THE CYSTS OF OSTEOARTHRITIS OF THE HIP

A Radiological and Pathological Study

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Bone cysts are a well recognised radiological feature of advanced osteoarthritis (Plewes 1940, Brailsford 1952). They are seen frequently in relation to the hip joint in both acetabulum and femur, but it is perhaps not always appreciated how numerous and extensive they may be. The present study attempts to correlate the radiological and pathological features of the cysts occurring in the head of the femur, and to elucidate their pathogenesis. A general account of the pathology of osteoarthritis of the femoral head, with a brief discussion of the cysts, was presented by Harrison, Schajowicz and Trueta (1953). The pathology of the cysts was described in detail by Landells (1953), who suggested that they were caused by the intrusion of synovial fluid under pressure. Landells believed that the cancellous trabeculae yield under the pressure transmitted through the fluid, and that the cysts are formed in enlarged marrow spaces. Accordingly, he interpreted the cysts as “virtually incomplete fractures” of the trabecular system, and attributed the development of dense bone around them to “bodily displacement” of trabeculae by fluid and to new bone formation in response to strain.

The hypothesis that subarticular cysts are excavations produced by synovial fluid is not new. It was propounded by Freund (1940) to explain the formation of cysts in rheumatoid arthritis and haemophilic arthrosis. In Freund’s opinion synovial fluid would be forced into the subarticular bone through defects in the surface by the sustained rise in intra-articular pressure resulting from an effusion into the joint. But Cruikshank, Macleod and Shearer (1954) reported the development of cysts in rheumatoid arthritis without clinical evidence of a significant increase of synovial fluid in the affected joints.

In our opinion satisfactory proof that the defects in the bone in osteoarthritis are caused by synovial fluid has not been established. The evidence to be presented is believed to prove that such lesions are foci of bone necrosis caused by violent impact between the opposing surfaces of the joint in the absence of the protection of healthy articular cartilage. Such injury is not unlikely, and it would explain the development of “kissing” lesions, as described by Plewes (1940), on opposing surfaces of the joint. Our studies lend support to the view that cavities filled with synovial fluid are often, but by no means invariably, found in the bony defects. We differ from Landells, however, in believing that bone destruction precedes the entrance of synovial fluid, and suggest that the cavities filled with fluid are formed in the course of repair as the dead bone is removed.

The use of the word “cyst” is liable to give rise to confusion because radiologists and pathologists employ it in such different ways. To the radiologist a “cyst” is simply a circumscribed area of translucency in bone; to the pathologist the term connotes an encapsulation of gas or fluid in any tissue. Since many, but by no means all, of the “cysts” detected radiologically in osteoarthritis do contain encysted fluid, the two usages of the word “cyst” are clearly not synonymous. In the hope of avoiding confusion we shall restrict the word “cyst” to the radiological lesion and use such circumlocutions as “loculi of fluid,” “encysted fluid,” or simply “fluid” for a cyst in the pathological sense.

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MATERIAL AND METHODS

The following observations are based on a study of the excised femoral heads of thirty-six patients with advanced osteoarthritis of the hip, treated by the method of Judet and Judet (1950). Thirty of the patients were suffering from primary osteoarthritis as defined by Mercer (1951), and in six the disease was superimposed on other lesions of the hip. All the patients were ambulant. They were selected for operative treatment because of pain both at rest and on movement. Most of them showed serious limitation of movement in the hip, but this was not an indication for operation in the absence of intractable pain. Operation was not advised merely because cysts had been detected radiologically.

Fourteen other femoral heads, excised in the treatment of fracture of the neck of the femur, have also been examined for the presence of cystic lesions. In ten the head was resected as a primary measure, and in the remaining four because the fracture showed no signs of union two or more months after injury; one femoral head had undergone avascular necrosis. The average age of the patients with a fracture of the femur was seventy-three years and of patients with osteoarthritis sixty years.

The excised femoral heads were radiographed in at least two planes at right angles. The severity of the osteoarthritic changes was graded in the manner suggested by Collins (1949) and all the cases were regarded as being Grade IV on the grounds of extensive loss of cartilage, osteophyte formation and eburnation, often accompanied by flattening of the articular surface. The specimen was then sawn into slices 0.5 to 1.0 centimetre thick and these were radiographed in series. In one case we injected a cyst with 40 per cent lipiodol before section and its extent and ramifications are shown in Figure 4. Histological examination was carried out in each case. The bone slices were fixed in 10 per cent formalin, decalcified and embedded in paraffin in the usual manner. Sections were cut at 10–20 micra and stained with haematoxylin and eosin. Specific features were investigated by Mallory's stain for connective tissue, Mayer's stain for mucin, and Weigert's fibrin stain, and Gomori's method was employed to detect haemosiderin. Serial sections were employed to a very limited extent.

RADIOLOGICAL OBSERVATIONS

In our experience cysts are detected pre-operatively in the hips of most, but not all, patients with advanced osteoarthritis. There appears to be considerable variation in the amount of cyst formation and in clinical radiographs it is often difficult to estimate with any exactness their number, extent and size. Most of the cysts in the femoral head are small and are situated close to the articular surface. Less common are the larger cysts which sometimes extend into the femoral neck (Fig. 1). We have found no correlation between the amount of cyst formation and the duration of the disease.

Radiographs of the excised but still intact femoral head provide little additional information about the cysts; occasionally they reveal cysts that had not previously been detected. On the other hand, radiography of the series of slices of the femoral head is an excellent method of examination. Typical examples of such radiographs are seen in Figures 2 (this femoral head is shown pre-operatively in Figure 1) and 6, which are representative of our series. By this method we have demonstrated cystic lesions in every patient with primary osteoarthritis.
Small subarticular cysts, many too small to be detected pre-operatively, are surprisingly common, while larger cysts—some of unsuspected magnitude—are more clearly visualised. In the six patients with secondary osteoarthritis, small cysts were seen in each femoral head although none had been detected in radiographs taken before operation. No cyst was demonstrated either before or after operation in the patients with fractures of the neck of the femur.

**CORRELATION OF THE RADIOLOGICAL AND MACROSCOPICAL APPEARANCES**

In advanced osteoarthritis the femoral head is characteristically deformed (Harrison, Schajowicz and Trueta 1953) (Fig. 2, section 5). Most of the central part of the articular surface is denuded of cartilage and the cysts occur beneath the exposed bone (Figs. 2 to 7). We have not encountered a cyst beneath intact articular cartilage, and only rarely in the infero-medial osteophyte. The surface bone overlying the cysts is eburnated and sclerosed, particularly on the superior aspect of the head. It is often stippled with minute pits, most of which end blindly just below the surface, although a few perforate the superficial bone overlying a radiological lesion. Sometimes there are small islands of cartilage in the denuded area, and on section this cartilage is seen to plug a defect in the surface bone, commonly overlying a larger defect in the underlying trabeculae. These fragments of cartilage are sometimes assumed to be a survival of the original layer, but histological examination clearly shows that the great majority are formed by metaplasia during repair of local damage to the bone.

The macroscopic appearance of the cysts shows great variation. Most are small, roughly spherical defects in the sclerosed subarticular bone (Fig. 2, section 4). The larger lesions are
A large irregular cyst in cross-section. It is covered by a thin layer of denuded articular bone. The radiograph of this lesion is shown in Figure 2, section 4. Note that small subarticular cysts are more clearly visualised by radiography. Occasionally bloodstained. Usually the greater part of the bony defect is occupied by such loculi, but in our experience there is no correlation between the extent of the defect in the bone and the size of the encysted loculi of fluid. Many loculi of fluid are spherical or less often spherical (Figs. 3 to 7) and may pursue an oblique course towards the articular surface. Even when there is such extensive destruction of the trabecular system as is shown in Figures 2 to 7 a thin plate of bone is usually present at the overlying surface. Histological examination frequently shows that this bone is in fact necrotic but we have been unable to identify necrotic bone in radiographs of the slices.

The defect in the trabecular system is occupied by connective tissue which usually, but by no means always, contains one or more loculi (Figs. 3 to 7) filled with thick, mucoid fluid that is occasionally bloodstained. Usually the greater part of the bony defect is occupied by such loculi, but in our experience there is no correlation between the extent of the defect in the bone and the size of the encysted loculi of fluid. Many loculi of fluid are spherical or less often spherical (Figs. 3 to 7) and may pursue an oblique course towards the articular surface. Even when there is such extensive destruction of the trabecular system as is shown in Figures 2 to 7 a thin plate of bone is usually present at the overlying surface. Histological examination frequently shows that this bone is in fact necrotic but we have been unable to identify necrotic bone in radiographs of the slices.

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FIG. 8
A focus of recent damage at an early stage of repair. Note the disrupted cartilage and bone at the surface (a) and the fractured trabeculae in the deeper part of the lesion (b). The lesion is filled with a highly cellular granulation tissue similar to that produced at the site of fracture. (H. and E., × 85.)

FIG. 9
The same focus as in Figure 8 but at a different level. Note the presence of large, darkly staining osteoclasts in relation to the spicules of fragmented bone (a) and the proliferation of undifferentiated cells (b) throughout the site of damage. (H. and E., × 130.)
pyriform, as Landells observed, but some conform with the irregular shape of the defect in the bone (Figs. 3 to 7). Some are incompletely partitioned by septa of connective tissue. Occasionally they communicate with the joint cavity through the minute defects observed in the articular surface, but in our experience such communications are rarely found, and the loculi are usually separated from the joint cavity by a thin plate of bone (Fig. 3) or by cartilage occupying defects in the surface plate.

**HISTOLOGICAL OBSERVATIONS**

In the following account our object is to review the nature of the damage to the bone at the sites of cyst formation and to describe the process of repair and the formation of cavities of fluid in the bony defects.

At the site of recent injury to the articular surface the surviving cartilage is disrupted, the subchondral plate is destroyed, and fractures are produced in the subjacent trabeculae (Figs. 8 and 9). Usually, however, such foci of injury are at a more advanced stage of repair by the time the femoral head is excised. Nevertheless the changes that result from repair can usually be interpreted and the extent and character of the original damage assessed from the persisting remnants of necrotic bone, both at and below the surface, and from the disappearance of a segment of the original trabecular system (Figs. 12 and 13).

In the later stages of the disease, when the more superficial bone is sclerosed, injury is likely to produce necrosis rather than fragmentation of this surface bone. The force of impact...
would be transmitted to the weaker underlying bone with resultant disruption and necrosis. This may result in loss of part of the trabecular system beneath an unbroken surface (Figs. 10 and 11). We have been impressed by the similarity of such lesions to those circumscribed defects that contain encysted fluid, where, in our experience, it is also usual to find necrotic bone at or below the surface (Figs. 14 to 16). Arterial thrombosis is sometimes seen within the necrotic segment and may be partly responsible for the development of such necrosis. However, we have seldom found thrombosed arteries proximal to the site of injury, and arteriosclerosis is never severe enough to seem a probable cause of serious ischaemia.

Repair of this bone damage bears a striking resemblance to the changes seen at the site of an imperfectly immobilised fracture. We have not seen a necrotic focus before the onset of repair, presumably because a period of comparative inactivity preceded operative removal of the femoral head. In a focus of recent injury (Figs. 8 and 9) the fragments of disrupted bone are being removed by osteoclasts, and a highly cellular and vascular granulation tissue, histologically similar to that seen at the site of fracture, fills the damaged focus and extends to the overlying surface. From an examination of many lesions at various stages of repair, we have concluded that cells with the potential capacity to form new bone, cartilage, or simply connective tissue are usually formed from the ends of interrupted trabeculae and surface plate, whereas granulation and scar tissue are usually formed from the bone marrow. Most foci of repair contain tissue derived from both sources (Fig. 12), but the relative amount varies in different lesions. Seams of osteoid may develop in the reparative tissue and so give

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Part of the section shown in the top right hand corner of Figure 10. The bone and marrow (a) are necrotic and fractures have developed in the trabeculae (b). Note the absence of fragmentation of the surface. (H. and E., × 85.)
Healing of a focus of damage. The surface is repaired by cartilage (a) arising from fractured trabeculae. Living bone is present at (b) and fractured dead bone at (c). The central zone of greatest bone destruction is filled with oedematous connective tissue derived from the marrow spaces, and contains several loculi of fluid. Note the presence of bone debris in the loculus (d) immediately below necrotic surface bone. (H. and E., ×30.)

Area outlined in Figure 12 to show the presence of fractured necrotic bone at the surface (a); living bone at (b). (H. and E., ×160.)
FIG. 14
A large irregular bone defect containing several loculi of fluid (a). The overlying surface is extensively damaged; a persisting defect is present at (b); new cartilage has replaced the surface plate at (c). (H. and E., × 5.)

FIG. 15
Field outlined in Figure 14. Fibrocartilage (a), derived from surviving fragments of the surface bone (b) occupies most of the breach in the surface. A defect (c) that may connect with the underlying loculi of fluid (d) is still present. Small loculi are seen at (e). (H. and E., × 5.)
Necrotic bone (a) at the surface of the large cyst illustrated at a different level in Figs. 14 and 15. The loculi of fluid (d) and (e) in Figure 15 are seen at (d) and (e). Some of the dead bone has a "moth-eaten" appearance which is probably due to osteoclastic activity, and is surrounded by vascular, oedematous connective tissue. Cartilage (b) has formed from interrupted living bone (c) at the surface. (H. and E., × 65.)

Hyaline cartilage (a) occupying defects in the trabecular system. Note that the cartilage is directly continuous with interrupted trabeculae (b). (H. and E., × 65.)
rise to new trabeculae (Fig. 18) or restore the damaged surface. The new bone is readily identified by its unusual cellularity and by its abnormal internal architecture. Instead of new bone, masses of cartilage may form by a process of metaplasia (Figs. 7, and 12 to 17) and some is converted into scar tissue (Fig. 10). As these illustrations show, the origin of cartilage or connective tissue from injured bone may be inferred when these tissues are in direct continuity with interrupted trabeculae. We believe that the cartilage found at the eroded surface is formed in this way, and is rarely a surviving fragment of the original articular layer. When healing is complete, new bone, cartilage or connective tissue separate the persisting defect in the trabecular system from the synovial cavity.

When dead bone persists in the foci of damage, it is enclosed in scar tissue, or incarcerated in newly formed bone or cartilage. The necrotic surface plate is slowly replaced in some instances by new bone growing in from the margin of the lesion. The amount of osteoclastic activity in relation to the dead bone is extremely variable, and in many long-standing lesions there is little evidence of continued bone removal (Figs. 11 and 16).

There is strong circumstantial evidence that synovial fluid enters the bony defects through a breach in the articular surface (Landells). We have confirmed that a communicating channel is sometimes present between the encysted fluid and the synovial cavity (Figs. 10 and 12) but as we did not study this point by serial sections, we feel that many were probably missed, although others may well be obliterated as the surface is repaired. On the other hand, we have been impressed by our failure to find such loculi when a defect in the trabecular system is covered by an intact surface (Fig. 10), and also at sites of recent injury (Figs. 8 and 9).

The loculi of fluid are surrounded by connective tissue of very varied appearance. In comparatively recent lesions the connective tissue is loose and oedematous (Fig. 12), and contains fibroblasts and capillaries, histiocytes swollen with debris, and lymphocytes. In older lesions more mature connective tissue is present (Fig. 7); it is often condensed around the loculi to form a capsule, and contains many large sinusoidal blood vessels (Harrison, Schajowicz and Trueta 1953) presumably derived from the capillaries formed during repair. In some
lesions the scar tissue is extremely dense, particularly around the loculi of fluid (Fig. 14). In some loculi, strands of necrotic tissue stretch across the cavity and are attached either to the capsule or to the septa from which they appear to originate (Figs. 14 and 15). Traumatic haemorrhages, often associated with thrombosis of small vessels, are common, particularly near the surface of the cysts, and bleeding may consequently occur into the loculi of fluid. However, we found no evidence that haemorrhage plays any part in the production of the cysts (Pommer 1913, Lang 1932, 1934).

We have never seen "bodily displacement" (Landells) of trabeculae around encysted fluid, even at an early stage of repair, and orientation of the trabeculae is not present at this stage (Fig. 12). Around the older cysts however the trabeculae do undergo remodelling and sclerosis (Fig. 14) and new bone sometimes continues to form slowly from seams of osteoid in the marginal connective tissue. This surrounding sclerosis is probably caused partly by pressure transmitted through the encysted fluid, but it may well be further increased by the stress thrown on these marginal trabeculae by the loss of a segment of bone from the weight-bearing area.

**DISCUSSION**

It is the very nature of osteoarthritis to expose the subarticular bone to injury, particularly in the weight-bearing joints. In our histological studies we have emphasised the importance of such injury as the cause of cyst formation during the evolution of the disease.

It is our belief that the shock of jolting movements at the hip is normally absorbed by the protective resilience of healthy articular cartilage and that in osteoarthritis such movements injure the bone at the site of impact and the shock is transmitted to the subjacent trabeculae. The nature and extent of the damage to the bone will depend on the degree of protection afforded by any surviving cartilage, the presence and extent of sclerosis in the subarticular bone and on the severity of the injury. Injury to the delicate subarticular bone before the cartilage is completely lost results, we believe, in the numerous small foci of bone destruction that we have described in the vicinity of the articular surface; the surface plate and the subjacent trabeculae are fractured and even fragmented, and the defect is open to the synovial cavity. At the opposite extreme, in the later stages of osteoarthritis, jolting injuries will affect densely sclerotic bone. In our experience injury at this stage is more likely to produce necrosis with less disruption of the superficial bone at the site of collision; at the same time, however, transmission of the force of impact through the trabecular system is liable to result in fractures and necrosis of weaker trabeculae, deep below the surface.

The presence of cavities filled with fluid at the site of injury is certainly the result of disruption of the articular surface so that synovial fluid is forced into the bone under the pressure of the weight of the body. We agree that the appearance of large defects filled with synovial fluid might reasonably be interpreted in the way suggested by Landells, but an examination of early lesions at different stages of evolution fails to provide evidence that bone destruction or "bodily displacement" of trabeculae is actually caused by an influx of synovial fluid. As we have shown, synovial fluid is not always present at a site of recent injury, even when the surface bone is disrupted, and fluid is absent from sites of bone necrosis when the continuity of the surface is not destroyed. We believe that such observations provide evidence that the bone destruction precedes, and is independent of, the entrance of synovial fluid into the bone, and that if the surface remains intact until the deranged focus is filled with granulation tissue the formation of cavities of synovial fluid will not occur. On the other hand, should synovial fluid enter the necrotic bone, it is likely to disrupt the already devitalised tissue, which may then be swept into the cavity of the joint, leaving a potential space in which synovial fluid becomes encysted during the stage of repair.

Extensive necrosis of the femoral head in osteoarthritis was interpreted by Pridie (1952) as the result of a "vascular catastrophe" and was aptly likened by Collins (1939) to the histological picture of avascular necrosis. The association of cystic lesions with such bone
necrosis was reported by Harrison, Schajowicz and Trueta (1953) who believed that a "series of infarctions" occurs in the "destructive phase" of osteoarthritis. These authors believed that infarction of the bone was caused by "collapse" of the femoral head, and concluded that "ischaemic changes" were a usual occurrence in the late stages of the disease. We agree that ischaemia may play a role in the development of these foci of bone necrosis, but in our opinion it is the result of arterial damage sustained at the time the bone is injured, and collapse of the femoral head is more likely to be the result of extensive bone destruction than its cause.

These sites of fracture and necrosis may become visible radiologically once devitalised fragments of bone have been removed, either by osteoclastic activity or by being shed into the cavity of the joint. In many instances the radiographic defect is perpetuated by the production of cartilage and connective tissue instead of new bone, and by the formation of cavities of synovial fluid. Such imperfect healing, analogous to that at the site of an imperfectly immobilised fracture is, we believe, also caused by lack of rest. Continued use of an injured osteoarthritic hip must result in the repeated transmission of the weight of the body through the defect in the trabecular system. In consequence, healing is likely to be impaired and the cavities of synovial fluid will tend to persist instead of being obliterated by granulation tissue. It is our contention, therefore, that the cysts of osteoarthritis are sites of injury that often, if not invariably, contain encysted synovial fluid, and the persistence of the radiographic defect is an indication that continued use of the joint has impeded the natural processes of repair.

**SUMMARY**

1. An attempt has been made to correlate the radiographic appearances and the morbid anatomy of the cystic changes that occur in the head of the femur in advanced osteoarthritis.
2. The suggestion is made that these lesions are foci of traumatic bone necrosis. Repair may be complicated by the subsequent entrance of synovial fluid through defects in the surface.

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