FOCAL LACUNAR RESORPTION IN THE ARTICULAR CARTILAGE
OF FEMORAL HEADS

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Human articular cartilage taken from 92 femoral heads at autopsy was examined macroscopically and microscopically. Fifty-two showed no changes except for occasional slight degeneration in the non-pressure areas; these changes were visible only microscopically. In the remaining 40 heads, different degrees of osteoarthrosis* were seen; half the heads also showed focal lacunar resorptive lesions in the cartilage.

The origin of this focal cartilage resorption is discussed and its possible association with necrosis, pannus formation and enzymatic synovial activities. We conclude that there is no evidence of a direct relationship between focal cartilage resorption and osteoarthritis.

More than 100 years ago Weichselbaum (1877) described a peculiar focal histological change in human articular cartilage, now known as Weichselbaum'sche Lücken in the German literature. These histological changes were characterised by lacunae where hyaline cartilage was replaced by loose fibrous tissue with spindle-shaped cells. Weichselbaum postulated a relationship between the lacunae, ageing cartilage and osteoarthritis*. In a second publication (1878), he suggested a relationship between inflammatory processes in the synovium and "bone caries", that is, articular tuberculosis. Later Pommer (1915) and Lang (1925) confirmed his observations.

These lacunae, originally observed in pathological material, were later described as one of the findings in ageing cartilage taken from apparently normal joints. Thus Bennett, Wain and Bauer (1942) frequently found focal matrix resorption in ageing cartilage from "normal" knee joints of various ages. Byers, Contepomi and Farkas (1976) and Vignon and Arlot (1981) also observed this change in ageing femoral heads. However, no further description or examination of this lacunar resorption, or pocket formation as the process was called, has been published. In order to obtain new data of its incidence and significance, we studied the cartilage of femoral heads from subjects of various ages without known clinical joint disease.

MATERIAL AND METHODS

Femoral heads were obtained at autopsy from patients who had died from causes not related to joint disease. A total of 92 femoral heads from 61 men and 31 women aged 21 to 89 years were examined. From the normal-looking heads (52), samples were taken from the pressure and non-pressure areas. From the heads which showed arthrotic changes or cavities (40), one or two samples were taken from the defective areas.

Diagram of a femoral head depicting the location of the samples taken.

In each case a strip of cartilage with underlying bone was cut out with a saw, fixed in 4% formalin and decalcified in Na-EDTA in 9.5% HCl solution† for five to seven days. The tissues were embedded in Paraplast and

* In this journal we normally use the word osteoarthritis. The authors of this paper have, however, requested that in view of their other publications the ending 'osis should, in this particular instance, be preserved.

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5 μm sections were stained with haemalum and eosin, Alcian blue at pH 2.5 and Goldner trichrome stain.

RESULTS

Macroscopic appearances. Fifty-two femoral heads were macroscopically unchanged; the remaining 40 showed degeneration of slight to moderate severity.

Figure 1 shows the location and distribution of cartilage defects; the number of defects at each site is given in parenthesis. It can be seen that the marginal area (MA) of the femoral head was the most common site of defects, and was involved in 25 samples. From the lateral area (LA) 13 samples showed defects, and from the non-pressure area (NPA) 14. The pressure area (PA), with only 9 samples involved, was the least affected site; the perifoveal area (PFA) had 10 samples with defects. Histology. Histological examination of the 52 femoral heads which on gross examination were normal, disclosed cartilage with a well preserved pressure zone and an occasionally slightly degenerate non-pressure zone.

The samples from the 40 femoral heads with gross changes revealed various osteoarthritic manifestations such as fibrillation, unmasking, cell clusters, multiplication of the tidemark and partially calcified cartilage. In addition to these well-known changes, we observed lesions similar to "lacrinar matrix resorption" or "pocket formation" in 21 of the 40 femoral heads. These were focal lesions and were sharply demarcated from the surrounding cartilage matrix; they were usually rich in cells such as chondrocytes, fibroblasts and chondroblasts. The collagen fibres of the matrix formed a loose network; when close to the surface, there was a parallel arrangement.

Lacunar resorption in its most typical form was found at the cartilage surface; it often occurred without...
any other lesion nearby (Fig. 2). In other cases, the resorption was part of (or connected with) pannus. An example of this type is given in Figure 3 where the deepest part of the defect is pannus-like tissue covered by dense fibrous connective tissue. At the surface the morphology of typical lacunar resorption is seen (Fig. 4). Islands of old cartilage matrix are enclosed in loose connective tissue. The bottom of some of the pockets of lacunar resorption displays a fibrillar structure with cells similar to osteocytes and with staining characteristics like those of bone.

Another variation of lacunar resorption combined with pannus, newly formed cartilage and bone, is seen in Figures 5 and 6. In the area of transition between the lesion and cartilage of normal thickness, young-looking cartilage is found (Fig. 6).

A further type of resorption is found in connection with a cavity such as that in Figure 7. This cavity is lined by a thin layer of residual cartilage (Fig. 8). In the transitional area of the cartilage, still another stage of resorption is seen (Fig. 9): here the demarcation to the normal matrix is clearly seen, and young active chondrocytes are also present. The amount of matrix is relatively small compared to the number of cells.

Although the resorptive changes appeared on femoral heads with slight to moderate osteoarthritis, they were usually located in non-arthrosic areas. Only occasionally were they found within an arthrosic area, suggesting that these alterations were independent lesions occurring incidentally in this location.

DISCUSSION

Our microscopic examination of the articular cartilage of femoral heads has shown that lacunar resorption is commonly associated with osteoarthritis of slight or moderate degree, an observation similar to that reported by Bennett, Waine and Bauer (1942). Thus Weichselbaum's original opinion (1878) that focal loss of matrix components must be related to chronic inflammatory processes, cannot be supported.

In a study of autopsy material Byers, Contepomi and Farkas (1976) refer to the occurrence of lacunar resorption in the cartilage of femoral heads as "chondrocytic resorption of the matrix". They found that the defect occurred at the surface of the cartilage and recognised it by "the presence of single and multiple cells lying in a depression or pocket in the cartilage matrix, the line of demarcation from the normal matrix being sharp and clear. This was found either alone or with an overlying fibrocellular tissue." Byers et al. (1976) did not go into the significance and origin of this lesion. The use of the term "chondrocytic resorption", however, indicates that they thought the lesion originated within the chondrocytes. Vignon and Arlot (1981) described the lesion as "cellular changes with pockets of matrix resorption in the superficial layer", also without mentioning anything of its significance.

Whether lacunar resorption is caused by a traumatic event leading to necrosis, and eventually to osteoarthrosis, is an open question. Bauer, Ropes and Waine (1940) reported evidence of a higher frequency of lacunar
resorption in traumatised joints. According to Axhausen (1912), trauma leads first to necrosis and then to replacement by different kinds of tissue, namely true hyaline cartilage or fibrous connective tissue. The emphasis on trauma and necrosis agrees well with our findings where resorption and arthrotic changes do not coincide topographically. An example of lacunar resorption without any other visible lesion, and where necrosis might be the initiating factor, is illustrated in Figure 10.

Another cause of focal resorption might be former pannus activity. Inherent in pannus formation are destructive as well as reparative processes. Once pannus has reached the burnt-out stage, the tissue which remains cannot be distinguished from lacunar resorption. The reaction of the cartilage in such a situation is shown in Figure 11. A third possibility is suggested in connection with marginal articular cavities, in which close contact with the synovium may play a part in the formation of lacunar resorption.

The reaction of cartilage to degrading enzymes derived from synovial tissue in organ cultures has been described by Fell and Jubb (1977). According to these authors, synovial tissue exerts an injurious effect on the matrix of living cartilage. During direct contact between the two, the synovial cells release enzymes which degrade first the proteoglycans and then the collagen of the matrix. Finally the matrix disappears, leaving a compact mass of viable, often actively dividing cells. These experiments suggest that a similar mechanism may also play a role in vivo and may be relevant to our findings. Thus, if lacunar resorption is located at the joint margin, the cartilage may be exposed directly to enzymes from the synovium (Figs 8 and 9). Here we find spots of resorption within the cavity lining. The most striking feature, however, is the transitional area from the cavity to the normal thickness of cartilage. In this area the matrix components have partly disappeared, leaving a mass of active chondrocytes. This finding is in accordance with the results from the in vitro experiments cited above. Figure 12 is a drawing of this phenomenon.

On the basis of our observations one may conclude that cartilage has limited possibilities of repair, and that repair may be triggered by a variety of events. Although arthrotic degeneration and lacunar resorption are usually at different sites, we have not found resorptive changes on femoral heads without degenerative changes. There is no direct evidence that lacunar resorption is an initial stage of osteoarthrosis. However, it is possible that lacunar resorption indirectly influences the development of arthrosis by disturbing the normal function of hyaline cartilage. A similar view has been expressed by Collins and McElligott (1960), who concluded that osteoarthrosis starts in the matrix and that secondary cellular changes are caused by scarification.
REFERENCES


