ORIGINAL ARTICLE

CiSE Clinics in Shoulder and Elbow

Clinics in Shoulder and Elbow Vol. 19, No. 4, December, 2016
https://doi.org/10.5397/cise.2016.19.4.197

Similar Degree of Degeneration in the Articular and Bursal Layers of Delaminated Rotator Cuff Tear

Chris Hyunchul Jo✉, Seung Hoo Lee, Ji Sun Shin, Ji Eun Kim[1]

Departments of Orthopedic Surgery and [1]Pathology, SMG-SNU Boramae Medical Center, Seoul National University College of Medicine, Seoul, Korea

Background: The purpose of the study was to compare the degree of degeneration of the articular and bursal layers of delaminated supraspinatus tendons based on histological examination.
Methods: Fifty-four patients with a full-thickness rotator cuff tear were included in the study. Tendon specimens were harvested during arthroscopic rotator cuff repair from the lateral torn edges of the articular and bursal layers of the delaminated tear. Harvested samples were stained with H&E dye and evaluated based on a semi-quantitative grading scale.
Results: There were no significant differences in the seven histological characteristics of tendon degeneration: fiber structure, fiber arrangement, round nuclei, regional variations in cellularity, vascularity, collagen stainability, and hyalinization between the articular and bursal layers of the delaminated rotator cuff tear (all $p>0.05$). Total degeneration scores of articular and bursal sides were 13.1 ± 3.85 points and 13.2 ± 3.42 points, respectively, and were not significantly different ($p=0.958$).
Conclusions: The study demonstrates that tendon degeneration was similar in the articular and bursal sides of the delaminated full-thickness rotator cuff tear, suggesting that degeneration would be a main etiology for the rotator cuff tear not only in the articular side but also in the bursal side. Considering potential disadvantages of subacromial decompression, this study tentatively suggests routine use of subacromial decompression as well as the need for halting or recovery from rotator cuff degeneration for better rotator cuff repair.
(Clin Shoulder Elbow 2016;19(4):197-201)

Key Words: Rotator cuff; Delamination; Degeneration; Histology

Introduction

Rotator cuff disease is one of the most common causes (up to 70%) of shoulder pain.[1)] The prevalence of symptomatic rotator cuff diseases increases with age, occurring in approximately 2.8% of those older than 30 years and in 21% of those older than 70 years.[2)] Rotator cuff diseases lead to more than 4.5 million physician visits per year, and over 300,000 rotator cuff repairs are performed annually costing more than $3 billion,[2,3)] suggesting that rotator cuff diseases are associated with high socioeconomic cost and burden.

Several theories were reported on the pathophysiology of rotator cuff disease such as impingement, overuse, and multifactorial as extrinsic ones as well as hypoperfusion, degeneration, degeneration-microtrauma, apoptosis, and extracellular matrix modifications as intrinsic ones.[4)] Whereas the exact etiology of rotator cuff tears remains to be elucidated, tendon degeneration has been recently considered as one of the most important reasons.[5-8)]

Delamination is defined as a horizontal split of the torn tendon end and is a common finding during arthroscopic shoulder surgery with a reported prevalence of 31% to 71%.[9-11)] Whereas the exact mechanism of delamination is not known, it can occur between layers 2 and 3 due to different histological properties

Received January 14, 2016. **Revised** May 27, 2016. **Accepted** May 30, 2016.

✉**Correspondence to:** Chris Hunchul Jo
Department of Orthopedic Surgery, SMG-SNU Boramae Medical Center, Seoul National University College of Medicine, 20 Boramae-ro 5-gil, Dongjak-gu, Seoul 07061, Korea
Tel: +82-2-870-2315, **Fax:** +82-2-840-2453, **E-mail:** chrisjo@snu.ac.kr

IRB approval (No. 06-2009-101).

Financial support: This work was supported by a clinical research grant-in-aid from the SMG-SNU Boramae Medical Center (03-2015-7).
Conflict of interests: None.

pISSN 2383-8337
eISSN 2288-8721

such as varying collagen direction and degeneration degree as well as biomechanical properties of the two layers. Therefore, delamination can be considered as a form of degeneration within the tendon.[10,12-15] Clinical implications of the delamination are also controversial. Some authors have reported it as negative effects on outcome,[16] whereas some have showed no significant difference.[10,11] Thus, despite commonly reported findings, little is known about delamination, and only one study has carried out a histological assessment.[12]

The purpose of the study was to compare the degree of degeneration of the articular and bursal sides of the delaminated supraspinatus tendon based on histological examination. The hypothesis is that there would be a difference in degeneration scores with respect to harvesting location.

Methods

Study Design and Patients

This study was approved by SMG-SNU Boramae Medical Center Institutional Review Board, and all patients provided informed consent. The inclusion criteria were a full-thickness rotator cuff tear with delamination and available tissue samples of the rotator cuff tendon harvested at the time of surgery. The exclusion criteria were inflammatory arthritis, including rheumatoid arthritis, a history of acute trauma and infection, previous subacromial injection within the past 3 months, systemic conditions associated with chronic pain, isolated subscapularis tear, rotator cuff arthropathy, calcific tendinitis, retear, and absence of tissue samples.

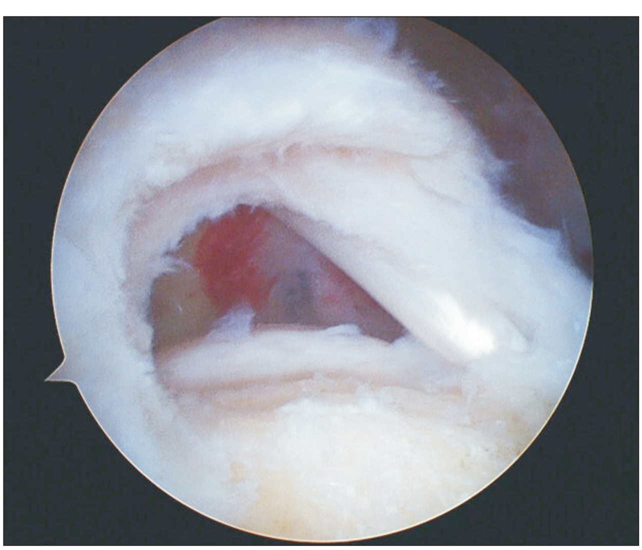

Fig. 1. Artroscopic findings of full-thickness rotator cuff tear with delamination.

Tendon Harvesting and Histological Assessments

A rotator cuff tendon was harvested during an arthroscopic rotator cuff repair using a basket forcep. Specimens with a size of 3×3 mm were taken from the articular and bursal layers of the delaminated supraspinatus tendon (Fig. 1). Harvested tendon was fixed with 5 ml of 10% formalin in a plastic pathology container. The samples were dehydrated, embedded in paraffin, cut into 4-μm sections, and stained with H&E dye. Three sections were made for each tendon. One section was randomly selected and examined with a light microscope. Whole area of each slide was observed, and the most severely degenerated area, possibly expecting the worst score, was selected and used in the study.[6] Examination was performed by a fellowship trained orthopedic surgeon and pathologist. Each slide was evaluated with a semi-quantitative grading scale of Aström and Movin modified by Maffulli.[6,17,18] The parameters included in the scale were fiber structure, fiber arrangement, rounding of nuclei, regional variations in cellularity, increased vascularity, decreased collagen stainability, and hyalinization. A 4-point scoring system was used; 0 indicates normal appearance, 1 slightly abnormal, 2 moderately abnormal, and 3 markedly abnormal. The following scheme was used: fiber structure (0=linear, no interruption, 3=short with early truncation); fiber arrangement (0=well ordered and regular, 3=no pattern identified); appearance of nuclei (0=flat, 3=rounded); regional variations in cellularity (0=uniform; 3=high regional variation); vascularity (0=absent, 3=high); collagen stainability (0=vivid, 3=pale); and hyalinization (0=absent, 3=high). The total score of tendon degeneration for a given slide could range from 0 (normal tendon) to 21 points (most severely degenerated). Intra- and interobserver reliability testing was proven to be high to excellent in a previous report.[6]

Statistical Analysis

Scale values of histological parameters and total degeneration scores were compared using t-test. Analyses were performed using SPSS ver. 13.0 Statistics (SPSS Inc., Chicago, IL, USA). The significance level was set at $p<0.05$ for all tests.

Results

Patients

Fifty-four patients were included in the study (Table 1).[16,19] The mean age was 61.4 ± 7.7 years. There were 16 males and 38 females. The majority of patients had medium-sized tears with a Goutallier grade of 1 or 2 for fatty infiltration and Tangent sign of 1 for muscle atrophy.

Histologic Assessments

Generally, specimens from both the articular and bursal sides showed similar characteristics of degenerated tendons, including loss of finer fiber structure and parallel arrangement, increased number of round nuclei, increased cellularity and vascularity, reduced and pale H&E stain, and evidence of hyalinization (Fig. 2).

Degeneration scores of the fiber structure, fiber arrangement, round nuclei, regional variations in cellularity, vascularity, collagen stainability, and hyalinization of the two groups were not significantly different (all $p>0.05$) (Table 2, Fig. 3). Total degeneration scores of the articular and bursal sides were 13.1 ± 3.85 points and 13.2 ± 3.42 points, respectively, and were not significantly different ($p=0.958$) (Fig. 4).

Table 1. Baseline Characteristics of Patients (n=54)

Characteristic	Value
Age (yr)	61.4 ± 7.7
Sex (male:female)	16:38
Symptom (mo)	13.0 ± 19.3
Cofield (small:medium:large:massive)	4:33:8:9
Boileau (I:II:III:IV)*	24:12:11:7
Visual tendon grade (A:B:C)†	0:52:2
Goutallier grade of the supraspinatus (0:1:2:3:4)	0:12:52:12:5
Tangent sign (1:2:3)‡	35:9:10
Occupation ratio (1:2:3)§	23:23:8

Values are presented as mean ± standard deviation or number only.
*Stage I:stage II:stage III:stage IV.[16)]
†Tendon grade assesses rotator cuff quality using three gross tendon criteria; (1) fraying over half of tendon thickness, (2) delamination of supraspinatus tendon, and (3) thinning of less than half of normal thickness. A, none of these criteria were met; B, fraying or delamination was identified; and C, both fraying and delamination or thinning regardless of the other criteria.[19)]
‡Tangent sign assesses muscle atrophy of the supraspinatus. Grade 1, negative, which means that the superior border of the supraspinatus was superior to the line tangential to the coracoid and scapular spine; grade 2, borderline, which means that the superior border was located about the tangential line; grade 3, positive, which means that the superior border was inferior to the tangential line.
§Occupation ratio means the ratio of the cross-sectional area of the supraspinatus to the fossa. Grade 1, 0.6 to 1.0; grade 2, 0.4 to 0.6; grade 3, <0.4.

Discussion

This study shows that the degree of degeneration was not significantly different between the articular and bursal sides of the delaminated tear ($p=0.937$). This result suggests that tendon degeneration would be an important etiology both in the articular and bursal sides of the rotator cuff tendon. Consequently, the result also suggests that acromioplasty might not be crucial for healing after repair. An additional strategy such as biological

Table 2. Parameters and Total Degeneration Scores for Harvested Tendons from Articular and Bursal Sides of the Supraspinatus Tendon

Parameter	Articular	Bursal	*p*-value
Fiber structure	2.3 ± 0.80	2.3 ± 0.78	0.628
Fiber arrangement	2.1 ± 0.86	2.1 ± 0.86	1.000
Rounding of the nuclei	2.4 ± 0.82	2.4 ± 0.74	1.000
Variations in cellularity	2.4 ± 0.85	2.5 ± 0.80	0.416
Increased vascularity	1.6 ± 1.18	1.4 ± 1.28	0.483
Decreased stainability	1.0 ± 0.87	1.0 ± 0.87	0.825
Hyalinization	1.1 ± 0.98	1.2 ± 0.95	0.487
Total	13.1 ± 3.85	13.2 ± 3.42	0.958

Values are presented as mean ± standard deviation.

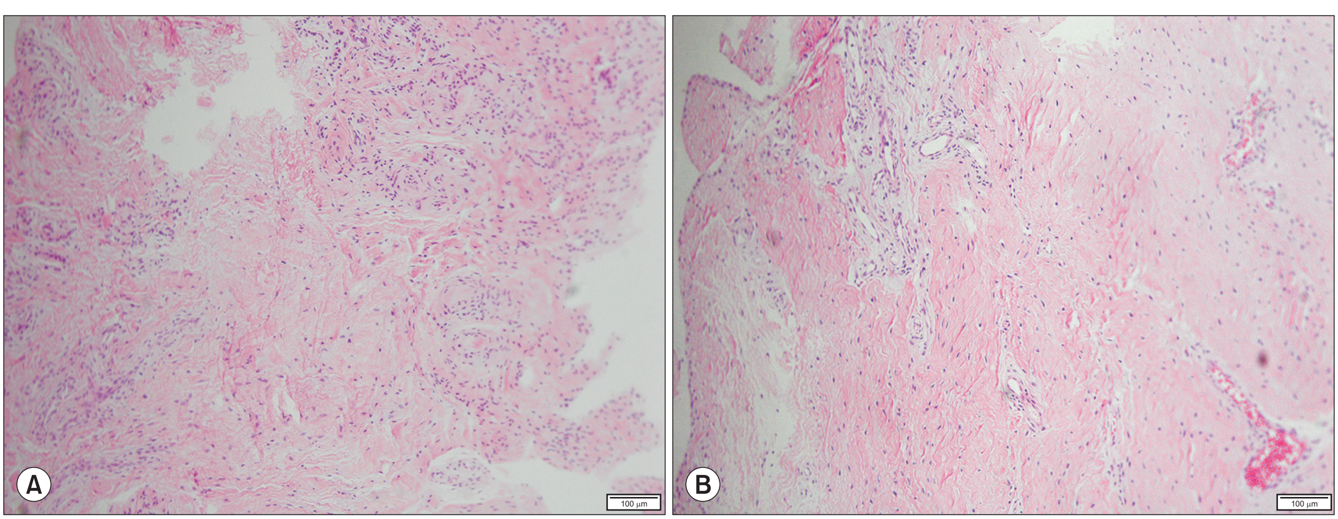

Fig. 2. Histology of torn rotator cuff tendon from the articular side (A) and the bursal side (B) (A, B: H&E). (A) Fiber structure, 2; fiber arrangement, 2; rounding of nuclei, 2; regional variation in cellularity, 3; increased vascularity, 2; decreased collagen stainability, 1; hyalinization, 1; total degeneration score 13. (B) Fiber structure, 2; fiber arrangement, 2; rounding of nuclei, 2; regional variation in cellularity, 2; increased vascularity, 2; decreased collagen stainability, 1; hyalinization, 2; total degeneration score 13.

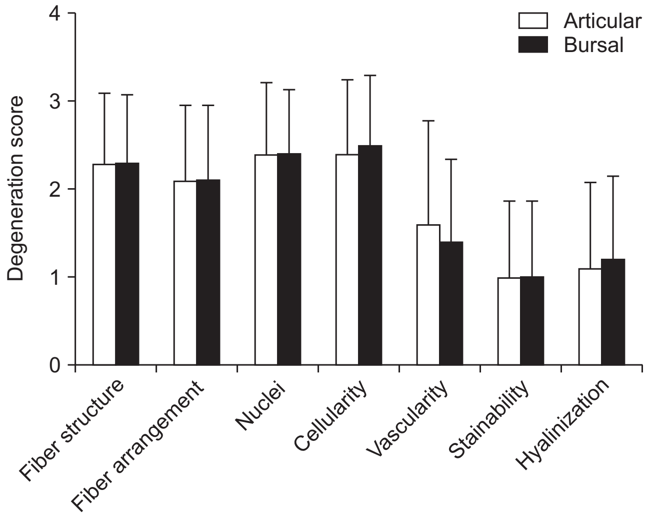

Fig. 3. Comparison of degeneration score between articular side and bursal side. There were no significant differences in all sever parameters between the two groups.

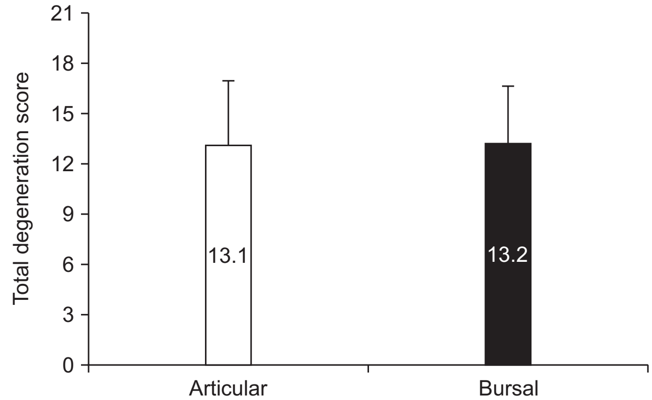

Fig. 4. Total degeneration scores in articular side and bursal side. Mean total degeneration scores of articular and bursal sides were not different (p=0.958).

augmentation would be necessary to reverse or, at the very least, stop or delay degeneration of the rotator cuff tendon besides secure fixation of the torn tendon end to the anatomical location.

To our knowledge, there has been only one previously reported histological evaluation of delaminated rotator cuff tears. Specifically, Matsuki et al.[12] examined seven patients with a delaminated full-thickness rotator cuff tear. The average degeneration score was 6.1 points in the superficial layers and 8.1 points in the deep layers ($p=0.03$), suggesting that the deep layers were more degenerated than the superficial layers. These findings suggest that a rotator cuff tear can initiate from the articular side and then extend to the bursal side and that the early degeneration and tearing of the deep layer would be an important factor in the pathogenesis of delamination.[12] Our results are not consistent with those of Matsuki et al.,[12] as we found no difference between the articular and bursal layers. We detail the possible explanations for the discrepancy as follows. First, the number of samples (n=7) in the study by Matsuki et al.[12] might have been too small to draw a solid conclusion. Second, we used a different histological evaluation system consisting of seven parameters, whereas they used a system with four parameters. However, it is clinically important that our and Matsuki et al.'s results[12] demonstrated that tendon degeneration was present in the bursal side of delamination in addition to the articular side, necessitating appropriate treatment for bursal side degeneration.

It is commonly believed that articular side tears are mainly associated with intrinsic pathology of the rotator cuff, whereas bursal side tears are associated with subacromial impingement.[20,21] For the same reason, it is recommended that a prominent acromial spur be decompressed while the rotator cuff is repaired.[20] However, there is no clear evidence whether acromioplasty facilitates healing of a repaired rotator cuff, prevents progression of rotator cuff tears, or protects the integrity of rotator cuff repair.[22] Routine use of subacromial decompression needs to be reconsidered in spite of potential disadvantages of subacromial decompression, including violation of the soft tissue envelope during arthroscopy leading to intraoperative soft-tissue swelling, weakening of the deltoid origin by detachment of some anterior fibers, anterosuperior instability in the presence of a failed rotator cuff or irreparable tear, and formation of adhesions between the raw exposed bone on the undersurface of the acromion and underlying tendon, which in turn can limit smoothness, motion, comfort, and range of motion.[23] The results of the study showing no differences in degeneration between the articular and bursal sides of the rotator cuff tear support these opinions.

Limitations of this study include a cross-sectional study that includes the later stage of rotator cuff disease, use of a semi-quantitative grading system, only H&E staining, and no evaluation of myxoid, lipoid, or calcific degeneration. The absence of evaluation of the association between acromial shape and bursal side degeneration would also be a potential limitation.

Conclusion

The findings of the study demonstrate that tendon degeneration was similar between the articular and bursal sides of the delaminated full-thickness rotator cuff tear, implying that degeneration would be a main etiology for rotator cuff tears not only in the articular side but also in the bursal side. Considering the potential disadvantages of subacromial decompression, this study suggests cautionary routine use of subacromial decompression as well as the need for halting or recovery from rotator cuff degeneration for better rotator cuff repair.

References

1. Hermans J, Luime JJ, Meuffels DE, Reijman M, Simel DL, Bierma-Zeinstra SM. Does this patient with shoulder pain have rotator cuff disease?: the rational clinical examination systematic review. JAMA. 2013;310(8):837-47.
2. Oh LS, Wolf BR, Hall MP, Levy BA, Marx RG. Indications for rotator cuff repair: a systematic review. Clin Orthop Relat Res. 2007;455:52-63.
3. Colvin AC, Egorova N, Harrison AK, Moskowitz A, Flatow EL. National trends in rotator cuff repair. J Bone Joint Surg Am. 2012;94(3):227-33.
4. Via AG, De Cupis M, Spoliti M, Oliva F. Clinical and biological aspects of rotator cuff tears. Muscles Ligaments Tendons J. 2013;3(2):70-9.
5. Kannus P, Józsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am. 1991;73(10):1507-25.
6. Jo CH, Chang MS. Degeneration exists along the entire length of the supraspinatus tendon in patients with a rotator cuff tear. Clin Shoulder Elbow. 2015;18(2):61-7.
7. Nirschl RP. Rotator cuff tendinitis: basic concepts of pathoetiology. Instr Course Lect. 1989;38:439-45.
8. Hashimoto T, Nobuhara K, Hamada T. Pathologic evidence of degeneration as a primary cause of rotator cuff tear. Clin Orthop Relat Res. 2003;(415):111-20.
9. Flurin PH, Landreau P, Gregory T, et al. Arthroscopic repair of full-thickness cuff tears: a multicentric retrospective study of 576 cases with anatomical assessment. Rev Chir Orthop Reparatrice Appar Mot. 2005;91(S8):31-42.
10. Sonnabend DH, Watson EM. Structural factors affecting the outcome of rotator cuff repair. J Shoulder Elbow Surg. 2002; 11(3):212-8.
11. MacDougal GA, Todhunter CR. Delamination tearing of the rotator cuff: prospective analysis of the influence of delamination tearing on the outcome of arthroscopically assisted mini open rotator cuff repair. J Shoulder Elbow Surg. 2010;19(7): 1063-9.
12. Matsuki K, Murata R, Ochiai N, et al. Histological assessemnt of delamination observed in rotator cuff tears. Shoulder Joint (Katakansetsu). 2006;30(3):461-4.
13. Clark JM, Harryman DT 2nd. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. J Bone Joint Surg Am. 1992;74(5):713-25.
14. Nakajima T, Rokuuma N, Hamada K, Tomatsu T, Fukuda H. Histologic and biomechanical characteristics of the supraspinatus tendon: reference to rotator cuff tearing. J Shoulder Elbow Surg. 1994;3(2):79-87.
15. Gwak HC, Kim CW, Kim JH, Choo HJ, Sagong SY, Shin J. Delaminated rotator cuff tear: extension of delamination and cuff integrity after arthroscopic rotator cuff repair. J Shoulder Elbow Surg. 2015;24(5):719-26.
16. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am. 2005;87(6):1229-40.
17. Movin T, Gad A, Reinholt FP, Rolf C. Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients. Acta Orthopaedica Scand. 1997;68(2):170-5.
18. Aström M, Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. Clin Orthop Relat Res. 1995;(316):151-64.
19. Jo CH, Yoon KS, Lee JH, et al. The effect of multiple channeling on the structural integrity of repaired rotator cuff. Knee Surg Sports Traumatol Arthrosc. 2011;19(12):2098-107.
20. Ko JY, Huang CC, Chen WJ, Chen CE, Chen SH, Wang CJ. Pathogenesis of partial tear of the rotator cuff: a clinical and pathologic study. J Shoulder Elbow Surg. 2006;15(3):271-8.
21. Fukuda H. The management of partial-thickness tears of the rotator cuff. J Bone Joint Surg Br. 2003;85(1):3-11.
22. Chahal J, Van Thiel GS, Mall N, et al. The role of platelet-rich plasma in arthroscopic rotator cuff repair: a systematic review with quantitative synthesis. Arthroscopy. 2012;28(11):1718-27.
23. Goldberg BA, Nowinski RJ, Matsen FA 3rd. Outcome of nonoperative management of full-thickness rotator cuff tears. Clin Orthop Relat Res. 2001;(382):99-107.