Discogenic Pain Etiology

Discogenic pain is attributed to degenerative changes in the intervertebral disc due to ageing or trauma. The healthy disc of an adult has scattered nerves which are mainly restricted to the outer lamellae. Degenerated discs have nerves that go through deeper intradiscal structures till the inner third of the annulus and the nucleus. These nerves contain nociceptive neurotransmitters and initiate production of cytokines, provoking nociceptive information from within the disc [1].

The von Mises stress on the lumbar region in the three loading conditions (flexion-extension, lateral bending, and axial rotation) was greater in most components of osteoporotic vertebrae than in normal vertebrae and the value was highest in the nucleus pulposus. Considering the increase in the measured von Mises stress and the local increase in the pressure distribution, they believed that these results can contribute to explaining discogenic pain and disc degeneration [2].

Inflammatory cytokines, such as interleukin 6 and tumor necrosis factor-α, are gaining attention as important etiologic factors associated with discogenic pain.

The expressions of IL-1 and IL-6 in the discs of discogenic low back pain (DLBP) patients were significantly elevated and negatively correlated with the Modified Japanese Orthopaedic Association scale (mJOA) of low back pain [3].

Pro-inflammatory chemokines CCL5 and CXCL6 are released by induced degenerative discs, and CCL5 has been associated with discogenic back pain. A case-control study was performed, based on the Hong Kong Disc Degeneration Population-Based Cohort of Southern Chinese, to investigate if systemic levels of CCL5 and CXCL6 were elevated in subjects with disc degeneration compared to non-degenerated individuals. Eighty subjects were selected, 40 with no disc degeneration (control group; DDD score 0) and 40 with moderate/severe disc degeneration (disc degeneration group; DDD score ≥5) as noted on MRI. Subjects were matched for age, sex, body mass index and workload. Blood plasma samples were obtained from each individual, and levels of CCL5 and CXCL6 were measured. Secondary phenotypes of lumbar disc displacement and cervical disc changes were also assessed. CCL5 concentrations were significantly increased in the disc degeneration (mean: 19.8 ng/mL) compared to the control group (mean: 12.8 ng/mL) ($p = 0.015$). The degeneration group demonstrated higher levels of CXCL6 (mean: 56.9 pg/mL) compared to the control group (mean: 43.4 pg/mL) ($p = 0.010$). There was a trend towards elevated CCL5 levels with disc displacement in the degeneration group ($p = 0.073$). Cervical disc degeneration was not associated with elevated chemokine levels ($p > 0.05$). This is the first study to note that elevated systemic CCL5 and CXCL6 were associated with moderate/severe lumbar disc degeneration, further corroborating tissue studies of painful discs. These chemokines may be systemic biomarkers for the diagnosis and monitoring of disc degeneration [4].

1)
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