Pathophysiology of the Partial-Thickness Rotator Cuff Tear

Hiroaki Fukuda, M.D.
Department of Orthopaedic Surgery, Saiseikai Hiratsuka Hospital, Hiratsuka, Japan.

Degeneration of rotator cuff tendons is defined as structural alterations in the tendon proper and/or in the insertion (enthesis). Minor degenerative changes do not always cause functional impairment, but many begin to suffer from painful shoulder once various degrees of tendon tearing have developed. In this review, I shall discuss partial-thickness rotator cuff tears (PTRCTs), because these conditions and their characteristics occupy a most significant position within the spectrum of rotator cuff pathology. Hereafter, based on the integrity of the cuff tendon, I would refer subacromial bursitis or tendinitis, a "pretear" stage, to Grade I; partial-thickness cuff tears to Grade II; and full-thickness tears (FTRCTs) to Grade III. PTRCTs should have a tear involving at least one-quarter thickness of the supraspinatus tendon (SSp), and are classified into bursal-side tears (BTs), intratendinous tears (ITs), and joint-side tears (JTs).

Our study on the histology of the SSp tendon began 20 years ago using 227 fresh cadaveric SSp tendons[1,2]; 144 tendons were obtained from males and 83 were from females. The ages ranged from 0 to 86 with an average age of 57.5 years; 89% were from those aged over 50. In normal aging, the tendon loses regular wavy patterns, and becomes homogeneous and hyalinized. Arterioles are obliterated with thickened intima, and the enthesis is disrupted. There appears a close topographical relationship between the site of "disruption" in the insertion and the site of PTRCTs, i.e. in BTs, the "disruption" is found at the bursal side of the insertion. Likewise in both ITs and JTs. Incidence of FTRCT is 7.9% and that of PTRCT 14.5% among the said samples.
Then, with surgical specimens, we investigated on-going changes occurring in PTRCTs, utilizing en bloc histologic sections[3,4,5,6,7]. In BTs, the depth of tear varies and while the distal stumps are well vascularized, the proximal stumps are round, avascular and often retracted, and contain chondrocyte-like cells. In ITs, the tear runs in the middle of the tendon parallel to the tendon fibers. The torn surfaces are smooth and lined by flat cells. Both proximal and distal ends often show fissures from tears. In JTs, the extent of the tear also varies. Tearing starts from the synovial reflexion and vicinity, and

runs parallel to the tidemark or connects with intratendinous tearing at right angle. Vascularity around tears is preserved.

Among three subtypes, concomitant subacromial bursitis and disruptions of the enthesis with granulation tissue are common. In our series of 35 en bloc histologic sections from the surgical specimens of Grade II lesions, no active repair was observed in any portion examined. Rather, there were many instances of impending FTRCTs with only narrow soft tissue connection.

We also found that histologic and biomechanical properties were different between the bursal-side and joint-side tendon layers of the SSp tendon[8]. The bursal layers, composed primarily of tendon bundles, may elongate to a tensile load and resist rupturing, whereas the joint-side layers, a complex of tendons, ligaments and joint capsule, are unstretchable and tear easily. These findings suggest that intratendinous lamination is caused by differential shear stress within the SSp tendon.

Our next project was an immunohistochemical analysis of interleukin-1β, cathepsin D, and matrix metalloproteinase-1, using the osteochondral destruction of the enthesis[9]. Sixteen SSp insertions with portion of the greater tuberosity including 8 Grade II and 8 Grade III lesions were obtained during surgery. Six fresh cadaveric SSp tendons without tears evident on gross examination served as control. Strong immuno-reactivity was found in all 16 torn SSp insertions but not in the 6 insertions of apparently intact tendons. Macrophages and multinucleated giant cells, showing reactions to these mediators, were found at the interface between the osteochondral margin of the enthesis and the granulation tissue, suggesting that these are involved in osteochondral destruction. Our conclusion is that this granulation tissue contributes to the development of rotator cuff tears by weakening the insertion, in addition to subacromial impingement.

To determine the healing potential and healing process of torn SSp tendons, we employed in situ hybridization to localize cells containing α1 typeI procollagen mRNA[10,11]. Biopsy specimens of torn SSp tendons from 13 patients with Grade II lesions and 19 patients with Grade III lesions were obtained during surgery. Four SSp tendons that were normal on macroscopic evaluation were obtained as control. In 14 of 19 Grade III tears, staining was clearly detected in the cells at the proximal stump of the tear. At a higher magnification, the label is identified in the cytoplasm of the tenocytes. The labeled cells at the proximal stumps of Grade II and III tears are significantly more

abundant than in the normal tendons. In Grade III lesions, the labeled cells in the specimens that were obtained less than 4 months after the trauma were significantly more abundant than in those obtained 4 months or longer after the injury. However, these labeled cells were observed at the torn portion even in long-standing Grade II lesions. Regarding localization of signal positive cells in Grade II lesions, intratendinous layers showed more labeled cells than the bursal-side or joint-side layers. Grade II lesions and concomitant intratendinous extensions may progress to rupture after the initial injury.

The torn SSp tendon apparently has an intrinsic healing capacity in the intermediate and late phases of tendon healing. An arthrographic follow-up on 40 JTs for 2 years revealed that 10% of them disappeared, another 10% decreased in size, but the remaining 80% had enlarged tears or progressed to FTRCTs[12].

Except on rare occasions, the spontaneous healing of the clinical PTRCT appears to be unlikely. Untoward factors include widening of the tear caused by muscular contraction and weight of the arm, hypovascularity, shear within the tendon, and subacromial impingement.

We regard most PTRCTs precursors to FTRCTs through vicious circle, as Neer indicated[13,14]. Factors related to the development of the rotator cuff tear are classified as intrinsic, extrinsic or traumatic. Intrinsic tendinopathy and/or enthesopathy due to changes in cuff vascularity or other metabolic alterations associated with aging, may lead to degenerative tears. Extrinsic subacromial impingement due to supraspinatus outlet narrowing by coracoacromial arch abnormalities can cause PTRCTs through cuff irritation. This impingement mechanism probably plays a major aggravating role in many PTRCTs. An excessive tensile load of the cuff, due to either a single traumatic injury or repetitive microtraumata may also cause PTRCTs. Our study with 103 PTRCTs, all surgically verified, disclosed that trauma was related differently to 3 subtypes of Grade II lesions; 18.2% in 44 BTs, 84.2% in 19 ITs, and 72.2% in 36 JTs. Four cases of combined BT and JT did not elicit history of obvious trauma. It is clear that more than one etiologic factor is involved, in addition to normal aging.

We conclude that genesis of PTRCT is multifactorial and in each subtype, the main cause is different, i.e. in BTs, subacromial impingement; in ITs, trauma with differential shear stress between the superficial and deep layers of the tendon; and in JTs, trauma on

degenerated tendons.

References

1. Yamanaka K, Fukuda H Pathological studies of the supraspinatus tendon with reference to incomplete thickness tear. In: Takagishi N (ed) The shoulder, Professional Postgraduate Service, Tokyo, 220-224, 1987.
2. Yamanaka K, Fukuda H Ageing process of the supraspinatus tendon with reference to rotator cuff tears. In: Watson MS (ed) Surgical disorders of the shoulder, Churchill Livingstone, Edinburgh. 247-258, 1991.
3. Fukuda H, Mikasa M, Yamanaka K Incomplete thickness rotator cuff tears diagnosed by subacromial bursography. Clin Orthop 223: 51-58, 1987.
4. Fukuda H, Hamada K, Yamanaka K Pathology and pathogenesis of bursal-side rotator cuff tears viewed from en bloc histologic sections. Clin Orthop 254: 75-80, 1990.
5. Fukuda H, Hamada K, Nakajima T, Tomonaga A Pathology and pathogenesis of the intratendinous tearing of the rotator cuff viewed from en bloc histologic sections. Clin Orthop 304: 60-67, 1994.
6. Fukuda H, Hamada K, Nakajima T, Yamada N, Tomonaga A, Gotoh M Partial-thickness tears of the rotator cuff:A clinicopathological review based on 66 surgically
verified cases. Int Orthop 20: 257-265, 1996.
7. Fukuda H Partial-thickness rotator cuff tears: A modern view on Codman's classic. J Shoulder Elbow Surg 9: 163-168, 2000.
8. 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 3: 79-87, 1994
9. Gotoh M, Hamada K, Yamakawa H, Tomonaga A, Inoue A, Fukuda H Significance of granulation tissue in torn supraspinatus insertions: An immunohistochemical study with antibodies against interleukin-1β, cathepsin D, and matrix metalloprotease-1. J Orthop Res 15: 33-39, 1997.
10. Hamada K, Okawara Y, Fryer N, Tomonaga A, Fukuda H Localization of mRNA of procollagen α1 TypeI in torn supraspinatus tendons: In situ hybridization using digoxigenin labeled oligonucleotide probe. Clin Orthop 304: 18-21, 1994.

11. Hamada K. Tomonaga A, Gotoh M, Yamakawa H, Fukuda H Intrinsic healing capacity and tearing process of torn supraspinatus tendons: In situ hybridization study of α1 (I) procollagen mRNA. J Orthop Res 15: 24-32, 1997.
12. Yamanaka K, Matsumoto T The joint side tear of the rotator cuff.: a follow-up study by arthrography. Clin Orthop 304: 68-73, 1994.
13. Neer CS Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 67A: 41-50, 1972.
14. Neer CS Impingement lesions. Clin Orthop 173: 70-77, 1983.