SURGICAL REPAIR OF ROTATOR CUFF RUPTURES

THE IMPORTANCE OF THE SUBACROMIAL BURSA

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We examined biopsy specimens obtained during surgery on 115 patients with complete rotator cuff rupture. The vascularised connective tissue covering the area of rupture and the proliferating cells in the fragmented tendons reflected more of the features of repair than of degeneration and necrosis. The main source of this fibrovascular tissue was the wall of the subacromial bursa.

These features clearly indicated a vigorous reparative response which might play an important role in tendon reconstitution and remodelling. We therefore suggest that extensive debridement along with subtotal bursectomy, commonly practised during surgical repair of rotator cuff rupture, should be avoided. Although strong suture margins are essential for good operative results, debridement should be judicious and preserve as much as possible of the bursa and the associated fibrovascular tissue.

The principal objective in the surgical repair of complete rotator cuff rupture is to restore continuity between the tendon and its insertion into bone. Before placing the sutures, however, it is usual to perform an extensive debridement, often including subtotal subacromial bursectomy. This allows good visualisation of the operative field, necrotic tissue is removed, the edges are freshened to improve blood supply and to enable the suture margins to withstand tension.

In the course of a study of structural changes in the supraspinatus tendon in cases of complete rotator cuff rupture, we found that the vascularised connective tissue covering the area of rupture and the associated proliferation of cells in fragmented tendons reflected more the features of repair than of degeneration and necrosis. The main source of the fibrovascular tissue was the wall of the subacromial bursa. In the light of those findings, the present study was undertaken to determine the contribution of the subacromial bursa towards repair of the rotator cuff, and to assess the rationale for debridement that often includes extensive portions of the bursa.

MATERIALS AND METHODS

We examined specimens obtained during surgery on 115 patients with complete rotator cuff ruptures. Almost all of the specimens were from the supraspinatus tendon. Each group of specimens consisted of two to four fragments measuring 3 to 12 mm in diameter. The mean age of the patients was 55 years, and there was a preponderance of men. A history of insidious onset was more common than one of trauma before the symptoms. At least three months of conservative treatment had preceded the operation.

The specimens were fixed in 10% neutral buffered formalin and embedded in paraffin. Sections were stained with haematoxylin and eosin for standard histology, and by Masson's trichrome method for the identification of collagen. Sections of intact supraspinatus tendons from 32 cadavers matched for age were used as controls.

RESULTS

The histological examination of the supraspinatus tendon of cadavers revealed that the wall of the subacromial bursa was continuous with the connective tissue comprising the epitenon with no plane of cleavage between them. The tissue between the synovial lining of the bursa and the collagenous fascicles of the tendon contained a small number of blood vessels.

In specimens from complete tears, there was a marked increase in vascular channels; they derived mainly from the bursal wall. The proliferation of synovial

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0301-620X/91/3112 \$2.00
J Bone Joint Surg [Br] 1991; 73-B:399-401.

cells, however, was inconstant, and when present, the degree varied considerably. Many of the vascular channels were lined by a single layer of endothelial cells and sprouting was conspicuous, but there were thick-walled vessels as well. The cells in between the vessels resembled fibroblasts. This proliferating fibrovascular tissue tended to encroach upon the area of rupture which contained randomly dispersed fragments of tendon and irregular margins of torn fascicles (Fig. 1). Many of the tendon fragments close to the site of the tear were markedly cellular and often contained vascular channels, the features of granulation tissue. A similar proliferation of cells could also be seen at the surface of the torn fascicles resembling a repair cap (Fig. 2). In the same area, however, some fragments were relatively or completely acellular, appearing as amorphous eosinophilic tissue probably in the process of absorption (Fig. 1).

DISCUSSION

We conclude from our study that a complete rupture of the rotator cuff always elicits fibrovascular proliferation in the area of the tear. It has been reported that the extent of this form of granulation tissue in response to rupture decreases with advancing age (Refior, Krödel and Melzer 1987). This was not apparent in our material.

Codman (1934) described the encroaching fibrovascular tissue upon the area of rupture as a "falciform edge of new tissue", and advised its removal because he felt it to be too friable to hold the stitches. During surgery, it is not difficult to delineate the presence of this granulation tissue which seems to extend from the subacromial bursa. Since Codman, debridement of this tissue including the so-called necrotic tendon edges and the bursa has been

recommended by a number of authors including DePalma (1950), Hawkins (1984, 1990), Nasca (1984) and Neviaser and Neviaser (1984), mainly in order to obtain a good definition of the edges to be repaired.

What became evident in our study, however, was that the existing practice of debridement might eliminate not only tissues that already show a great deal of repair activity, but also the major source of cells and blood vessels that are necessary for the repair. The majority of the randomly dispersed frayed edges were contiguous with highly cellular fibrovascular tissue from the bursal wall. They contrasted markedly with the poorly cellular or acellular fragments at the margins of the tear (see Fig. 1). The former clearly represented repairing tendinous tissue while the latter was the necrotic element that appeared destined to be absorbed in the course of time. Furthermore, the aggregation of cells at the edge of the rupture was equivalent to the 'capping' that has been described in experimental conditions indicative of intrinsic healing (Manske and Lesker 1984).

Bateman (1978) has recommended that the bursa be retracted to facilitate surgery but that it should be preserved. Similar advice against the excision of the bursa was made by Hanamura, Yasuhara and Maeda (1987). Watson (1985) states that it is not necessary to resect the tendon edge back to 'normal tendon'. He also suggests that the bursa is not removed but dissected and sutured separately after cuff repair. Codman (1934), although he recommended the removal of the 'falciform edge', added that he might "be wrong in recommending the removal of this new tissue with which nature is attempting to repair the damage".

Our study supports the concept that unless tissue is grossly necrotic, trimming of the margins should be kept

Fig. 1

Bursal tissue (B) covered by a lining of synovial cells shows marked vascular proliferation. The tissue encroaches upon the area of rupture and comes close to the tendinous fragments (arrow). Hematoxylin and eosin (magnification $\times 150$).

Fig. 2

A tendon fragment in the area of rupture shows accumulation of cells at the edge resembling a repair cap (arrow). Hematoxylin and eosin (magnification $\times 200$).

to the minimum. Should there be a stump of vascularised connective tissue attached to the greater tuberosity, it should not be excised. The torn tendon margins should be held in close approximation with unabsorbable sutures anchored into the bone and covered by the bursal tissue. Partial removal of the parietal wall of the bursa, however, is unavoidable during acromioplasty or in the course of segmental resection of the coracoacromial ligament.

In the context of flexor tendon healing in the hand, Peacock (1964, 1965) identified three phases that begin with the migration of peripheral vessels and cells into the wound site, followed by collagen production that unites the tendon ends. In the final phase, the remodelling of the tendon restores the smooth surface that is necessary for gliding. In this scheme, any contribution to repair by cells originating intrinsically from the tendon was not envisaged. Since then, participation of intrinsic cells in the repair of flexor tendons has been convincingly

demonstrated, particularly in 'in vitro' conditions (Gelberman et al 1983; Mass and Tuel 1990). In our study, we found both extrinsic and intrinsic forms of repair. However, the marked fibrovascular proliferation in the bursal wall, and the relative paucity of a similar reaction in the tendon proper, indicated that for nutrition as well as for procurement of new cells, the healing tendon was largely dependent upon the overlying bursa. Although phagocytic activity was not readily discernible histologically, it is conceivable that repairing cells could provide collagenolytic enzymes to digest necrotic fragments. Thus, in the overall process of repair of rotator cuff rupture, it seems that the mechanism of extrinsic healing supersedes the intrinsic form.

This study was supported by a grant from the Physicians' Services Incorporated Foundation.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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