

Histology and pathology of the human intervertebral disc.

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Abstract

The intervertebral disc is a highly organized matrix laid down by relatively few cells in a specific manner. The central gelatinous nucleus pulposus is contained within the more collagenous anulus fibrosus laterally and the cartilage end plates inferiorly and superiorly. The anulus consists of concentric rings or lamellae, with fibers in the outer lamellae continuing into the longitudinal ligaments and vertebral bodies. This arrangement allows the discs to facilitate movement and flexibility within what would be an otherwise rigid spine. At birth, the human disc has some vascular supply within both the cartilage end plates and the anulus fibrosus, but these vessels soon recede, leaving the disc with little direct blood supply in the healthy adult. With increasing age, water is lost from the matrix, and the proteoglycan content also changes and diminishes. The disc-particularly the nucleus-becomes less gelatinous and more fibrous, and cracks and fissures eventually form. More blood vessels begin to grow into the disc from the outer areas of the anulus. There is an increase in cell proliferation and formation of cell clusters as well as an increase in cell death. The cartilage end plate undergoes thinning, altered cell density, formation of fissures, and sclerosis of the subchondral bone. These changes are similar to those seen in degenerative disc disease, causing discussion as to whether aging and degeneration are separate processes or the same process occurring over a different timescale. Additional disorders involving the intervertebral disc can demonstrate other changes in morphology. Discs from patients with spinal deformities such as scoliosis have ectopic calcification in the cartilage end plate and sometimes in the disc itself. Cells in these discs and cells from patients with spondylolisthesis have been found to have very long cell processes. Cells in herniated discs appear to have a higher degree of cellular senescence than cells in nonherniated discs and produce a greater abundance of matrix metalloproteinases. The role that abnormalities play in the etiopathogenesis of different disorders is not always clear. Disorders may be caused by a genetic predisposition or a tissue response to an insult or altered mechanical environment. Whatever the initial cause, a change in the morphology of the tissue is likely to alter the physiologic and mechanical functioning of the tissue.

PMID: 16595436 [PubMed - indexed for MEDLINE]