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OSTEOCHONDRITIS DISSECANS

A Histologic and Autoradiographic Study in Man

B. LINDÉN & H. TELHAG

Department of Orthopedic Surgery, Malmö General Hospital, (University of Lund), Malmö, Sweden.

Surgically excised cylindrical specimens from 14 adult patients with osteochondritis dissecans in the knee joint were studied with histologic and autoradiographic techniques. The lesions were found to be composed of normal hyaline cartilage with no pathological changes. In the bone part of the specimens there were well-defined fissures at various distances from the tidemark. On both sides of the fissure, but especially on the OD side, there were scattered osteonecroses in complete disarray. No tetracycline fluorescence activity could be seen in the lesions.

Key words: osteochondritis dissecans; autoradiography; fluorescent microscopy

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More than 100 years have elapsed since Sir James Paget in 1870 described osteochondritis dissecans (OD) and called it "quiet necrosis". König in 1887 first used the name OD for lesions he had seen in the human knee. Since these first descriptions of the lesion, many theories have been put forward as to its etiology. Several etiologic factors, i.e., trauma, ischemia and hereditary abnormalities of ossification have been suggested.

The condition has been described in animals (Reiland 1975). Here the primary disease is in the epiphyseal cartilage with secondary changes in the bone (Olsson 1976). But most authors consider that in human beings there is primarily a separation of a cartilage-bone fragment from the subchondral bone (Conway 1937, Smillie 1960, Aichroth 1971 a).

Although the long-term course of the disease has been clarified by Lindén (1976), morphological studies have so far been sparse. Scintimetric studies *in vivo* (Lindén & Nilsson 1976) have shown a very moderate, increased radioactive uptake in the lesion. No autoradiographs or fluorchromic studies of OD have been published.

The purpose of the present investigation was to submit a histologic and autoradiographic study of the cartilage and the bone in OD in adults.

MATERIAL AND METHODS

The series consisted of 14 patients (9 men and 5 women) aged 15–33 years (average 20 years). The patients had had symptoms of OD in the

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knee joints for periods ranging from 9 months to 17 years (average 4.9 years). The lesions were *in situ* in the medial femoral condyles in 10 knee joints. In another four joints the fragment had left the condyle.

Five days before the operation, nine patients received a peroral dose of tetracycline 1 g daily.

Two or three cylindrical biopsy specimens, 2 mm in diameter, were obtained at operation from the osteochondritic lesion of all the knee joints. The specimens comprised articular cartilage and subchondral bone. The biopsies were immediately placed in 10 ml of Eagles solution (37° C), containing 50 μCi ^{3}H-thymidine and shaken for 3 hours. One of the sections was washed in physiologic saline solution and fixed in 10 per cent neutral buffered formalin, decalcified in 40 per cent formic acid and 20 per cent sodium citrate. The decalcified section was embedded in paraffin. Sections for histologic and autoradiographic study were prepared to a thickness of 7 μ, and stained with hematoxylin-eosin, Safranin-O, toluidine-blue and according to van Gieson. Autoradiographs of routine histological sections, 7 μ thick, were prepared according to the dipping method with Ilford K2 liquid emulsion. After 3 weeks exposure, the autoradiographs were developed in Gevaert X-ray developer G230 and fixed in Gevaert X-ray fixer G305. The sections were stained through the emulsion with Mayer's hematoxylin. The second slice was fixed in formalin, dehydrated and embedded in metylmetacrylate without decalcification. Sections, 7 μ thick, were cut with a bone microtome. These sections were stained with Goldner stain (Schenk 1965) and examined for osteoid tissue. One part of the second slice was left unstained for fluorescent microscopy.

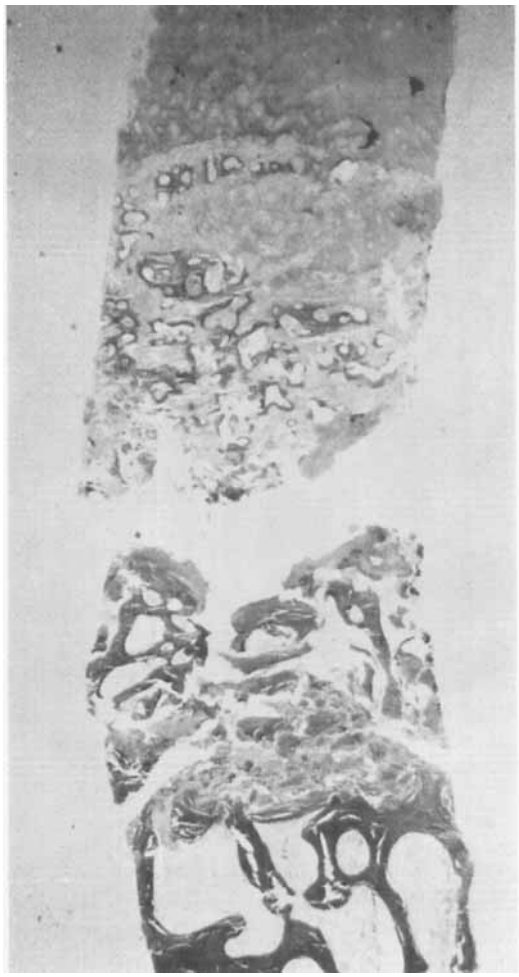

Figure 1. Surgically excised cylindric specimens from osteochondritis dissecans lesion in a knee joint. On the top, the columnar and basal layer of the hyaline cartilage with a normal appearance. In the bone part, a clear fissure is seen with disorganization and osteonecrosis on both sides (Goldner × 1).

RESULTS

No thymidine-labeled chondrocytes were found in the autoradiographic preparations. The number, the orientation and the appearance of the chondrocytes in the hyaline articular cartilage were within normal limits.

Hematoxylin-eosin stained sections, and sections stained according to van Gieson, showed normal hyaline articular cartilage. Safranin-O staining demonstrating the content of glycosaminoglycans in the cartilage varied from none at all to very deep staining in the different preparations. The nuclei of chondrocytes in the cartilage were normal as seen with toluidine-blue staining. The appearance of the hyaline articular cartilage was that of normal viable laying over an ossific nuclei. No clefts were seen. In the preparations from the four patients with empty beds the bottom was covered with fibrous cartilage of varying thickness.

In the ossific part a well-defined fis-

Figure 2. The same specimens as in Figure 1. On both sides of the fissure new mesenchymal tissue has formed (Goldner × 2.5).

sure was found in the bone located, in different specimens, at a varying distance from the overlying articular cartilage. The fissure was situated immediately under the hyaline articular cartilage in one preparation, but in another at a distance of up to 10 mm from the tidemark in the articular cartilage (Figure 1). On both sides of the fissure, but especially in the detached fragment there were islands of necrotic bone with disorganization of the trabeculae in the form of fragmentation, but also coarse and clumsy trabeculae. Some of the lacunae were empty, but normal osteocytes could also be seen in the lacunae (Figure 2). Both osteoblastic and osteoclastic activity were observed. In preparations stained according to Goldner, an increased number of osteoid seams near the fissure could be visualized on both its sides.

In some preparations newly formed connective tissue, at various stages of differentiation, had begun to fill out the fissure. In some, this differentiation had gone so far as to be fibrous cartilage as seen with toluidine-blue staining.

Further from the fissure the bone became more normal on both sides with normal marrow and gracile trabeculae with lacunae accompanied by osteocytes. No osteoid seams could be seen here.

With fluorescent microscopy no tetracycline labeling was visible in the bony part of the osteochondritic lesion.

DISCUSSION

Osteochondritis dissecans may be defined as a partial or complete separation of a cartilage-bone fragment from a joint with the separation of the fragment from the surrounding tissue taking place gradually (Smillie 1960).

The cause of this separation being obscure, no theory is fully accepted. The hypothesis of *ischemia* postulates an interruption of the blood supply to an area of subchondral bone, which would then lead to sequestration of the bone together with the overlying articular cartilage (Riegan 1920, Axhausen 1922, Watson-Jones 1952). This interruption is supposed to be caused by emboli of fat, bacteria or red corpuscle aggregation.

Some form of osteonecrosis has been postulated. The osteonecrosis may appear as the result of an interrupted blood supply consequent upon traumatic squashing without fracture of the articular bone end (Roessner 1922, Jaffe 1972).

The hypothesis of *trauma* has been accepted by various authors, from the first investigation of König in 1887 up to the present time. The trauma can be an impact of the bone by the opposing joint surface (Axhausen 1914), rotating forces (Kappis 1920), bone impression of the

patella (Hellström 1922), or injuries from the eminentia intercondyloidea (Fairbank 1933, Smillie 1960, Scheller 1960).

There is a certain amount of experimental support for the traumatic hypothesis (Rehbein 1950, Langenskiöld 1955, Tallquist 1962, Aichroth 1971 a). Indeed, there is no clear borderline between OD and an old osteochondral fracture, although by definition the osteochondral fracture occurs momentarily in conjunction with trauma and hemarthrosis (Aichroth 1971 b).

In animals, the basic feature of OD is a disturbance of endochondral ossification. The lesion is primarily located in the cartilage of the joints and the growth plate, this becoming thicker than normal and dying in the deepest layers. Cracks and fissures occur, and synovial fluid gets into the cracks and reaches subchondral bone causing inflammation in the joint (Olsson 1976).

In a histologic and microradiographic analysis of surgically excised lesions, Chiroff & Cooke (1975) postulated that the bone of the ossific nucleus was newly formed and not merely separated from the femoral condyle by trauma.

This investigation demonstrated a fissure at a varying distance from the cartilage surface, a fact which could also be seen in radiograms. From both sides the fissure tended to be filled out with fibrous cartilage as a sign of a reparative process but blocked by a poor blood supply to the fragment. The low uptake of tetracycline and strontium-85 (Lindén & Nilsson 1976) is an indication of a poor blood supply.

The articular cartilage of the lesion was normal in all respects and no thymidine-labeled chondrocytes were found, i.e., no DNA synthesis and consequently no cell division. The thickness of the cartilage was normal in all preparations.

It is apparent that the sequence of events is primarily in the bone in man and not in the cartilage as in animals (Olsson 1976). Aichroth (1971 a) has shown that unstable osteochondral fractures fail to unite. Histologically, these ununited fragments resembled OD lesions with a viable hyaline articular cartilage lying over acellular necrotic bone. On the basis of the present investigation one cannot deny that some form of trauma plays a role in the formation of OD.

This investigation showed that OD in adults seems to represent some form of subchondral fracture with intact articular cartilage. If the healing process is restricted, the mobility of the fragment gives rise to cracks in the cartilage.

In treating OD, the aim must be to improve the blood supply to the lesion, i.e., to obtain connection with the medullary cavity.

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Correspondence to: Hans Telhag, M.D., Department of Orthopaedic Surgery, Malmö General Hospital, S-214 01 Malmö, Sweden.