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DAMAGE TO THE YELLOW LIGAMENT IN DEGENERATIVE DISEASES OF THE LUMBAR REGION OF THE SPINE

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Article history:	Abstract:
Received: June 28 th 2023	This study studied the pathomorphological changes that develop in the yellow
Accepted: July 28 th 2023	ligament of the spinal canal in all types of degenerative diseases of the lumbar
Published: August 30 th 2023	spine. Yellow body fragments covering the posterior border of the spinal canal were taken as Material. The results show that in some degenerative diseases of the lumbar spine, the yellow ligament has only undergone hypertrophy, while in others it has developed dystrophy, limescale, and in others it has ossification and deformity. Developing after hypertrophy, thickening, degenerative and inflammatory diseases of the yellow ligament of the spine, it begins with an increase in cysticitra cells around the blood vessels and in the intermediate tissue. Hypertrophy of the yellow ligament shows a sharp increase in collagen
	fibers, the development of the fibroelastosis process, the breakdown and
	destruction of elastic fibers, and a decrease in the amount of bialn.

Keywords: spine, yellow ligament, degenerative diseases, morphology, calcinosis, limescale.

RELEVANCE OF THE PROBLEM. The spinal yellow ligament is located inside the spine, forming the posterior border of the spinal canal. If the yellow body thickens, the spinal canal narrows. Narrowing of the spinal canal occurs in congenital and acquired manifestations, in any case, the canal torax can compress the spinal cord and lead to important complications, including paralysis of the legs or the development of disorders of the function of the taz organs (1). This pathology is most common in men compared to women, geographically it is most common Africa, in the population of Japan, North afroamekanada. The causes of this pathology have not been studied, in most cases they attribute to metabolic and endocrine diseases (2, 3). Morphologically, it is observed that the yellow ligament undergoes ectopic ossification, infiltration with fibroblast cells, calcification in conjunction with the hard veil of the spinal cord. Clinically, in most cases, chronic myelopathy of the thoracic spine, muscle weakness, shoulder pain, paresthesia in the legs are observed (4, 5). In computed tomography, there is a thickening of the yellow ligament of different degrees at the expense of liming and ossification, in magnetic resonance tomography (MRI), the appearance of hypo-and hyperintensive stripes on the yellow ligament. The pathology of the yellow ligament will need differential diagnosis with degeneration of the branched ligaments of the spine and meningioma (6, 7). The consequence of this pathology is often bad, it can lead to the development of myelopathy in the spinal cord, the development of irreversible changes in the cranial brain. From the above discussion, it turned out that in degenerative-dystrophic diseases of the lumbar region of the spine of various types, there is no data bias in the literature on the development of yellow ligament damage and pathomorphological changes in it. This study aims to identify specific aspects of the pathomorphological changes that develop in the yellow ligament in each of the degenerative-dystrophic diseases of the lumbar spine.

MATERIAL AND METHODS. As the material of this scientific study, a yellow bond was obtained in the Department of Neurosurgery of the adti clinic, which during the 2019-2022 surgical procedures in degenerative-dystrophic diseases of various types of spine, namely discectomy, laminectomy, binds the vertebrae together. The tissue flakes were hardened in formalin dissolved in a 10% phosphate buffer for 72 hours, then all the Flakes were rinsed in running water for 3-4 hours, dehydrated in alcohol with increased concentration, and paraffin with wax was poured and the bricks were prepared. Histological incisions 5-7 µm thick were made from paraffin bricks and painted in the paints of hematoxylin-eosin and van-Gison, Weigert methods. The preparations were studied under a light microscope and pictures were taken from their desired areas.

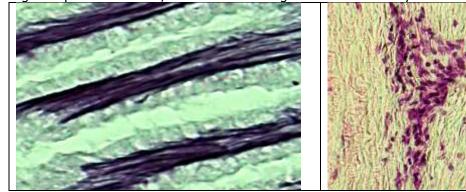
Results of morphological examination and their discussion.



The yellow ligament of the spine is sufficiently resistant and elastic tissue without meioria. In all types of degenerative diseases of the lumbar region of the spine, various pathomorphological changes develop in the vellow ligament. As risk factors that lead to it, the following can be indicated: above 40 years of age, a large number of pregnancies in women, high body weight, smoking, alcohol Isthmus, injuries. One of the main variations is considered to be the hypertrophy of the yellow bond, which is diffusely thickened in one place - in the furnace or throughout its entire area. Most often, it is most common in the spine of the lumbar region when considering the anatomical-physiological structure and multiple lesions. Microscopically, the cells it contains proliferate, increasing fibrosis fibers, sometimes with chronic inflammation found to have developed. Thickening of the yellow ligament can cause the following complications: narrowing of the spinal canal, paralysis of the limbs, dystrophy of the muscles of the shoulders, arms and legs, chronic diseases associated with the vegetative nervous system, pathologies of the internal organs, circulatory disorders of the brain, kidney diseases, pathology of the intestines, bile ducts, gallbladder and taz organs. Sooner or later, hypertrophy of the yellow ligament begins to have a negative effect. The yellow band thickness or weight change automatically narrows the spinal canal. If, in addition to it, there is protrusion, fibrosis of the sac hernia, displacement of the spine, its negative effect on the spinal cord becomes even more severe. Compression of the spinal cord occurs, especially if a hernia appears on the back of the disc. This leads to total paralysis in the body. This pathology, if it develops in the neck or neck-chest area of the spine, leads to sudden cardiac arrest, paralysis of the respiratory tract.

In the case of a Schmorl hernia, a change in the yellow ligament was found to have a tasivr as follows. The cause of a schmorl hernia is the rapid development of the soft tissues of the spine due to the rapid growth of the body in a person's youth, the lag behind the growth of bone tissue and the formation of a cavity in the cartilaginous part of the bone, the curtain covering the articular surface of the spinal cord sinking into the cartilaginous part of the bone over time. In the cartilaginous part of the spine, strong and irreversible dystrophic and destructive changes in the compact bone columns are found to develop, resulting in fibrosis dysplasia and calcinosis, with the bone columns losing their histotopography and entering a structureless state. In the cartilage bone pores, blood clots, a carbohydrate and protein substance, connective tissue Tufts and calcinosis, in some cases complete lipomatosis, are found to have developed instead of bone cuticle cells in the meiore. Due to the fact that the elastic fiber curtain covering the cartilaginous Bone also shows the breakdown of elastic fibers, homogenization, formation of coarse protein, vacuolization and breakdown of the intermediate substance, the yellow ligament is relatively poorly damaged and only the ligament is deformed and hypertrophied in the foci. . Considering that the hernia of the lumbar region of the spine is mainly damaged by the thoracic disc, the fibrosis curtain, the ligaments around it, the yellow ligament is more damaged by the fact that its placement in these structural units is close. In fact, the yellow bond consists of a densely formed connective tissue. Collagen and elastic fibers are located in the structure, collagen fibers provide durability, elastic fibers provide shrinkage property. In the yellow bond, elastic fibers are located in a significantly larger amount compared to collagen fibers. Collagen fibers consist of a tuft located parallel to each other, between which elastic fibers occupy a place (1rasm). These totalic structures are first firmly attached to the outer veil of the bone, then adhere to the bone.

In the hypertrophy and thickening of the yellow ligament of the spine, an inflammatory process initially begins in the tissue of the ligament structure, an increase in histicitra cells is detected around the blood vessels and in the intermediate tissue (Figure 2). The increased histiocytic cells proliferate in place and spread to the surrounding tissue, producing collagen fibers in excess of their quantity. As a result, the collagen fibers in the yellow bond change their order and settle unevenly.



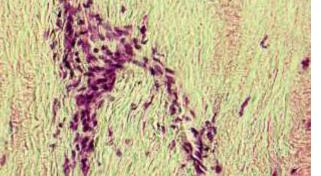
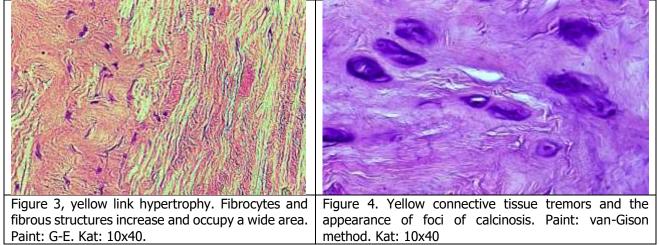




Figure 1. The spine is a yellow band. Elastic fibers
are located in the form of dense Tufts, which are
located separately from each other. Paint: Weigert
method. Kat: 10x100.Figure 2. Yellow ligament hypertrophy. Proliferation of
histiocytic cells with foci and diffuse proliferation. Paint:
G-E. Kat: 10x40.



The fibrocytes contained in the yellow bond collagen fiber Tufts undergo proliferative activation and increase numerically, with a sharp increase in the amount of collagen fibers around them. In some areas of the yellow ligament, it is observed that collagen fibers are sharply increased, fibrous tissue areas that cover a wide area appear, in some areas they are homogenized and turned into fibroelastosis (Figure 3). In other areas of the yellow band, however, it is found that collagen fibers are titrated, homogenized in some areas, and spread between elastic fibers. In the process of hypertrophy and thickening of the yellow bond, it is observed that in the structure of the bond tissue, elastic fibers atrophy and decrease, collagen fibers are produced in large quantities, occupying most of the space of the yellow bond structural area. When the van-Gison method is used from histochemical methods for determining collagen fibers of the connective tissue, it is found that most of the area of the structure of the vellow connective tissue is occupied by collagen fibers, which are colored with picrophoxin in a positive red color. It is observed that the amount of elastic fibers between collagen fibers decreases and gaps appear in some areas.

Osteophytes that appear on the anterior edge of the spine in spondylesis disease have been shown to have morphologically а concentric structure, fibrous structures and the main substance are located betartib, consisting of a tissue rich in limescale (Figure 4) and pigmentation foci. In the living nucleus, it was initially found that the chondroid substance was roughened, impaired, the fibrous structures staining and intermediate contained in it were dispersed and thickened, the chondrocyte content increased, and they underwent processes such as dystrophy and destruction to varying degrees. In chronic spondylesis disease, chondrocytes in the dirhyl nuclear tissue were confirmed to have been completely destroyed and necrobiosed to become structurless substances, with foci of limescale. As a result of the sinking of osteophytes into the fibrosis sac tissue of the disc, it was found that its fibrous structures were fragmented, destroyed, a coarse dispersive substance appeared, and the development of limescale and chondromatous metaplasia in the structure also spread to the yellow ligament tissue around them, hypertrophied in the yellow ligament, developed Liming, ossification in some areas, and narrowing of the spinal canal.

Spondyloarthrosis of the lumbar region of the spine saw damage to all tissue structures in and around the Joint, including the femur, lower bone, vertebrae, fibrosis curtain, yellow ligament, and surrounding muscles. A protein and carbohydrate dystrophy of an intermediate chondroid substance was found in the thoracic disc of the joint, a betartib localization of chondrocytes, the development of vacuolization in the pvcnosalib and cytoplasm of its nuclei. Deformity of solid bone columns in bone tissue below the ankle of the joint, vacuolization of the osteoid substance and destruction of fibrous structures in it, the appearance of foci of calcinosis in all parts of the process due to migration to the yellow ligament. In the fibrosis tissue lining and veil around the ridge, hydrotasia from the transformation of mucopolysaccharides into a sour environment was initially observed in the intermediate substance of the connective tissue, resulting in the formation of edema, as a result of which the fibrous structures were deformed and destroyed.in skeletal muscle tissue around the T-ridge, it was found that the intermediate tissue undergoes a strong swelling as a result of the



weight that falls on them, being destroyed by dystrophic changes in myofibrils in muscle fibers.

CONCLUSION

- In some of the degenerative diseases of the lumbar spine, the yellow ligament has only undergone hypertrophy, while in others it has developed dystrophy, limescale, and in others it has ossification and deformity.

- Develops after hypertrophy, thickening, degenerative and inflammatory diseases of the yellow ligament of the spine, begins with an increase in cysticitra cells around the blood vessels and in the intermediate tissue.

- Hypertrophy of the yellow ligament shows a sharp increase in collagen fibers in the structure, the development of the fibroelastosis process, the breakdown of elastic fibers, destruction, and a decrease in the amount of bialn.

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