INTRODUCTION:

The characteristic pathological finding in carpal tunnel syndrome (CTS) is non-inflammatory fibrosis of the synovium. How this fibrosis might affect tendon function, if at all, is unknown.

The subsynovial connective tissue (SSCT) lies between the flexor tendons and the visceral synovium (VS) of the tenosynovial bursae. Fibrosis of the SSCT may well affect its gliding characteristics (Figure 1).

To investigate this possibility, the relative motion of the middle finger flexor digitorum superficialis (FDS III) tendon and VS was observed during finger flexion in patients undergoing carpal tunnel surgery (CTR), and in an in vitro cadaver model with cadavers with and without a CTS history.

METHODS:

We monitored the gliding motion of the FDS III tendon and SSCT in 8 patients with carpal tunnel syndrome during their release and in 8 cadavers with an antemortem history of CTS and compared these with simulated active flexion in 8 cadaver controls.

After the carpal flexor retinaculum was transected during surgery or during dissection, a small window (5x5mm) was made in the visceral synovium and subsynovial connective tissue to expose the middle finger FDS tendon. The middle superficial flexor tendon, the visceral synovium and the flexor retinaculum were marked with a marker pen (Figure 2). The FDS tendons were actively pulled proximally by one investigator till maximum flexion of the fingers either for the individual cadaver specimen or by actively flexing the digits by the patients during surgery. The motion of these three markers was detected by anteroposterior recording with a digital camcorder. The data was digitized with the use of Analyze™ Software (Biomedical Imaging Resource, Mayo Clinic, Rochester, MN).

RESULTS

In the CTS patients and cadavers with an antemortem history of CTS, we found that the displacement of the visceral synovial layer and surrounding soft tissue is either increased or decreased as compared to the controls. In 2 patients and 1 cadaver patient the SSCT moved en bloc with the tendon and in 6 patients and 7 cadaver patients at a certain level in the SSCT there was damage to the SSCT which formed a gap between adjacent layers making the visceral synovial layer not move at all. Whereas, in all controls the SSCT moved smoothly and separately from the tendon (Figure 3.).

While only moving the middle finger (differential finger movement) there was a mean slope of 0.14 ± 0.14 in the 6 patients and in the 7 cadaver patients this was 0.08 ± 0.04. In the 8 controls the slope was 0.12 ± 0.06.

CONCLUSION:

These findings suggest that in patients with CTS the synovial fibrosis has altered the gliding characteristics of the SSCT. The alterations in the gliding characteristics of the SSCT may affect the ability of the tendons in the carpal tunnel to glide independently from each other, or from the nearby median nerve, and might break as a product of this limitation, leaving a gap between adjacent layers as the result. More patients should be included to correlate the different stages of SSCT characteristics with the severity of the CTS. The changes in the SSCT could potentially aggravate or even cause median nerve neuropathy.

Clinical Relevance:

By identifying possible pathways in the ethiology of CTS, different therapies may be developed to prevent CTS in the future.