

SHOULDER AND ELBOW

Unrepaired rotator cuff tears following acromioplasty

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Abstract

Background: The natural history of rotator cuff tears suggests that not all tears progress and small isolated rotator cuff tears might heal. To date there have been no long-term studies assessing the MRI changes of unrepaired full thickness supraspinatus tears after acromioplasty.

Methods: A database of patients from a single surgeon that have had acromioplasty without rotator cuff repair in the last five to 15 years was reviewed. The number of shoulders examined in this study was 17. Patients all completed an Oxford score, Constant score and had an MRI to assess fatty infiltration and atrophy.

Results: Mean follow-up time 7.2 years (range, 5–13 years). The patients were divided into three groups depending on the size of the tear at time of surgery: C1 tears (<1 cm) n=5, C2 tears (1–1.9 cm) n=8, C3 tears (2–3 cm) n=4. In the C2 group, MRI evaluation showed there were two patients that had tears that had regressed in size (CI 1.4–36, 4). In the C3 group, MRI evaluation revealed that all (100%) of the rotator cuff tears had significant fatty infiltration and atrophy. There was no statistical difference between groups when assessing Oxford scores (p=0.75) and Constant scores (p=0.69). There was significant association between increase in tear size and fatty infiltration (p=0.028). A possible association was noted between increase in tear size and atrophy (p=0.054).

Conclusion: Patients had good long-term clinical outcomes irrespective of tear size. It showed that not all tears progressed to significant fatty change and atrophy.

Level of evidence: Level 3

Key words: unrepaired, full thickness supraspinatus tear, acromioplasty

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Introduction

Impingement syndrome and its association with rotator cuff tears is well documented. The clinical outcome after acromioplasty has been well researched and the literature reports good long-term results. Debate, however, continues regarding the optimal and most cost-effective treatment of the older patient group with an associated rotator cuff tear, with studies reporting good functional outcomes without rotator cuff repair.¹

Recent studies have assessed the structural changes of unrepaired rotator cuff tears; however, there has been little research into the long-term clinical outcomes and MRI changes in isolated full thickness tears of supraspinatus after acromioplasty.

The aim of this study was to evaluate the long-term clinical and structural outcomes of a cohort of patients with isolated full thickness tears of supraspinatus who underwent an acromioplasty without rotator cuff repair.

Methods and material

The cohort consisted of patients from a single surgeon database who had been operated on for symptomatic impingement syndrome without repair of a full thickness supraspinatus tear diagnosed at arthroscopy. Tears were categorised as C1 (less than 1 cm), C2 (between 1 and 1.9 cm), and C3 (between 2 and 3 cm) according to the Snyder classification.²

All C1 and C2 rotator cuff tears at arthroscopy were noted to be within the rotator cable as postulated by Burkhart *et al.*³

All patients had failed conservative treatment, and on examination had full active abduction, forward elevation and external rotation, minimal weakness of supraspinatus and full strength of infraspinatus and subscapularis.

Exclusion criteria were follow-up of less than five years, rheumatoid arthritis, diabetes, long-term corticosteroid use, and previous surgery to the shoulder.

Of the 64 patients contacted telephonically, 16 agreed to participate. The other 48 patients were either not contactable, were too ill, had relocated or had passed away.

We had 16 patients that met these criteria; of these 16 patients, there was one patient that had bilateral pathology, so the number of shoulders investigated was 17 (n=17).

Imaging

Patients did not receive an MRI on presentation to the primary surgeon as it was not clinically indicated at the time, but were assessed clinically and had X-ray confirmation of type 3 acromial morphology as described by Bigliani *et al.*²

On follow-up, all study participants had an MRI of both shoulders. The MRI images were assessed by an experienced musculoskeletal radiologist and a fellowship-trained shoulder surgeon who had no information regarding the patients' clinical outcome.

Continuity or rupture was assessed on T2-weighted coronal imaging sequence. A tear was diagnosed if there was no continuity in the muscle fibres of the rotator cuff.

The quality and quantity of the rotator cuff was assessed on parasagittal T1-weighted turbo spin echo images taken parallel to the glenohumeral joint.

Fatty infiltration was assessed as described by Goutallier *et al.*,⁵ modified by Fuchs *et al.*⁶ for MRI.

Supraspinatus atrophy was determined using the tangent sign as described by Zanetti *et al.*⁷

Clinical assessment

Clinical assessment was done according to a standardised technique using a handheld goniometer and dynamometer.

The Constant score⁸ was recorded where a maximum of 100 points could be obtained. Shoulder range of movement was measured using a handheld goniometer as described by Constant and Murley.⁸ Abduction strength was measured using a handheld dynamometer applied to the wrist. The arm was abducted in the scapular plane, elbow extended, and forearm pronated. Three readings were taken and the average of the three readings was recorded.

An Oxford score⁹ was completed by all patients examined, where a maximum score of 48 could be obtained.

Statistical methods

The data was analysed using Stata 13.0 (Statacorp LP, 4905 Lakeway Drive, College Station, TX77845, USA). Categorical data between the groups was analysed using the chi-squared test or Fisher's exact test as appropriate. Normality of continuous data was assessed using the Shapiro Wilk test. Normally distributed data was summarised using means and standard deviations, and compared using the Student's t-test. Skewed data was summarised using medians and ranges, and groups compared using the Mann-Whitney test. Statistical significance was set at p<0.05 and all tests were two-sided.

Results

There were 17 shoulders in 16 patients (nine females). Mean follow-up was 7.2 years (range, 5–13 years). The mean age at surgery was 65.8 years (range, 50–82 years) and the mean age at most recent follow-up was 73 years (range, 60–89 years).

The patients were divided into three groups depending on the size of the tear at time of arthroscopy. In this study, we had five C1 tears, eight C2 tears and four C3 tears (Table 1).

C1 tears (n=5)

The average age at surgery was 60.4 years (range, 49–75 years). The average age of patients with C1 tears at most recent follow-up was 67.4 years (range, 60–82 years).

The median Constant score was 86 (range 83–96) and median Oxford score was 47 (range 46–48).

Only one patient in the C1 tear group had significant fatty infiltration (grade IV) and a positive tangent sign indicating significant muscle atrophy.

C2 tears (n=8)

The average age at surgery was 65.3 years (range, 58–81 years). The average age of patients with C2 tears at most recent follow-up was 76.7 years (range, 69–81 years). The mean Constant score was 72.3 (range 62–98) and mean Oxford score was 45 (range 42–48).

Only one patient in this group had significant fatty infiltration (grade IV).

On MRI two tears in the group had regressed in size and were reported as partial thickness tears.

Table 1: Comparison of tear size and age at surgery/follow-up

	C1 tears	C2 tears	C3 tears
Age at surgery (range years)	60.4 years (49–75)	65 years (58–71)	69 years (59–73)
Age at follow-up (range years)	67.4 years (60–82)	76.7 years (69–81)	76.2 years (72–83)

Table II: Table showing the clinical parameters for the three categories of tear (based on tear size at surgery) (n=17)

	C1 tears	C2 tears	C3 tears	p-value
Mean abduction in kg (SD)	11.1 (7.5)	11.9 (7.1)	6.4 (3.3)	*p=0.4
Mean forward flexion Degrees (SD)	168.2 (7.5)	170 (7.8)	157.2 (7.2)	*p=0.04
Mean lateral elevation Degrees (SD)	171 (4.3)	170.8 (5.2)	162.8 (5.9)	*p=0.04
Median external rotation average (range) *** see key	4 (2–5)	5 (5–5)	4.5 (3–5)	**p=0.7
Median internal rotation Average (range) * see key	5 (5–6)	5 (5–6)	5 (5–6)	**p=0.8

*Means were compared between the three categories using ANOVA

**Medians were compared between the three categories using the Mann-Whitney U test

***External rotation: 1) hand behind head, elbow forward; 2) hand behind head, elbow back; 3) hand to top of head, elbow forward; 4) hand to top of head, elbow back; 5) full elevation

*** Internal rotation: 1) lateral thigh; 2) buttock; 3) lumbosacral junction; 4) waist; 5) T12 vertebra; 6) interscapular T7

Table III: Comparison of median Oxford and median Constant scores by tear size (n=17)

	C1	C2	C3	p-value
Median Oxford score (range)	47 (25–48)	45 (37–48)	45 (42–48)	p=0.75
Median Constant score (range)	86 (52–96)	72.3 (78–98)	72.3 (65–86)	p=0.69

Medians were compared between the three groups using Kruskal-Wallis chi-squared test.

Table IV: Comparison of tear size and proportion of muscle with fatty infiltration at follow-up, n=17

	Stage 0 (%)	Stage 1 (%)	Stage 2 (%)	Stage 3 (%)	Stage 4 (%)
C1 tears	40.0	20.0	20.0	20.0	0.0
C2 tears	0.0	62.0	25.0	0.0	12.5
C3 tears	0.0	0.0	0.0	0.0	100.0

(Fisher's exact test, p=0.028)

Table V: Comparison of tear size and proportion atrophy

	Tangent sign negative (%)	Tangent sign positive (%)
C1 tear	80.0	20.0
C2 tear	75.0	25.0
C3 tear	0.0	100.0

(Fisher's exact test, p=0.032)

Table VI: Comparison of fatty infiltration and abduction strength

Fatty infiltration stage (Goutallier ⁵)	Stage 0 (n=2)	Stage 1 (n=6)	Stage 2 (n=4)	Stage 3 (n=1)	Stage 4 (n=4)
Mean abduction strength kg (range kg)	9.4 kg (4.8–14.0)	16.0 kg (6.6–22)	10.4 kg (5.3–15.3)	3.5 kg	4.2 kg (1.4–8.4)

Spearman's rho, p=0.009

C3 tears (n=4)

The average age at surgery was 69 years (range, 59–73 years) The average age of patients with C3 tears at most recent follow-up was 76.2 years (range, 72–83 years). The mean Constant score was 72.3 (range 65–98). The mean Oxford score was 45 (range, 42–48).

On MRI all four patients had stage 4 fatty infiltration and a positive tangent sign.

Comparison of clinical parameters

There was no significant difference between the three groups when comparing abduction strength: Oneway ANOVA p=0.31); internal rotation (Kruskal-Wallis chi-squared p=0.8); and external rotation Kruskal-Wallis chi-squared p=0.7). There was, however, a statistical difference in range of motion when comparing forward flexion (Oneway ANOVA p=0.04); and lateral elevation (Oneway ANOVA p=0.04) between the three categories of tears (Table II).

There was no significant difference in Oxford (Kruskal-Wallis chi-squared p=0.75) and Constant scores (Kruskal-Wallis chi-squared p=0.69) when comparing the three groups (Table III).

Comparison of MRI findings

When assessing the fatty infiltration, atrophy and tear size, the C3 tears did have a statistically higher chance of having fatty infiltration (Pearson chi-squared p=0.028). The C3 tears also had significant atrophy (Pearson chi-squared p=0.032), as indicated by the tangent sign (Tables IV and V).

In the C2 group, two of the full thickness tears were now reported as being partial thickness tears and smaller in dimension than reported on initial surgical assessment. This equates to 12% of the total number of shoulders assessed (CI: 1.4–36.4).

The association between increasing age and atrophy Wilcoxon rank-sum (p=0.05) was also noted to be significant, as well as age and fatty infiltration Wilcoxon rank-sum (p=0.008).

The comparison between fatty infiltration stages and abduction strength revealed that stage 0 (n=2) had mean strength of 9.4 kg (4.8 kg–14.0 kg); stage 1 (n=6) abduction strength mean 16.0 kg (range 6.6 kg – 22.kg); stage 2 (n=4) mean abduction strength 10.4 kg (range 5.3 kg – 15.3 kg); stage 3 (n=1) abduction strength 3.5 kg; and the stage 4 (n=4) abduction strength was 4.2 kg (range 1.4 kg – 8.4 kg). There was a statistically significant association between muscle atrophy and abduction strength, Spearman's p=0.009 (Table VI).

Discussion

The reason for progression of isolated tears of the rotator cuff in impingement syndrome is multifactorial. Understanding the natural history of rotator cuff tears is important when evaluating whether acromioplasty has any effect on rotator cuff tear progression.

The article by Melis *et al.*¹⁰ that looked at the natural history of fatty infiltration and atrophy in tears of the supraspinatus muscle, suggests that tears progressed to stage 2 fatty infiltration by four years and severe fatty infiltration (stage 3/4) by six years. Traumatic tears progressed faster, and fatty infiltration of at least stage 2 was present at three years. Muscle atrophy was noted in all tear types to occur at an average of 4.5 years post-symptomatic onset. We did not see this progression in atrophy and fatty infiltration in the small isolated tears of the rotator cuff in our study.

This assumption that all tears progress is now being disputed. Fucentese *et al.* and Maman *et al.*^{11,12} have both shown that small tears may get smaller and may even heal if they are isolated tears. The progression of isolated rotator cuff tears in the context of impingement syndrome following acromioplasty and subacromial decompression has not yet been quantified. In this study we confirmed that small tears may appear to heal on MRI, and our figure of 12% is in keeping with the literature of 8–9%^{11,12} healing rates in patients without impingement syndrome.

The aetiology of rotator cuff tears in impingement syndrome can be attributed to extrinsic, intrinsic and genetic factors.

Acromial morphology as an extrinsic cause for rotator cuff tears and their progression is well described. A recent article¹³ looking at relationship of radiographic acromial characteristics and rotator cuff diseases showed that the presence of an acromial spur is highly associated with a rotator cuff tears in the symptomatic and asymptomatic patient. In Bigliani's⁴ description of acromial morphology, a type 3 acromion has a higher incidence of rotator cuff tears. However, in the Maman *et al.*¹² review there was no statistical correlation between acromial spur and rotator cuff tears. The presence of an acromial spur or abnormal acromial morphology does not always correlate with clinical impingement symptoms. Therefore, there has been suggestion that intrinsic causes may be responsible for symptomatic impingement syndrome and rotator cuff degeneration.

The intrinsic theories of cuff degeneration and tears suggest that there is a zone of hypovascularity and relative hypoxia that may lead to apoptosis and rotator cuff damage.^{14–16} This abnormal cuff is now dysfunctional which results in an uncentred high riding humeral head which causes the acromial morphological changes.

Articular-sided tears are also more common than bursal-sided tears, which may suggest intrinsic pathology. Ogata and Uthoff¹⁷ showed that cuff degeneration and tears progress with age but acromial degeneration did not. Hyvönen *et al.*¹⁸ reported that open acromioplasty did not prevent tear progression in impingement syndrome as they found that tears may appear after acromioplasty in the shoulder where no tear was present at time of surgery.

There have also been studies looking at surgical versus non-surgical or structured exercise regimens for the treatment of impingement syndrome, which have shown that long-term outcomes are similar when comparing acromioplasty versus non-surgical interventions.^{19–21}

A randomised controlled study performed by Kukkonen *et al.*¹ demonstrated that operative treatment is no better than conservative treatment in the management of supraspinatus tears in impingement syndrome. Follow-up was one year in this study. Kukkonen suggests that conservative treatment be the primary method of treatment.¹

These findings may support the theories that cuff degeneration is due to intrinsic mechanisms, and surgery may not alter the natural history of rotator cuff pathology in impingement syndrome.

There has been evidence to suggest that rotator cuff pathologies and symptoms may be genetic and there is a subset of patients that have increased genetic susceptibility in developing tears and subsequent tear progression. Harvie *et al.*²² has shown that siblings have a 2.42 relative risk of developing full thickness rotator cuff tears and a 4.65 relative risk of the tears being symptomatic.

The variability noted in the healing response of the rotator cuff in a cross-section of patients is now being attributed to alterations in genetic expression. Genetic expression which controls cellular, vascular and extracellular matrix formation is controlled by biological and local mechanical factors at the tendon edge. It is postulated that surgical repair alters the biological environment at the tendon edge and therefore modulates gene expression.²³ Repair of the rotator cuff in sheep and rabbits however, has not shown to reverse fatty infiltration, but early repair may result in partial recovery according to Kang and Gupta,¹⁵ and therefore early repair in the traumatic full thickness tears and early presenting degenerative tears is still of value. Established stage 3 and 4 fatty infiltration and atrophy; however, was not reversible with surgical repair.^{10,11,24} The genetic expression of protein degradation genes which results in proteolysis and muscle degeneration is up-regulated in massive rotator cuff tears.²⁵ This may explain the association with larger rotator cuff tears and significant fatty infiltration and atrophy.

The pathophysiology of fatty degeneration is related to unloading of the muscle due to its loss of insertion into bone; this result in changes in physiological structure and function of the muscle and ultimately atrophy.^{24,26} This unloading of the muscle may ultimately be the reason that the larger tears progressed with respect to fatty changes and atrophy. In smaller tears there is less unloading of the muscle and therefore less propensity to atrophy and degeneration.

The rotator crescent and rotator cable theory by Burkhart *et al.*³ describes two distinct areas of the rotator cuff with different load characteristics. The rotator cable which is relatively thicker supports more load and shields the crescent area. Burkhart hypothesised that as people age there is progressive thinning of this relatively avascular crescent zone. This hypothesis has been recently supported by Kim *et al.*²⁷ who have shown that most degenerative tears occur in a more posterior location near the junction of the infraspinatus and supraspinatus and not anteriorly as initially thought. The biomechanical relevance of this tear position is that the cable is still intact, which has been shown in biomechanical studies by Meisha *et al.*²⁸ to be the primary load-bearing structure in the supraspinatus. Therefore, tear position and not only tear size are important to consider when assessing the clinical effects of rotator cuff tears. In this study it was noted at arthroscopy that all the C1 and C2 tears were within this cable, hence the good clinical parameters achieved by the study participants.

The fibrogenic and adipogenic progenitor cells in muscle that are responsible for fatty change are normally inactive in healthy muscle. The activation and differentiation of these cells has been found to be initiated by ageing, oxidative stress and muscle degeneration. This fibroproliferative response in the older patient negatively affects the rotator cuff muscle's ability to regenerate.^{24,26} The age of the patient is therefore important in assessing the healing capability of a rotator cuff tear and is of relevance in this study as we had an elderly patient cohort.

In this study it was noted that isolated small full thickness tears had the best outcomes when assessing the clinical parameters of Constant and Oxford scores. The small tears also had better structural outcomes, whereas the larger tears did considerably

worse structurally but not clinically. Norlin and Adolfsson²⁵ concluded that small full thickness tears do well post acromioplasty at 10 to 13 years post-surgery, which correlates with our findings.

Ozaki et al.³⁰ showed that 38% of cadavers over 60 years had cuff tears; this figure rose to 80% in cadavers over 90 years of age, where most of these tears are asymptomatic. The prevalence of asymptomatic full thickness tears in the population aged 40–60 years is 4% and patients over 60 years is 24%, according to an MRI study by Sher et al.³¹ From this data it can be noted that rotator cuff tears are relatively common and that not all patients with cuff tears present with a loss in function or pain, which might explain the good clinical outcomes we found in this age group of patients.

Weaknesses of this study include poor patient follow-up, and low numbers available for this study which was due to the fact that 11 patients had died, seven were too ill, eight patients refused, five had moved and 15 were not contactable. The patients were also pre-selected as having good functional capacity prior to surgery which was one of the reasons not to repair the rotator cuff. No pre-operative MRI was done to evaluate progression of fatty infiltration and atrophy with current MRI findings at follow-up.

Conclusions

Currently this is the only long-term MRI study of patients with full thickness supraspinatus tears that were not repaired at the time of acromioplasty.

This small cohort of patients had good long-term clinical outcomes irrespective of tear size. It also showed that not all tears progressed to significant fatty change and atrophy in the long term. Full thickness tears may progress or heal despite surgical intervention. The process of muscle regeneration or degeneration in muscles with tears seems to be more influenced by local biological factors, genetic expression and tear location, and therefore acromioplasty in isolation does not influence rotator cuff tear healing. The recommendation that can be made from this study is that small tears within the cable do not need to be fixed and in this elderly population this would facilitate less rehabilitation and earlier mobilisation. Larger C3 tears, upon discussion with the patient, can be treated without repair and with shorter rehabilitation time if patients are willing to accept some functional loss.

Ethics statement

This study received approval by the Human Research Ethics Committee, University of Cape Town: ref number 183/2013.

Declarations

The authors declare authorship of this article and that they have followed sound scientific research practice. This research is original and does not transgress plagiarism policies.

Author contributions

DC was the primary author and researcher. AL was the primary surgeon in all the cases mentioned and provided the patient data base. BV reviewed the written submission. RD contributed in data collection and research. SR provided the original idea and assisted in writing the article.

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References

1. Kukkonen J, Joukainen A, Lehtinen J, Mattila KT, Tuominen EK, Kauko T, et al. Treatment of non traumatic rotator cuff tears, a randomised control trial with one year clinical follow-up. *The Bone and Joint Journal* 2014;**96**-B:75-81. [http:// dx. doi: 10.1302/0301-620X.96B1.32168](http://dx.doi.org/10.1302/0301-620X.96B1.32168)

2. Snyder SJ, editor. Arthroscopic classification of rotator cuff lesions and surgical decision making. In: *Shoulder Arthroscopy*. 2nd edition. Philadelphia Lipencott Williams & Wilkins; 2003. pp 201-207.
3. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable : an anatomic description of the shoulder's 'suspension bridge'. *Arthroscopy: The Journal of Arthroscopic and Related Surgery* 1993;**9**(6):611-16.
4. Bigliani BU, Morrison ES, April EW. The morphology of the acromion and its relationship to rotator cuff tears. *Orthop Trans* 1986;**10**:216.
5. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre and postoperative evaluation by CT scan. *Clin Orthop Relat Res*.1994;**304**:78-83.
6. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. *J Shoulder Elbow Surg*. 1999;**8**:599-605.
7. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. *Invest Radiol*. 1998;**33**:163-70.
8. Constant CR, Murley AH. A clinical method of functional assessment of the shoulder. *Clin Orthop Relat Res*.1987;**214**:160-64.
9. Dawson J, Fitzpatrick R, Carr A. Questionnaire on the perception of patients about shoulder surgery. *Journal of Bone and Joint Surgery*. 1996;**78**:593-600.
10. Melis B, DeFranco MJ, Chuinard C, Walch G. Natural history of fatty infiltration and atrophy of the supraspinatus muscle in rotator cuff tears. *Clin Orthop Relat Res*. 2010;**468**(6):1498-1505. [http:// dx. doi: 10.1007/s11999-009-1207-x](http://dx.doi.org/10.1007/s11999-009-1207-x)
11. Fucentese SF, Von Roll AL, Pfirrmann WA, Gerber C, Jost B. Evolution of nonoperatively treated symptomatic isolated full-thickness supraspinatus tears. *J Bone Joint Surg*. 2012;**94**(9):801. [http:// dx. doi: 10.2106/JBJS.I.01286](http://dx.doi.org/10.2106/JBJS.I.01286).
12. Maman E, Harris C, White L, Tomlinson G, Shashank M, Boynton E. Outcome of nonoperative treatment of symptomatic rotator cuff tears monitored by magnetic resonance imaging. *J Bone Joint Surgery Am*. 2009;**91**(8):1898-1906. [http:// dx. doi: 10.2106/JBJS.G.01335](http://dx.doi.org/10.2106/JBJS.G.01335).
13. Hamid N, Omid R, Yamaguchi K, Steger-May, Keener JD, Stobb G. Relationship of radiographic acromial characteristics and rotator cuff disease: prospective investigation of clinical, radiographic and sonographic findings. *J Shoulder Elbow Surg*. 2012 Oct; **21**(10):1289-98. [http:// dx. doi: 10.1016/j.jse.2011.09](http://dx.doi.org/10.1016/j.jse.2011.09)
14. Harrison AK, Flatow EL. Subacromial impingement syndrome. *J Am Acad Orthop Surg*. 2011;**19**(11):701-708.
15. Kang JR, Gupta R. Mechanisms of fatty degeneration in massive rotator cuff tears. *J Shoulder Elbow Surg*. 2012;**21**:175-80. [http:// dx.oi.org/10.1016/j.jse.2011.11.017](http://dx.doi.org/10.1016/j.jse.2011.11.017)
16. Lohr JF, Uthoff HK. The microvascular pattern of the supraspinatus tendon. *Clin Orthop Relat Res* 1990;**254**:35-38.
17. Ogata S, Uthoff HK. Acromial enthesopathy and rotator cuff tear: A radiologic and histologic post-mortem investigation of the coracoacromial arch. *Clin Orthop Relat Res*. 1990;**254**:39-48.
18. Hyvönen P, Lohi S, Jalovaara P. Open acromioplasty does not prevent the progression of an impingement syndrome to a tear. Nine-year follow-up of 96 cases. *J Bone Joint Surg Br*. 1998;**80**(5):813-16.
19. Budoff JE, Nirschl RP, Guidi EJ. Debridement of partial thickness tears of the rotator cuff without acromioplasty: long-term follow-up and review of the literature. *J Bone Joint Surg Am* 1998;**80**(5):733-48.
20. Ketola S, Lehtinen J, Rousi T, Nissinen M, Huhtala M, Konttinen YJ, Arnala I, et al. No evidence of long-term benefits of arthroscopic acromioplasty in the treatment of shoulder impingement syndrome. *Upper Limb* 2013;**2**(7):132-39. [http://dx. doi: 10.1302/2046-3758.27.2000163](http://dx.doi.org/10.1302/2046-3758.27.2000163)
21. Kuhn JE. Exercise in the treatment of rotator cuff impingement: A systematic review and a synthesized evidence-based rehabilitation protocol. *J Shoulder Elbow Surg* 2009;**18**(1):138-60. [http:// dx. doi: 10.4085/1062-6050-45.5.483](http://dx.doi.org/10.4085/1062-6050-45.5.483)

22. Harvie P, Ostlere SJ, Teh J, McNally EG, Clipsham K, Burston BJ. Genetic influences in the aetiology of tears of the rotator cuff. Sibling risk of full-thickness tear. *J Bone Joint Surg Br* 2004;**86**:696-700. DOI: <http://dx.doi.org/10.1302/0301-620X.86B5.14747>
23. Chaudhury S, Carr AJ. Lessons we can learn from gene expression patterns in rotator cuff tears and tendinopathies. *J Shoulder Elbow Surg* 2012;**21**:191-99. <http://dx.doi.org/10.1016/j.jse.2011.10.022>
24. Barry J, Lansdown DA, Cheung S, Feeley BT, Benjamin C. The relationship between tear severity, fatty infiltration, and muscle atrophy in the supraspinatus. *J Shoulder Elbow Surg* 2013;**22**:18-25 <http://dx.doi.org/10.1016/j.jse.2011.12.014>
25. Schmutz S, Fuchs T, Regenfelder F, Steinmann P, Zumstein M, Fuchs B. Expression of atrophy mRNA relates to tendon tear size in supraspinatus muscle. *Clin Orthop Relat Res*. 2009;**467**(2):457-64. <http://dx.doi.org/10.1016/j.jse.2011.10.022>
26. Laron D, Samagh SP, Liu X, Kim HT, Feeley BT. Muscle degeneration in rotator cuff tears. *J Shoulder Elbow Surg* 2012;**21**:164-74. <http://dx.doi.org/10.1016/j.jse.2011.09.027>
27. Kim HM, Dahiya N, Teefey SA, Middleton WD, Stobbs G, Steger M, Yamaguchi K. Location and initiation of degenerative rotator cuff tears: an analysis of three hundred and sixty shoulders. *J Bone Joint Surg Am*. 2010 May;**92**(5):1088-96.<http://dx.doi.org/10.2106/JBJS.I.00686>
28. Meisha MM, Derwin KA, Sibole SC, Erdemir A, McCarron JA. The biomechanical relevance of anterior rotator cuff cable tears in a cadaveric shoulder model. *J Bone Joint Surg Am*. 2013 Oct 16;**95**(20):1817-24. <http://dx.doi.org/10.2106/JBJS.L.00784>
29. Norlin R, Adolfsson L. Small full-thickness tears do well ten to thirteen years after arthroscopic subacromial decompression. *J Shoulder Elbow Surg*. 1993;**17**(1 Suppl):12S-16S.
30. Ozaki J, Fujimoto S, Nakagawa Y, Mashura K, Tamai S. Tears of the rotator cuff associated with pathological changes in the acromion. *J Bone Joint Surg* 1988;**70**:1224-30.
31. Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on MRI of asymptomatic shoulders. *J Bone Joint Surg* 1995;**77**:10-15. ■