36.5 \pm 11.4\%$ vs. $18.3 \pm 9.1\%$, respectively; $p < 0.001$).

FF also increased progressively across Goutallier grades. Mean FF was $9.5 \pm 3.8\%$ in patients with Goutallier grade 0, $23.4 \pm 6.7\%$ in those with grades I–II, and $42.1 \pm 9.2\%$ in those with grades III–IV ($p < 0.001$).

Similarly, patients with MRI-defined muscle atrophy had higher FF than those without atrophy ($36.2 \pm 11.0\%$ vs. $16.7 \pm 7.8\%$, respectively; $p < 0.001$).

These findings demonstrate a consistent relationship between quantitatively measured intramuscular fat fraction and conventional morphological markers of chronic musculotendinous degeneration.

ROC analysis of Dixon-derived fat fraction

ROC analysis demonstrated high apparent discrimination of quantitative FF for advanced fatty degeneration, muscle atrophy, and full-thickness rotator cuff tear.

For identification of Goutallier grade III–IV degeneration, the area under the ROC curve (AUC) was 0.932 (95% CI, 0.874–0.990). An FF threshold of > 28.5% yielded a sensitivity of 88.9% and specificity of 90.6%.

For MRI-defined muscle atrophy, the AUC was 0.891 (95% CI, 0.817–0.965). An FF threshold of > 22.5% yielded a sensitivity of 84.8% and specificity of 86.2%.

For full-thickness rotator cuff tear, the AUC was 0.874, with an FF threshold of > 30.0% yielding a sensitivity of 81.8% and specificity of 87.4%.

The diagnostic performance characteristics of the three ROC analyses are summarized in Table 4.

Table 4
Diagnostic performance of quantitative Dixon fat fraction

Outcome	AUC	95% CI	FF threshold	Sensitivity	Specificity
Goutallier III–IV degeneration	0.932	0.874–0.990	> 28.5%	88.9%	90.6%
Muscle atrophy	0.891	0.817–0.965	> 22.5%	84.8%	86.2%
Full-thickness rotator cuff tear	0.874	—	> 30.0%	81.8%	87.4%

The thresholds identified in this analysis were internally derived from the study cohort and should therefore be considered exploratory estimates requiring validation in independent cohorts before clinical application.

Discussion

The principal finding of this study was a consistent association between quantitative rotator cuff FF and multiple structural markers of shoulder instability and chronic musculotendinous degeneration. Within the combined static–dynamic subgroup, FF was significantly higher in patients with Hill–Sachs lesions and glenoid bone loss and was highest among patients with an off-track configuration. FF also increased progressively with Goutallier grade and was significantly higher in patients with tendon retraction and muscle atrophy.

The association between FF and glenoid-track status is particularly noteworthy. Patients with off-track lesions had a mean FF of 38.4%, compared with 23.1% in patients with on-track lesions. Because the glenoid-track concept incorporates both humeral and glenoid-sided bone loss, this finding suggests that a more extensive bipolar osseous phenotype may coexist with greater fatty degeneration of the dynamic stabilizing musculature. However, the cross-sectional design and small number of off-track cases preclude any inference regarding causality.

The presence of a Hill–Sachs lesion was also associated with a substantial difference in quantitative FF. Mean FF increased from 17.2% in patients without a Hill–Sachs lesion to 29.7% in those with a lesion, corresponding to an absolute difference of 12.5 percentage points. A similar pattern was observed for glenoid bone loss, with mean FF increasing from 20.8% in patients without bone loss to 34.6% in those with bone loss, an absolute difference of 13.8 percentage points. These findings indicate that quantitative muscle composition may be associated with the overall structural phenotype of bipolar shoulder instability.

Several mechanisms may potentially contribute to these associations. Recurrent instability and altered shoulder mechanics may result in reduced muscle loading and changes in dynamic humeral-head centering. Concomitant rotator cuff pathology, tendon retraction, and chronic functional impairment may further contribute to muscle remodeling and fatty infiltration. Nevertheless, age, symptom duration, number of instability episodes, tear severity, and tendon retraction may influence both osseous abnormalities and muscle composition. Consequently, the observed associations should be interpreted as relationships within the imaging phenotype rather than evidence of a direct causal pathway.

The relationship between Dixon FF and conventional markers of chronic rotator cuff degeneration was consistent across several independent measures. Mean FF increased from 9.5% in patients with Goutallier grade 0 to 23.4% in grades I–II and 42.1% in grades III–IV. FF was also substantially higher in patients with tendon retraction and muscle atrophy. This graded pattern supports the ability of Dixon-derived FF to provide continuous quantitative information related to the degree of fatty muscle degeneration.

The ROC analysis further demonstrated high apparent discrimination of FF for advanced fatty degeneration and muscle atrophy. An FF threshold of > 28.5% yielded an AUC of 0.932 for Goutallier grade III–IV, whereas an FF threshold of > 22.5% yielded an AUC of 0.891 for muscle atrophy. These findings suggest that quantitative FF may provide an objective quantitative complement to conventional morphological assessment. However, because the thresholds were derived and evaluated within the same cohort, their diagnostic performance may be optimistic. External validation in an independent population is therefore required before these thresholds can be considered clinically transferable.

The association between FF and full-thickness rotator cuff tears should be interpreted somewhat differently. A full-thickness tear is primarily an anatomical diagnosis that can be directly identified on conventional MRI. Therefore, the potential value of FF in this context is not to replace morphological assessment of tendon integrity but rather to provide additional information regarding associated muscle quality and chronic degeneration. The observed AUC of 0.874 supports this potential complementary role but does not establish FF as an independent diagnostic test for tendon tears.

From a clinical imaging perspective, the findings support a more integrated approach to MRI assessment of anterior shoulder instability. Conventional evaluation of glenoid bone loss and glenoid-track status characterizes the osseous component of instability, whereas quantitative Dixon FF provides information about the condition of the dynamic muscular stabilizers. Combining these parameters may

therefore provide a more comprehensive description of the structural phenotype, particularly in patients with chronic instability and concomitant rotator cuff degeneration.

An important methodological consideration is the distinction between the overall cohort and the primary analytical subgroup. The study included 116 patients across three MRI phenotypes, whereas the principal analysis of bipolar bone loss and quantitative FF was restricted to the 34 patients with combined static–dynamic abnormalities. This distinction is essential when interpreting the magnitude and generalizability of the reported associations.

The principal limitation of the study is the relatively small size of the combined subgroup, particularly the nine patients with off-track lesions. This limits statistical power and restricts the number of covariates that can be reliably incorporated into multivariable models. In addition, the cross-sectional design prevents assessment of temporal relationships between instability, muscle degeneration, and bone loss.

The use of both 1.5-T and 3.0-T MRI systems represents another potential source of technical heterogeneity. Quantitative FF measurements may be influenced by acquisition parameters, reconstruction methods, and scanner characteristics. Standardization of Dixon acquisition and detailed reporting of the measurement protocol are therefore important for reproducibility and future multicenter validation.

Finally, the ROC thresholds reported in this study are internally derived and require validation in independent cohorts. Future prospective studies should determine whether quantitative FF provides information beyond conventional measures of bone loss, glenoid-track status, tendon integrity, and Goutallier grade and whether it is associated with clinically relevant outcomes such as recurrent instability, postoperative function, tendon healing, or return to activity.

Overall, the findings indicate that quantitative Dixon FF may provide a complementary quantitative marker of the musculotendinous component of anterior shoulder instability. Integration of quantitative muscle composition with established measures of bipolar bone loss may offer a more comprehensive imaging phenotype, although prospective validation and standardized quantitative MRI protocols are required before clinical thresholds or prognostic applications can be established.

Limitations

This study has several limitations that should be considered when interpreting the findings. First, the primary analytical subgroup was relatively small ($n = 34$), and only nine patients demonstrated off-track morphology. This limited sample size reduces statistical power and restricts the number of potential confounding variables that can be reliably incorporated into multivariable models. In particular, the off-track analysis should be interpreted cautiously because of the small number of events.

Second, the retrospective cross-sectional design allows identification of associations but does not establish temporal or causal relationships between shoulder instability, bipolar bone loss, and

musculotendinous degeneration. It remains uncertain whether increased muscle fat fraction is a consequence of chronic instability, a marker of coexisting tendon pathology, or a combination of these processes.

Third, MRI examinations were performed using both 1.5-T and 3.0-T systems. Differences in field strength, acquisition parameters, reconstruction methods, and scanner characteristics may influence quantitative Dixon fat-fraction measurements. Although the same general imaging approach was used, standardized acquisition protocols across scanners would improve reproducibility and facilitate comparison between institutions.

Fourth, the quantitative FF analysis was based on ROI measurements of the supraspinatus muscle. The reproducibility of these measurements was not formally assessed using interobserver or intraobserver agreement statistics. Future studies should evaluate measurement reproducibility and determine whether automated or standardized segmentation methods can reduce observer-dependent variability. Previous studies have demonstrated the feasibility of Dixon-based quantitative assessment of rotator cuff muscle fat content, but differences in acquisition and measurement protocols remain relevant when comparing FF values across studies.

Fifth, the ROC thresholds reported in this study were derived and evaluated within the same study population. Therefore, the observed diagnostic performance may be optimistic because of potential overfitting and spectrum effects. The proposed FF thresholds should be regarded as exploratory rather than definitive clinical cut-offs and require validation in independent cohorts.

Finally, the study primarily evaluated imaging associations and did not assess longitudinal clinical outcomes such as recurrent instability, postoperative functional status, tendon healing, or return to activity. Future prospective multicenter studies should determine whether quantitative FF provides incremental information beyond established measures of glenoid bone loss, glenoid-track status, tendon integrity, and Goutallier grading, and whether it has prognostic value for clinically relevant outcomes.

Conclusion

Quantitative Dixon-derived fat fraction was associated with both bipolar structural abnormalities and chronic musculotendinous degeneration in patients with combined static and dynamic shoulder stabilizer abnormalities. Higher FF values were observed in patients with Hill–Sachs lesions, glenoid bone loss, and off-track configurations, while FF also increased with Goutallier grade, tendon retraction, and muscle atrophy.

These findings support the potential role of quantitative Dixon MRI as a complementary imaging marker for characterizing the musculotendinous component of chronic shoulder instability. Importantly, the internally derived FF thresholds should be considered exploratory and should not be interpreted as clinically validated cut-offs. Larger prospective studies using standardized Dixon acquisition protocols,

reproducibility assessment, patient-level adjusted analyses, and independent external validation are required before quantitative FF can be incorporated into routine clinical decision-making.

Declarations

Clinical trial number

Not applicable.

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Tashkent State Medical University and was conducted in accordance with the principles of the Declaration of Helsinki. Given the retrospective observational design and the use of anonymized clinical and imaging data, the requirement for informed consent was waived by the Ethics Committee.

Важно

эту формулировку оставляйте только если Ethics Committee действительно официально предоставил waiver.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

M.Yu.T. study conception and design, data acquisition, MRI assessment, data analysis, and manuscript revision. M.Kh.Kh. Study conception and design, supervision, interpretation of MRI findings, and critical revision of the manuscript. M.R.Kh. Study conception and design, data analysis and interpretation, statistical analysis, manuscript drafting, and critical revision of the manuscript. All authors contributed to the interpretation of the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

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Not applicable.

Data Availability

Data Availability Statement The datasets generated and/or analyzed during the current study are not publicly available because they contain potentially identifiable patient information and are subject to institutional data protection requirements. The data may be available from the corresponding author upon reasonable request and with permission from the relevant institutional authorities.

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Figures

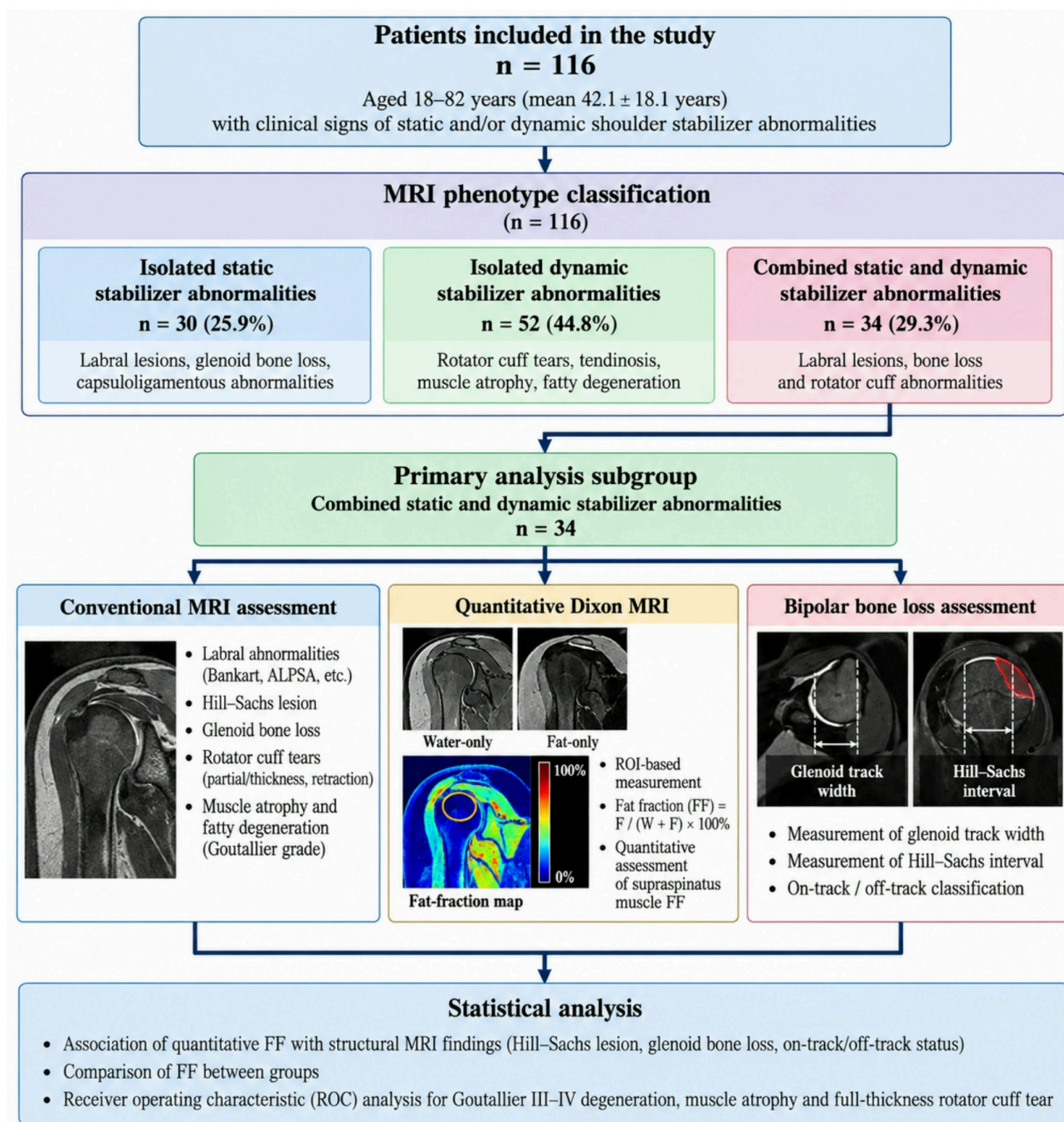


Figure 1

Study population and analysis workflow. Flow diagram showing the overall cohort of 116 patients, classification into isolated static, isolated dynamic, and combined static-dynamic MRI phenotypes, and selection of the combined subgroup (n = 34) for the primary analysis.