CRITICALLY APPRAISED PAPER (CAP)

Evidence / Title of article

" Sensory findings after stimulation of the thoracolumbar fascia with hypertonic saline suggest its contribution to low back pain"

Schilder A et al (2014) PAIN 155:222-231

Introduction/ background

Muscle and fascia have been thought to have a poor role as sources of Low back pain. Evidence exists of a relationship between changes in muscle and fascia and LBP. Fascia is innervated by nociceptive free nerve ending and the lumbar dorsal horn neurons receive nociceptive input from the fascia. Connective tissue seems to be in general more sensitive than muscle.
The aim of this study is to investigate the contribution of Thoracolumbar fascia, the erector spine muscle and the sub-cutis to Low back pain. An injection of hypertonic saline into the thoracolumbar fascia has been hypothesized to cause the most intense pain, the largest radiation and the most sensitization to pressure.

Methods

Inclusion criteria:

- no history of low back pain
- German language

Exclusion criteria:

- medication or surgery to abdomen, legs or back

After signing a written consent form, all the participants attended 3 study sessions (5 days between each session). Subject lay on a bench face down (to minimize the back muscle contraction). within each session, the protocol was performed twice, with hypertonic saline on one side and isotonic saline on the contralateral side. The participants were informed that they would receive 2 injections per session.

Design

Randomized controlled trial

Setting

Not mentioned

Subjects Population (P)

Twelve healthy volunteers (6 female, 6 male; mean age: 24,0±1.5 years)

Intervention (I)

Bolus injections of hypertonic saline (5.8%) were made into the posterior layer of the thoracolumbar fascia, the erector spine muscle and the overlying sub-cutis at lumbar level (L3/4), about 4 cm lateral to the spinous processes. The position of the injection needle was guided by ultrasound (Acuson X150; Siemens). The solution was administered using a 1-mL syringe and a 27G cannula.
All participants were blinded with regard to the injection solution and tissue.

Comparison (C)

Isotonic saline (0.9%) injections were made following the procedure as for the intervention group.

Outcome(s)(O)

Before and after the injections these outcomes were determined:

- pain intensity on a numeric rating scale (0-100) at 10-second intervals for the first 2', and then at 30-second interval for the following 23 minutes.
- pain quality: a list of verbal descriptors was submitted (Pain Perception Scale comprising 14 affective and 10 sensory items). Descriptors were rated on a 4-levle ordinal scale.
- pain distribution on a standard human body schema
- PPT : pressure pain threshold measured with a pressure algometer (Wagner instruments, Greenwich, CT, USA). PPT was measured at 4 differentt locations, including the point of injection(central) and 3 other areas (5cm cranial, caudal and lateral to the injection site) At the beginning and then at 25' after the injection and at 50' after saline injection.

Dates Analysis

Statistical analysis was performed using SigmaPlot software, version 12.0 (Systat Software, Inc, Chicago, IL, USA). Significant differences (at P-values <0.05) were determined by 2-way repeated-measures analysis of variance (ANOVA; factors: tissue and saline concentration) followed by Holm-Sidak post hoc test correcting for multilevel comparison. The data of pain intensity ratings and PPT were transformed into decadic logarithms to achieve normal distribution. For analysis of pain qualities, the statistical significance was determined separately for every single descriptor item and tissue. The descriptors were grouped into sensory and affective summary scores and then subjected to 2-way ANOVA. The areas of the pain drawing were digitized and transformed into color-coded image using Adobe Photoshop CS4. Body areas with high or low pain were illustrated in dark red or light yellow and areas without pain appeared white.

Results

Fascia depth location had an upper margin of 6.2$\pm$1.9 mm and lower margin of 8.3$\pm$2.1 mm; approximate fascia thickness was 2.1$\pm$0.5 mm. The injection liquid showed a maximal detectable spread of 19.5$\pm$2.6 mm in length and a maximal detectable spread of 2.3$\pm$0.6mm in height.
The hypertonic saline injection induced higher and longer-lasting pain ratings in all tissue than isotonic injection. Hypertonic saline injection into the fascia evoked longer pain duration and higher pain intensities compared to subcutis and muscle injection. injection of isotonic saline only led to very weak pain ratings in all tissues. Injections into the fascia induced the highest perceived mean peak pain.

Both hypertonic and isotonic saline injections induced LBP located unilaterally on the side of injection. Hypertonic injection led to a more widespread distribution of pain.
Pain qualities that were significantly stronger after hypertonic than isotonic and differed between tissues: for subcutis “burning”,”throbbing”,”scalding”,”stinging”,”hot”; for fascia “beating”,”throbbing”,”scalding””hot”; for muscle “beating”. Affective pain descriptors (“agonizing”, “heavy”, “horrible”) were chosen for subcutis and fascia.
No significant pressure hyperalgesia was identified after hypertonic saline injection into the fascia or subcutis, compared to isotonic injection.

Discussion/ Conclusion (study author)

Many osseous and soft tissues are related to the development of LBP and this study confirm that the human thoracolumbar fascia is more sensitive to chemical stimulation than the underlying tissues and muscles. Contrary to the author’s hypotheses, hyperalgesia to blunt pressure (frequently related to acute and chronic LBP) could only be induced by injections into the muscle, but not the fascia or subcutis. Although fascia stimulation led to a higher pain intensity, duration and radiation, the muscle seems to be more prone to chemically induced hyperalgesia than the fascia and subcutis. Nociceptive afferents within the thoracolumbar fascia are important for the detection of chemical stimulation in the lower back. According to the authors, the thoracolumbar fascia provides a higher afferent barrage and may play a more dominant role in the development or persistency of LBP than the muscle. The pain was confined to the ipsilateral side with no radiation to the contralateral side. The pain radiation after fascia injection was in the typical location of LBP. Moreover, the descriptors used by the participants were similar to the sensory and affective pain components usually reported by nonspecific acute LBP patients. Authors concluded that an inflammation or disorganization of the thoracolumbar fascia may contribute to chronic LBP.

Comments of CAP- summarizer

Several tissues may be sources of nociceptive input for low back pain. Fascia and soft tissue have been less investigated compared to joint and nervous system.

In this study the contribution of fascia as a potential source for low back pain has been investigated. The hypothesis of an involvement of fascia as a potential source of pain comes from recent studies including the one from Tesarz (2011) in which it was found that the thoraco-lumbar fascia may be an important source for low back pain since it is densely innervated and the outer layers of the fascia have more sensory and nociceptive fibres than the muscle.

In the last few years a lot of publications have been released on fascia. Some studies pointed out the relationship between fascial tissue alterations and LBP. Augmented fascial thickness and reduced shear strain were found in chronic LBP patients compared to healthy subjects (Langevin et al 2009; 2011). Fascial stiffness seems to be correlated to low back pain, even if a correlation to pain or recurrence of low back pain episodes hasn’t been proved (Langevin et al 2011). Therefore fascial alterations may be a consequence and not a cause of low back pain. Further studies should be done to investigate movement patterns in low back pain and fascial stiffness.
The study from Schilder et al is the first which investigate the pain pattern of fascia and

subcutis in asymptomatic subjects. A previous study done on rats showed evidence that lumbar dorsal horn neurons receive nociceptive input from the fascia (Hoheisel et al 2011), therefore this was a premise to further investigate fascial tissue as a source of symptoms.

The pain pattern found in this study, the distribution and the description, as mentioned by the authors were consistent with the patient's usual complaint of low back pain. Therefore Fascia could be a source of low back pain.

Overall this study was performed with a good methodological quality. All the procedures in the study are well described and this makes the reproducibility of the study high.
The inclusion criteria used only considered absence of previous low back pain episodes and exclusion criteria only considered previous surgery to low back or leg. This criteria might be extended to other factors like previous spinal pain or diseases (rehumatoid arthritis or ankilosing spondilitis, fractures, history of whiplash..).

Only the participants were blinded, while assessors were not blinded. Further studies should consider having the assessors blinded as well.

This study provides information that fascia may have nociceptive input; the sensory innervation in the non-specialised connective tissue has been investigated by Conrey et al (2011) to see how this may potentially contribute to pain perception. They observed that the contribution comes probably from A-delta and C fibres due to the immunoreactivity to calcitonin-gene-related (CRPG) and the size of this nerve fibres; this again supported the hypothesis of the contribution of fascia and connective tissue to the development of chronic low back pain (Corey et al 2011).

As fascia can be a source of nociception, it means that it can be subjected to other phenomena which involve the peripheral and the central nervous system like peripheral sensitization and central sensitization. Previous studies confirm the role of soft tissue in various pain mechanisms. Myofascial pain seems to be one of the sources of peripheral nociceptive input inducing central sensitisation (Ge et al 2011). Further evidence for the contribution of fascia in processing mechanisms is given by the study from Corey et al in which it was concluded that the functional changes associated with neurogenic inflammation and persistent pain signals to the central nervous system may be one possible contribution (Corey et al 2011)
Fascial stiffness can also be considered an output mechanism induced by altered Ph, nutrition and chemical stimulation (Schleip 2006).
Even if the Fascia can be a source of nociception and potentially of low back pain, an assessment of the dominant pain mechanism in the patient's problem is mandatory and this will be a guidance for the management.

Related references

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