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Effect of suprascapular nerve injury on rotator cuff enthesis

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Background: Numerous reports have shown that retracted rotator cuff tears may cause suprascapular nerve injury, and nerve injury causes atrophy and fat accumulation in the rotator cuff muscles. However, the effect of suprascapular nerve injury on rotator cuff enthesis has not been directly defined. This study aimed to investigate the effect of suprascapular nerve injury on rotator cuff enthesis.
Methods: Twenty-four Wistar albino rats underwent bilateral transection of the suprascapular nerve. Additional 6 rats were used as the sham group. Bilateral supraspinatus and infraspinatus entheses were examined after 1, 4, 8, and 12 weeks of nerve transection. Histomorphometric analyses were performed for each zone of enthesis.
Results: Compared with normal enthesis, significant and consistent decrease in cellularity were observed in the tendon and bone at all time points ($P < .001$). Collagen bundle diameter in the tendon also decreased in a similar manner ($P < .001$). Apart from the tendon and bone zones, fibrocartilage and calcified fibrocartilage zones showed similar response, and significant decrease in cellularity was observed 8 weeks after nerve transection ($P < .001$).
Conclusion: This study identifies suprascapular nerve injury as an underlying mechanism leading to compromise of the rotator cuff enthesis structure. Suprascapular nerve injury may be considered as an etiologic factor for the impaired healing after repair of a massive tear.
Level of evidence: Basic Science Study; Histology; Animal Model

Keywords: Rotator cuff; suprascapular nerve; enthesis; histomorphometry; injury; shoulder

The shoulder joint complex is controlled by a sophisticated neuromuscular system that provides both motion and stabilization. Injury in this system may exacerbate the muscle changes induced by rotator cuff tendon tear.[12,14,15] As a part of this structure, special emphasis has been given to the suprascapular nerve (SSN) because it innervates the supraspinatus and infraspinatus muscles and tends to entrapment. SSN injury may be associated with atraumatic rotator cuff tears (RCTs). A cadaveric study reported the effects of RCT retraction on SSN and concluded that supraspinatus retraction dramatically changed the course and caused kinking of the nerve.[2] In addition, repair of the retracted RCTs can set the SSN on tension. Anatomic studies showed that maximum lateral advancement of a retracted RCT is between 1 and 3 cm;

Consent was given for the use of laboratory animals by the Research Ethics Commission for animal experiments (Acibadem University- ACU-HADYEK 2018/02).

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with increased advancement, the neurovascular pedicle is placed under increased tension.[8,35] Clinical studies have also shown that large retracted RCTs can lead to SSN injury, and 38% of patients with massive RCTs involving the supraspinatus and infraspinatus tendons had electrodiagnostic evidence of isolated SS neuropathy.[5] Rotator cuff repair can result in reversal of neuropathy.[5,17] These studies have mostly reported the nerve injury secondary to RCTs. Primary nerve injury can also affect the rotator cuff. Animal studies for fatty degeneration of rotator cuff muscles demonstrated that the RCT and SSN injury cause significant changes in the rotator cuff muscles such as atrophy, fat accumulation, and fibrosis. These changes are more severe in tears with both tendon and nerve detachment than in those with only tendon detachment, suggesting an additional role of nerve injury in muscle degeneration.[12,15] These studies demonstrated the impact of primary nerve injury on the rotator cuff muscles; however, the relationship between SSN injury and rotator cuff enthesis has not been directly defined.

This study aimed to analyze the effect of SSN injury on rotator cuff enthesis. We hypothesized that surgically created SSN injury in rats would cause detrimental effects on rotator cuff enthesis, similar to the degeneration observed in rotator cuff muscles. Thus, SSN injury may be considered as an etiologic factor for the impaired healing after repair of a massive tear. In addition, nerve injury may cause different reactions between the entheseal zones that will provide information for a better understanding of the rotator cuff enthesis.

Materials and methods

Animal care complied with the international laws regarding the use of laboratory animals. The study included 30 adult male Sprague-Dawley rats weighing between 400 and 500 g. Twenty-four rats were prepared for the bilateral SSN transection surgery (nerve transection group), and 6 rats were used as the sham group with bilateral healthy tendons. The animals were anesthetized using an intraperitoneal injection of sodium thiopental (50 mg/kg Pentothal; Abbott Laboratories, Chicago, IL, USA). A preoperative dose of intramuscular cefazolin sodium (0.1 mg/kg Cefozin; Bilim ilaç, Turkey) was injected for infection prophylaxis. The animals were treated aseptically throughout the experiment. Rats were positioned prone on the operating table. A 2-cm dorsal incision was made over the bilateral scapula, followed by blunt dissection down to the trapezius muscle consecutively. SSN was identified by pulling the scapula posteriorly (Fig. 1). After it was exposed, a 5-mm-long segment of nerve was removed to prevent healing. The muscle and skin were then closed. The rats were allowed unrestricted cage activity. Wound dressings or casts were not used postoperatively. Paracetamol tablets were added to tap water to control pain. Animals were housed in wire-topped cages in groups of 2 and were exposed to a 12-hour light/12-hour dark cycle.

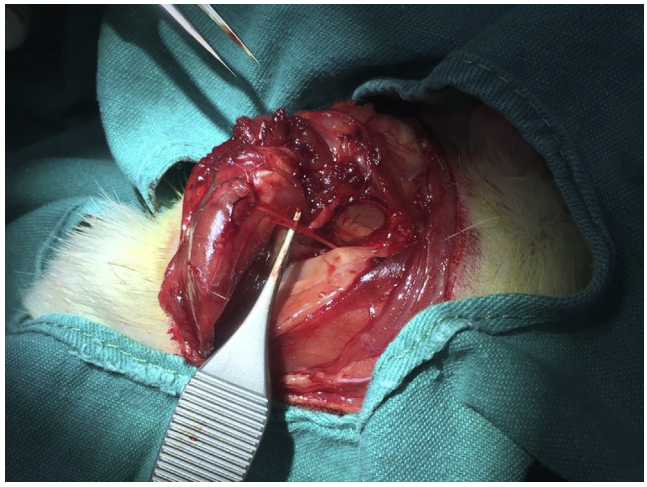

Figure 1 Suprascapular nerve and scapula in posterior view of left shoulder of a rat.

Six rats from the nerve transection group were sacrificed on the 1st, 4th, 8th, and 12th week after surgery. Six rats from the sham group were sacrificed for samples of bilateral healthy tendon attachment. Bilateral posterosuperior rotator cuff tendon attachment with muscle and a flake of bone were removed for histomorphometric analysis. After tendon removal, a lethal dose of pentobarbital (150 mg/kg) was used for sacrifice.

Histomorphometric analysis

Each tissue sample was fixed in 10% neutral-buffered formalin for 24 hours and decalcified in TBD-2 decalcifier solution (Shandon TBD-2 Decalcifier, Fisher Scientific, Loughborough, UK) at room temperature for 5 days. Following fixation and decalcification, the tissue blocks were dehydrated using graded ethanol (80%, 95%, and 100% sequentially), cleared in xylene, and embedded in paraffin wax. A Leica MR 2145 microtome (Leica Microsystems, Germany) was used to cut 5-μm-thick serial sections. The sections were stained with hematoxylin-eosin and Masson trichrome for evaluating enthesis histology and histomorphometry. For every sample, 5 serial sections were obtained, and a minimum of 5 adjacent fields focusing on the individual entheseal layer in each section were quantified (×40, ×100, ×200, ×400 magnification). All measurements were made with a semi-automatic image analysis system (The University of Texas Health Science Center at San Antonio image tool). Histomorphometric analysis was performed for each entheseal zone: tendon, uncalcified fibrocartilage, calcified fibrocartilage, and bone.[4,7] The number of fibroblasts, fibrochondrocytes, and osteoblasts in consecutive entheseal zones and collagen bundle diameter in the tendon zone were measured, recorded, and statistically compared among the nerve transection and sham groups at each time point.

Statistical analysis

Power calculation showed that 6 rats per group would result in a power level of 0.8 and the ability to detect a difference between groups using Student *t* test. Descriptive statistics are provided as

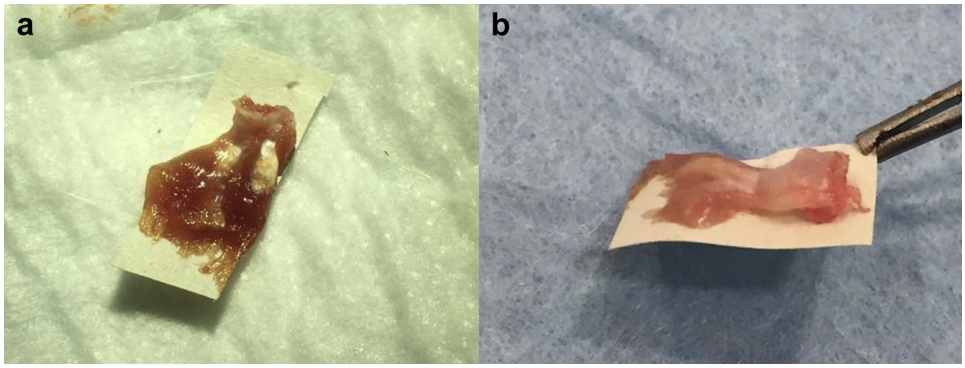

Figure 2 The nerve transection group tendons and entheseal areas showed attenuation and fading. (**a**) Healthy rotator cuff. (**b**) Nerve transection group rotator cuff at 12 weeks postoperatively.

mean and standard deviation. $P < .05$ was considered statistically significant. Statistical analyses were performed using NCSS software 2007 (Number Cruncher Statistical System, Kaysville, UT, USA).

Results

Gross observations

Progressive muscle atrophy was observed at each time point in the nerve transection group, indicating paralysis. The tendons and entheseal areas in this group also showed attenuation and fading at 12 weeks postoperation (Fig. 2).

Enthesis histomorphometry

Histomorphometric measurements are presented in Table I and Figure 3.

Tendon

There was a significant decrease in the number of fibroblasts and collagen bundle diameter in the nerve transection group in comparison with those in the sham group at all time points ($P < .001$) (Fig 4).

Uncalcified fibrocartilage and calcified fibrocartilage

Uncalcified fibrocartilage and calcified fibrocartilage zones showed similar response, and a significant reduction in fibrochondrocyte counts was observed after 8 weeks of nerve transection ($P < .001$).

Bone

Significant and consistent decrease in osteoblast counts was observed in the nerve transection group compared with that in the sham group at all time points ($P < .001$).

Discussion

The results of this study demonstrated that SSN injury can impair rotator cuff enthesis by reducing the cellularity at all entheseal zones and diminish the collagen bundle in the tendon zone. This negative effect was observed in all entheseal zones with varying severity, which progressed with time. Because cellularity and collagen bundles are major factors influencing enthesis vitality, impairment of rotator cuff resistance against repetitive microtrauma and deficiency of healing response after repair of a massive cuff tear can be expected.

Treatment of chronic massive RCTs is a difficult clinical concern. When performing surgical repair of chronic massive tears, the retear rate is quite high.[6] The risk of repair failure is correlated with patient age, tear size, and muscle degeneration, which indicates altered healing response.[16] Histopathologically, chronic massive RCTs show atrophy, fibrosis, and fatty infiltration in the muscle, reduced fibroblast cell counts in the tendon, and decreased density of the bone.[11,13,20,34,36] Degeneration and retraction of the rotator cuff caused by tears have been suggested to place excessive traction on the SSN and to promote compressive injury at the suprascapular notch.[2,8,35] Considering the present study results, SSN injury can affect the muscle, tendon, and bone simultaneously and lead to further alteration of the healing capacity in chronic massive RCTs.

Various anatomic variations have been reported to cause SSN entrapment such as morphology or ossification of the superior transverse scapular ligament,[21,23,33] shape of the suprascapular notch,[22] and close relationship with the subscapularis muscle.[3] In addition, the dynamic stretch and compression of SSN caused by kinematic impairment of the scapula should not be neglected. The incidence of anatomic and kinematic predispositions in patients with RCTs and concomitant SSN neuropathy merits further investigation, especially for the rerupture of repaired massive rotator cuff tear.

Denervation of a muscle causes loss of physiological loading and induces structural alterations in the enthesis.[25,30] Studies have shown that denervation and long-term unloading reduces the collagen alignment,[26,30,31] cellularity,[29] interdigitation structure complexity, and calcified fibrocartilaginous matrix in the enthesis.[30] In addition,

Table I Histomorphometric measurements of the parameters at each time point

	Fibroblasts, mean ± SD (*P* value)	Collagen bundle diameter, mean ± SD (*P* value)	Fibrochondrocytes in uncalcified fibrocartilage, mean ± SD (*P* value)	Fibrochondrocytes in calcified fibrocartilage, mean ± SD (*P* value)	Osteoblasts, mean ± SD (*P* value)
Control	45.77 ± 10.30	9.91 ± 3.63	40.84 ± 12.32	41.50 ± 15.95	60.80 ± 6.77
1 week	36.95 ± 11.09 (.001)	7.23 ± 2.47 (.001)	35.85 ± 15.75 (.096)	36.18 ± 13.43 (.091)	47.20 ± 3.99 (.001)
4 weeks	35.85 ± 11.45 (.001)	7.12 ± 2.66 (.001)	35.87 ± 14.17 (.096)	36.97 ± 12.66 (.136)	42.97 ± 4.21 (.001)
8 weeks	33.25 ± 13.01 (.001)	5.90 ± 1.80 (.001)	21.00 ± 5.97 (.001)	25.79 ± 7.21 (.001)	34.98 ± 5.86 (.001)
12 weeks	35.07 ± 11.76 (.001)	5.88 ± 1.64 (.001)	22.63 ± 6.62 (.001)	30.08 ± 6.59 (.001)	32.05 ± 3.16 (.001)

Student *t* test; $P < .01$.

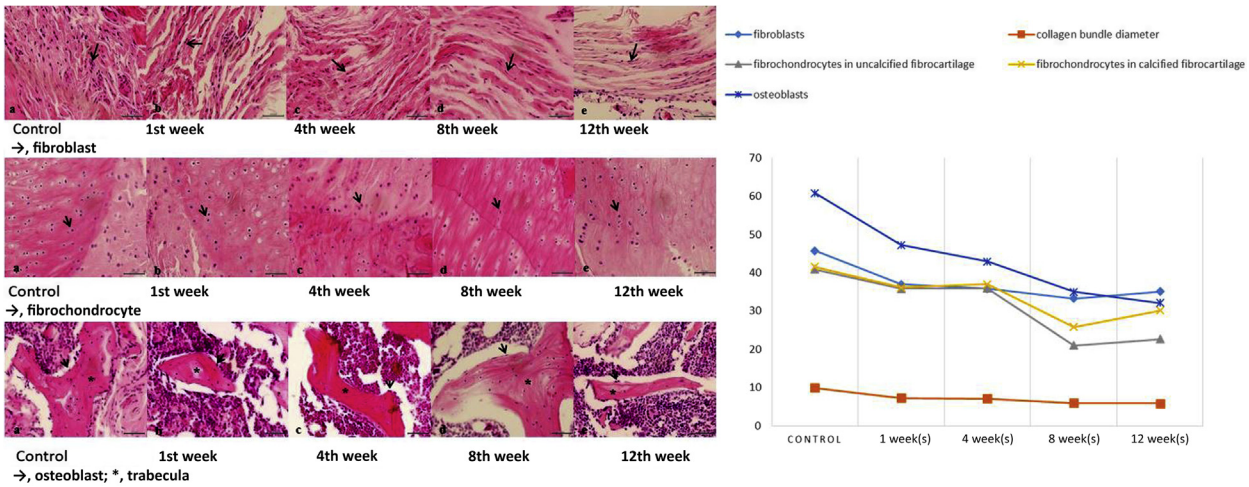

Figure 3 (*Left*) Histologic images of each entheseal zone: the first line showing tendon, the second line, fibrocartilage; and the third line, bone (hematoxylin and eosin ×100; scale bars 200 μm). (*Right*) Diagram showing the response of the rotator cuff enthesis to SSN injury according to cell types.

reduced mineral apposition rate and increased osteoclast surface were observed adjacent to the enthesis following denervation.[32] Our results are supported by similar findings reported in these studies, but rotator cuff enthesis may show different response after denervation in the different anatomic locations. The changes observed in the enthesis after denervation are dependent on the location-specific relationships between humoral, neural, and mechanical factors; however, the level of relationship necessary to maintain the structure of the enthesis remains unclear. The muscular changes following SSN denervation in the rat shoulder has been extensively investigated. Normal muscle mass is maintained by a balance between protein synthesis and degradation. Muscle atrophy results from an imbalance between these components.[24,28] The ubiquitin-proteasome pathway is a key protein degradation mechanism in skeletal muscle atrophy.[34,37] SSN denervation has been suggested to cause increased expression of muscle atrophy f box (*MAFbx*) and muscle ring finger protein 1 (*MuRF1*) genes, which leads to activation of the ubiquitin-proteasome pathway.[9,14] Similar mechanisms may be possible for the rotator cuff enthesis after SSN injury and should be studied for each zone.

In this study, SSN injury also led to different responses among the entheseal zones. Significant and consistent decrease in osteoblast and fibroblast counts was observed from the first week of SSN injury, whereas decrease in fibrochondrocyte counts was observed after 8 weeks. A possible explanation for this observation may be the difference in metabolism between the fibrochondrocyte and osteoblast/fibroblast cells. Because the entheseal fibrocartilage is relatively avascular, fibrochondrocytes function in a low-oxygen environment with low metabolic turnover; thus, they may become more resistant against external factors.[1] Moreover, the difference between the tendon-fibrocartilage zone and the calcified fibrocartilage-bone zone in their response to nerve injury indicates a weak relationship between these interfaces, and the starting point of RCTs may be these interfaces.

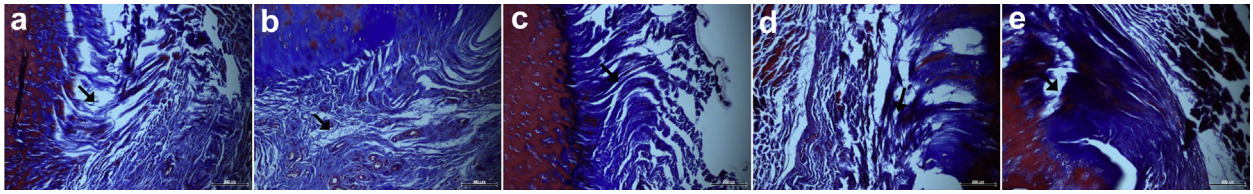

Figure 4 Histologic images of the collagen fibers (→): (**a**) healthy rotator cuff; (**b**) 1st week, (**c**) 4th week, (**d**) 8th week, and (**e**) 12th week postoperatively. (Masson trichrome × 100; scale bars 200 μm)

There are several limitations to the present study. The rat shoulder model does not exactly replicate the human counterpart. However, it has been well established that the rat shoulder has similar bony and soft tissue anatomy as humans.[27] Acute surgical transection of the SSN is different from a gradual traction or compression injury of the nerve and would have different effects on the enthesis. Therefore, observation of histologic changes in the enthesis in this animal model may not be generalized to the human condition. The parameters used in this study were commonly accepted for histologic analysis; however, numerous other factors could be involved in the response to SSN injury.[7] The small sample size reduced the statistical significance. Despite these shortcomings, to our knowledge, this is the first study focusing on the effect of SSN injury on rotator cuff enthesis. The method used in the measurements (histomorphometry) allowed a more objective analysis by providing numerical values.[7,10,18,19]

Conclusion

The present study identifies SSN injury as an underlying mechanism leading to compromise in the rotator cuff enthesis structure. SSN injury may be considered as an etiologic factor for the impaired healing after repair of a massive tear.

Disclaimer

The authors, their immediate families, and any research foundations with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article.

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