A Histological Observation on the Flexor Tendon Healing within Intact Sheath

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Summary: In this study we allowed the sutured chicken flexor tendons to glide back into the uninjured sheaths in order to keep the healing process of flexor tendon from being affected by the healing of surrounding wounded tissues. By observing 12 chickens, 72 digits, with light microscope and transmission electron microscope, it was found that the visceral and parietal synovium of the sheath were the regions with earliest and most active cell proliferation and the major source of repairing cells during the healing process of the flexor tendon. Tendon cells had the ability of intrinsic healing, but delayed as compared to synovium cells. Adhesion between intact parietal synovium and healing tendon and its surrounding tissue could not be avoided.

Key words: flexor tendon, synovium, adhesion

The purpose of this study was to observe the healing process of flexor tendon within uninjured sheath and the source of the repairing cells.

MATERIALS AND METHODS

12 Leghorn chickens hatched in the same period were used. The skin incisions were separately made at the metacarpophalangeal joints of the three long digits of both feet, then the profundus flexor tendons were exposed. By fully flexing the interphalangeal joints and pulling proximally on the profundus tendon, the underlying tendon was partially cut, then using 7-0 nylon, a Kessler repair stitch was accomplished, the tendon was completely divided, and the suture was tied (fig. 1A). At the proximal site, 8 mm from the sutured point of tendon, the tendon was cut off. Then, by passively extending the digit, the sutured ends of the tendon were drawn back into the intact part of the sheath (fig. 1B). The skin and sheath were closed with 3-0 nylon. The chickens were sacrificed at day 3, week 1, 2, 3 and 4 after surgery. Two chickens, 6 digits, were available for study at each observation period. Two chickens serving as the control group were left uninjured. In all,72 digits were observed with light microscope and transmission electron microscope (TEM).

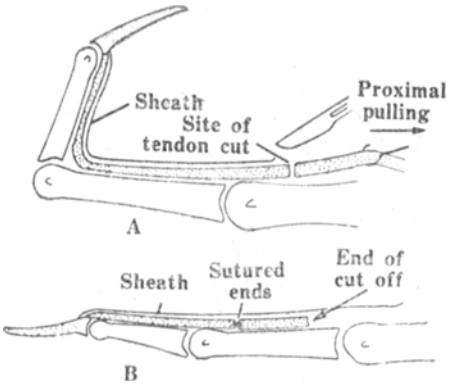

Fig.1. Diagram of the operation.

RESULTS

3 days after operation, light microscope observation showed that the cells of the visceral synovium at the site of the repaired tendon presented proliferation with the layers of the cells increased and linked up with the repaired ends of the tendon. The gap between the ends, being infiltrated with leukocytes, was clearly seen. The parietal synovium opposite to the site of the repaired tendon was swollen, the normal fusiform cells in it became larger, and elliptic or round in shape. The synovial cavity was discernible (fig. 2). TEM observation showed that the tenocytes at the sutured ends of the tendon underwent little change. However, the cells in the parietal synovium presented active proliferation, rough endoplasmic reticulum in the cells became increased and hypertrophic, chromatin increased in the nucleus and aggregated towards nuclear membrane (fig. 3).

At 1 week postoperatively, light microscope observation showed that proliferating cells of visceral synovium had migrated into the repaired ends of the tendon. The proliferating parietal synovium was protruding towards the gap between the sutured ends of the tendon narrowing the cavity of the sheath. The closer the visceral and parietal synovium to the site of the repaired tendon, the more obvious was their proliferation (fig. 4). TEM observation showed that the tenocytes at the repaired ends or the tendon still remained unchanged. The cells with stellate processes in the parietal synovium had rich rough endoplasmic reticulum. The irregular nucleus of the cells had large nucleoli. There were many reticular fibers. By this period, the parietal synovium tissue had developed to granulation tissue consisting of fibroblasts, hemangioendothelioblasts and reticular fibers. It was difficult to recognize synovium cells.

At 2 weeks postoperatively, light microscope observation showed that proliferating visceral and parietal synovium narrowed the synovial cavity, and adhesion occurred at some sites. The granulation tissues originating from the synovium were invading the repaired ends of the tendon and growing in the form of buds. Cells, reticulum fibers and collagenous fibers in the new tissue increased in number and thickened. Quiescent tenocytes in the repaired ends transformed into juvenile fibroblasts (fig. 5). TEM observation revealed that larger fibroblasts with stellate processes appeared in the repaired ends of the tendon. There were increased chromatin and rich rough endoplasmic reticulum in the cells. Granulation tissues were seen in all specimens from parietal synovium.

At 3 to 4 weeks following operation, light microscope observation showed that the range of adhesion between parietal and visceral synovium extended at the repaired site of the tendon. The granulation tissue originating from the synovium continued growing into the repaired ends of the tendon, tending to collagenation. TEM observation showed that the tenocytes in the repaired ends of the tendon were actively proliferating and gradually matured. These cells and collagen bundles were orientated in the direction of the long axis of the tendon. Most

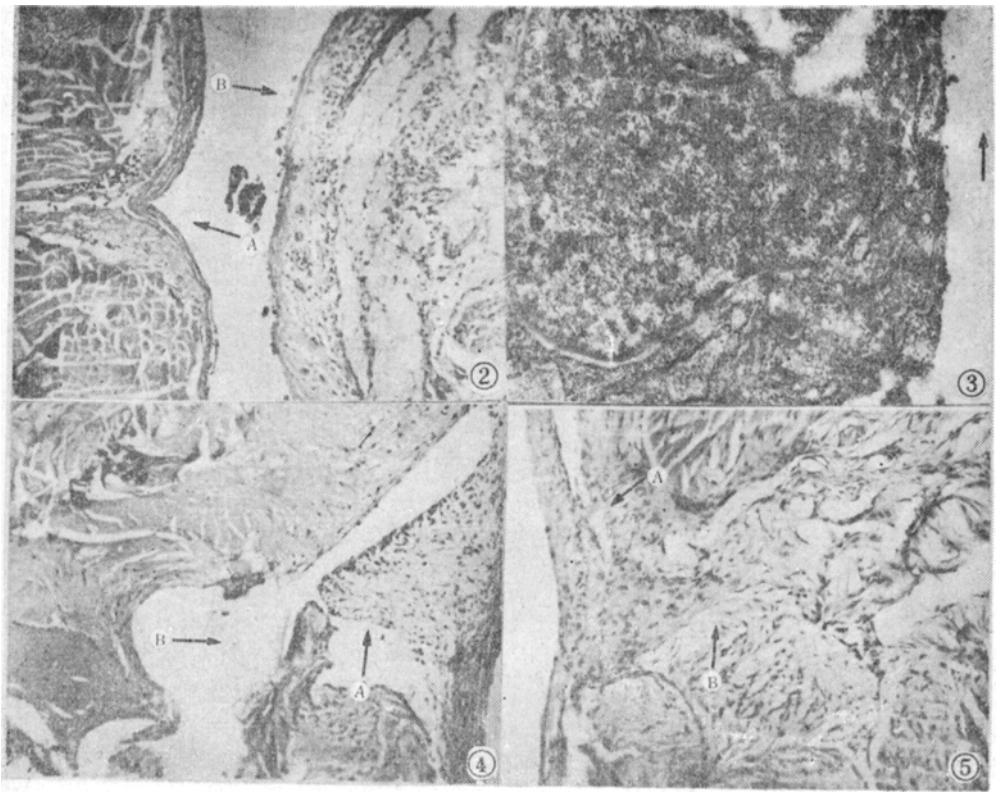

Fig.2. 3 days after operation: Proliferating cells of visceral synovium link up the repaired ends of tendon (arrow A). The parietal synovium is swollen (arrow B). 100×.

Fig.3. 3 days after operation: Proliferating cells of the parietal synovium. The arrow points to the synovium cavity. TEM 8000×.

Fig.4. 4 weeks after operation: Proliferating parietal synovium protrudes towards the sutured ends (arrow A). Suture (arrow B). 100×.

Fig.5. 2 weeks after operation: Adhesion occurs between the visceral and parietal synovium (arrow A). Granulation tissue grows in form of buds into the sutured ends (arrow B). 100×.

specimens from the parietal synovium presented scar tissue. Synovium cells could hardly be recognized.

DISCUSSION

The cells which participate in tendon healing may originate from the tendon, the visceral synovium, parietal synovium (synovial sheath), tissue surrounding the sheath, or be a combination of these. Experimental studies by Potenza[1] and Lindsay[2] suggested that the fibroblasts participating in tendon healing come from the sheath or surrounding tissues, and the tendon has little intrinsic healing ability. Lundborg[3] placed isolated segments of rabbit flexor tendon in the synovial pouch of the knee joint; 3 weeks later, the tendon segments were healing without adhesion, and the flexor tendon was found to have the ability of intrinsic healing. But other researchers[4]

believed that the results were due to the fact that synovial cells from the knee joint "seeded" onto non-viable tendon as a source of repairing cells. By in vitro experiments using tendon segments maintained in tissue culture, Manske et al[5] discovered that the flexor tendon had intrinsic healing capacity and that the repairing cells originating from either of the visceral synovium or endotenon proliferated and clonized.

Our experimental results showed that the earliest repairing reaction to the wound of flexor tendon occurred in visceral and parietal synovium, then in the tenocytes in the tendon and the connective tissues surrounding the tendon, with the parietal synovium invading. In histology, both visceral and parietal synovium belong to mesothelium tissue with remarkable regenerative ability, and there are rich vascular networks under the layers of the synovium, with rich blood supply providing synovial fluid to nourish the tendon[6]. We believe that the factors mentioned above are the basis for active participation of the parietal synovium in the repair of wound. Furthermore, we believe that the early change in the parietal synovium participating in the repair of wounds was the reaction of the synovium to inflammation, that is, injured flexor tendon released some inflammatory factors affecting the uninjured parietal synovium. The present study showed that in the initial period, the site of repaired tendon appeared swollen and infiltrated with leukocytes, the parietal synovium opposite protruded in the form of buds to the injured ends of the tendon, narrowing the synovial cavity and causing adhesion. In previous studies, the sites of sutured flexor tendons were just opposite to the sites of lacerated skin and sheath, so the healing of repaired flexor tendon was blended with the healing of injured peritendon tissue. It was difficult to distinguish the share of synovium, especially the parietal synovium in healing or in preventing adhesion. In this study, we let the site of repaired tendon slide back into uninjured sheath to avoid the effects of wounded peritendon tissues healing on the flexor tendon healing, but adhesion still occurred. Therefore, it may be suggested that the intact sheath can not avoid injured flexor tendon from getting adhered.

This study was undertaken to determine the origin of the cells participating in the healing process by TEM. Hitoshi observed the surface of repaired tendon during the healing process by scanning electron microscope [7]. He found that the injured tendon was covered with lining cells from parietal synovium after two weeks. Our observation showed that visceral synovium covered the ends of the repaired tendon, and the parietal synovium encircling the ends presented proliferation at 3 days after operation. There are 2 types of cells in synovium with type A containing little and type B containing rich rough endoplasmic reticulum. Under normal conditions type A cells predominate in the synovium, while under pathological conditions type B cells become significantly increased. We could hardly identify type B cells in control uninjured synovium, but many cells with rough endoplasmic reticulum appeared in parietal synovium at 3 days after operation. These cells together with those in visceral synovium were growing towards the sutured ends of tendon in the form of buds, but no obvious proliferation of tenocytes was seen in the repaired ends of tendon until 2 weeks after operation. Proliferating synovial tissues in the form of granulation tissues participated in the healing process. By this time, the primary histological characteristics of the synovium tissues had disappeared. From this study it was suggested that the visceral and parietal

synovium of the flexor tendon were the regions with earliest and most active hypercellularity. It may be inferred that the synovium cells might be a kind of cells with potential ability to differentiate and might be a major source of repairing cells. This is further confirmed by Garner's recent observation that synovium cells can produce collagen by monoclonal technique[8].

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