# Modeling of Osteochondrosis of the Spinal Column

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Focal persisting infection was reproduced in rabbits using *St. aureus* culture. Histological and histochemical study of intervertebral disks 1, 2, 3, 5, and 8 months after the infection revealed dystrophic and degenerative changes and calcium deposits.

**Key Words:** *osteochondrosis of spinal column; experimental model; intervertebral disks*

Pathological changes in intervertebral disks (IVD) are a major cause of numerous reflex and compression syndromes [11-13]. Formation of intervertebral hernia and development of disko-radicular or disko-medullar conflicts, which often lead to invalidization of patients [1], occur at a certain stage of IVD degeneration. Therefore, it is important to know the cause and the phase of osteochondrosis for correct choice of therapeutic strategy at each stage of the disease. However, many aspects of this pathological process remain poorly investigated. This is at least partially due to the absence of an adequate experimental model in which dystrophic and degenerative processes could be reproduced and investigated from the initial stages to calcium deposition in IVD.

Bearing in mind the finding that degenerative changes in rabbit IVD similar to those occurring in humans can be reproduced in rabbits by immunization with an allogeneic antigen [6,7], the data on the influence of chronic infection on cartilages [2,4,8-10], and the fact that the foci of chronic infection are often identified in the majority of patients with osteochondrosis [3,5], we think that it is reasonable to investigate the influence of chronic inflammation on the structure of IVD.

## MATERIALS AND METHODS

Chronic osteomyelitis of the femur was reproduced in 16 adult male Chinchilla rabbits using *Staphylococcus aureus* culture (strain 207). Morphological changes in IVD were studied after 1, 2, 3, 5, and 8 months of the infection. Adult intact rabbits served as a control.

Three IVD from cervical, thoracic, and lumbar segments of the spine were collected for the investigation. They were fixed in 12% neutral formalin and 96° alcohol and embedded in paraffin. Serial sections (8-10 μ) were stained with hematoxylin and eosin and picrofuchsin by the method of Van Gieson. Calcium salt deposits were visualized by silver nitrate impregnation (Koss techniques) with or without counterstaining with safranine. For detalization of morphological data, some sections were stained for neutral glycoproteins (PAS reaction, McManus techniques) and glycosaminoglycans (toluidine blue).

## RESULTS

By the end of the first month, metachromatic extracellular space was observed in IVD of some animals (Fig. 1). In individual chondrocytes and chondrocytes forming isogenic groups the nuclei were deformed and pyknotic. In some chondrocytes, chromatin granules were located at the periphery of karyoplasm or at the nuclear pole as a small hyperchromatous particle. Some chondrocytes had no nuclei; the remains of their cytoplasm were eosinophilic.

Two months after the infection, all these changes were observed in the majority of studied IVD. Disintegration of fibrous plates forming the fibrous ring occurred in some disks.

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After 3 months, areas with impaired concentrical arrangement of the fibers were observed. These areas contained numerous round or triangle chondrocytes located along the fibers and occasional large cells. In the overwhelming majority of cells the nuclei were deformed. Sometimes large deformed cavities were seen (Fig. 2). They were surrounded by fragmented collagen fibers and were partially filled with poorly stained amorphous substance. In preparations stained for calcium salts (impregnation with silver nitrate), black granules varying in size or shape were accumulated at the periphery of fibrous rings between the fibers. Sometimes considerable accumulations of these granules were observed.

After 5 months, the hyaline cartilage was thinned. The ingrowth of loose fibrous connective tissue into the pulpy substance through small defects of the cartilage was observed. Impregnation with silver nitrate revealed calcium deposits at the periphery of IVD. The granules varied in shape and size and were located along collagen fibers near destroyed chondrocytes. Sometimes destroyed chondrocytes were replaced by considerable calcium deposits which looked like conglomerates of various shape (Fig. 3).

Eight months after infection, all the above-mentioned alterations were aggravated, particularly in the areas of necrosis and hyalinization.

Thus, our observations have shown that persisting focal bacterial infection induces dystrophic degenerative changes with formation of calcium salt deposits in the intervertebral disks. This model can be recommended for the investigation of spinal pathologies; it may be useful in the study of the reversibility of degenerative processes in IVD and in evaluation of the effects of biologically active substances, pharmacological agents, and physical therapy at the initial stages of IVD degeneration.

**Fig. 1.** Fragment of rabbit intervertebral disk with markedly decreased basophilia of the extracellular space. One month after initiation of chronic focal infection. Staining with hematoxylin and eosin, ×600.

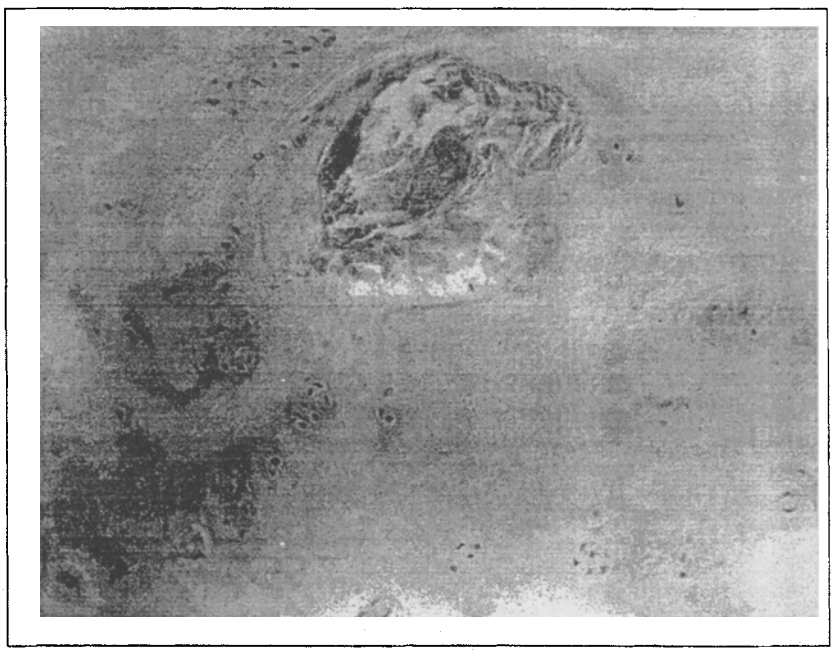

**Fig. 2.** Large cavity filled with amorphous substance, fragmented chondrite fibers and dead chondrocytes in rabbit invertebral disk. Five months after initiation of chronic focal infection. Van Gieson stain, ×200.

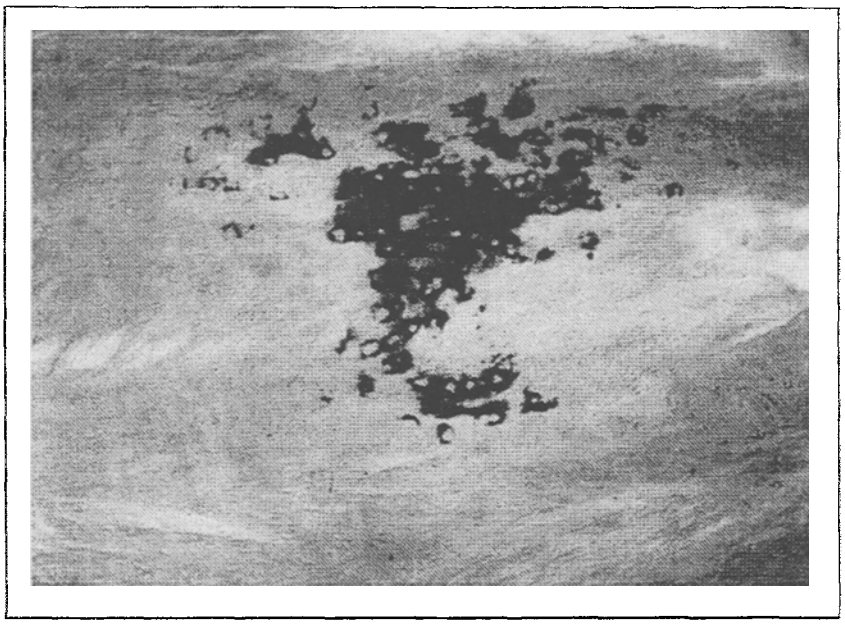

**Fig. 3.** Calcium salt deposits as conglomerates of various shape in the fibrous ring of intervertebral disk. Three months after initiation of chronic focal infection. Impregnation with silver nitrate with subsequent staining of the nuclei with safranine, ×200.

## REFERENCES


1. L. S. Gitkina, G. S. Severin, I. P. Antonov, *et al., Peripheral Nervous System* [in Russian], Minsk, Vol. 12, pp. 136-141 (1989).
2. V. I. Pokrovskii *et al* (Eds.), *Immunology of Infectious Process* [in Russian], Moscow (1993).
3. N. I. Komandenko and Zh. A. Astreiko, *Klin. Med.*, **69**, No. 7, 86-89 (1991).
4. T. N. Kop'eva, *Pathology of Rheumatoid Arthritis* [in Russian], Moscow (1980).
5. T. M. Kukhnina, *Kazan' Med. Zh.*, **59**, No. 1, 21-22 (1978).
6. V. Ya. Latysheva, *Peripehral Nervous System* [in Russian], Minsk, Vol. 5, (1982), pp. 50-58.
7. V. Ya. Latysheva and I. N. Shevchenko, *Ibid.*, Minsk, Vol. 4, (1981), pp. 60-67.
8. M. A. Markov, *Specific Clinical Manifestations and Remote Results of the Treatment of Reactive Arthritis in Nasopharyngeal Infection in Young People* [in Russian], Abstr. Ph. D Thesis, Yaroslavl (1986).
9. G. D. Ovrutskii, *Chronic Odontogenic Focus* [in Russian], Moscow (1993).
10. D. Pashkauskene, In: *Problems of Rheumatoid Arthritis* [in Russian], Vilnius (1981), pp. 72-75.
11. Ya. Yu. Popelyanskii, *Vertebragenic Diseases of the Nervous System* [in Russian], vol. 2, part, 1, Yoshkar-Ola (1983).
12. Ya. Yu. Popelyanskii, *Vertebragenic Diseases of the Nervous System. Pelvicomembraneous Syndromes of Lumbar Osteochondrosis* [in Russian], part 1, Kazan (1983).
13. Ya. Yu. Popelyanskii, *Diseases of the Peripehral Nervous System* [in Russian], Moscow (1989).