EFFECTS OF A FASCIITS ON THE INNERVATION OF THE RAT THORACOLUMBAR FASCIA

U. Hoheisel and S. Mense

Chair of Neurophysiology, Center for Biomedicine and Medical Technology, Ruprecht-Karls-University Heidelberg, Medical Faculty Mannheim, Ludolf-Krehl-Str. 13-17, D-68167 Mannheim, Germany
e-mails: ulrich.hoheisel@medma.uni-heidelberg.de, Tel. x49-621 383 9637
siegfried.mense@medma.uni-heidelberg.de, Tel. x49-621 383 9713

BACKGROUND Noxious stimulation of fasciae – including the thoracolumbar fascia (TLF) g– evokes pain. Recent publications have described a dense innervation of the TLF. Here, the posterior lamina of the TLF was studied, which covers the multifidus muscle. The aim was to see if the innervation changes when the fascia is inflamed.

METHODS The experiments were approved by the local ethics authority and carried out in accordance with the German law on the protection of animals. SD rats received an intrafascial injection of complete Freund's adjuvant (CFA). The immunohistochemical evaluation of the tissue was carried out 12 days after the CFA injection. In the quantitative evaluation, the fiber length of fibers of passage and free nerve endings were measured. The following markers were used: protein gene product 9.5 (PGP 9.5) as a universal marker for all nervous structures, tyrosine hydroxylase (TH-) for sympathetic nerve fibers, calcitonin gene-related peptide (CGRP) and substance P (SP) for sensory peptidergic nerve fibers.

RESULTS The inflammation-induced changes in innervation density did not occur in all fascia layers alike. In the middle layer – which consists of massive collagen fiber bundles - the fiber density was close to zero for all fibers. In inflamed fascia, CGRP-positive fibers showed a significant increase in the inner layer. In intact TLF, the nociceptive SP-containing nerve fibers were present exclusively in the outer layer. In inflamed fascia, SP-positive structures were also found in the inner layer. In inflamed TLF, there was a significant decrease in the mean length of sympathetic fibers.

CONCLUSIONS The increase in length of the nociceptive SP-positive fibers may explain the pain of patients with a pathological alteration of the fascia. However, the large middle layer of the TLF remained devoid of SP fibers, also in inflamed fascia. Great portions of the CGRP fibers are likewise nociceptive. The increase of these fibers in the inflamed fascia could contribute to the fasciitis pain. An unexpected finding was the significant decrease in postganglionic sympathetic fibers. Many of these fibers outlined blood vessels in the outer layer close to the subcutaneous tissue and probably were vasoconstrictors. The decrease in sympathetic fibers may be associated with a vasodilation in the outer layer and subcutaneous tissue.