We present a case in which the growth of an intraosseous cyst arising from the proximal tibiofibular joint appeared to have been increased by polyethylene wear particles from a medial unicompartmental knee replacement. Histological examination of the cyst wall showed a histiocytic response associated with numerous polyethylene wear particles. This case demonstrates that there is a direct communication between the joint cavity and the cyst. Such communication is probably through openings in the articular cartilage large enough to allow the passage of these particles.

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In addition to the destruction of cartilage, pathological changes in subchondral bone are known to play an important role in osteoarthritis (OA). Although the cause of primary OA is not known, increased bone remodelling is a distinctive feature of the condition. The pathogenesis of these bone changes is poorly understood, but they appear to be influenced by alterations in abnormal mechanical forces around the affected joint. Characteristic radiological changes associated with the degeneration of cartilage include narrowing of the joint space, subchondral sclerosis, and the appearance of osteophytes, intra-articular osteochondral bodies and subchondral cysts. Although the last are associated with OA they can develop as primary lesions independent of it.

Previous studies have shown that prosthetic wear particles are associated with the release of bone-resorbing inflammatory mediators such as interleukin-1, interleukin-6 and tumour necrosis factor and are related to osteolytic lesions seen around joint arthroplasties. Few studies, however, have addressed the possible cellular mechanisms of bone resorption which occur in osteoarthritic cysts. Recent work by Jiranek et al indicated that the cytokine and cellular profiles of osteoarthritic cysts resemble those of osteolytic lesions around failed joint arthroplasties. We report a case which suggests that the enlargement of a subchondral cyst is due to the injection of joint fluid associated with the deposition of polyethylene particles. We postulate that polyethylene-associated macrophages in the cyst lining are capable of stimulating local osteolysis in a manner comparable to that occurring in failed joint replacements.

Case Report

A 77-year-old man was first seen with a painful left knee in 1978. Plain radiographs showed medial OA and a well-preserved lateral compartment (Fig. 1). A cystic lesion was noted in the lateral tibial condyle and in the head of the fibula. After failed conservative treatment and progression of his symptoms, a medial unicompartmental arthroplasty was performed in February 1985.

At follow-up 25 months after the operation, he complained of discomfort in the knee. Plain radiographs showed lucencies around the tibial component and expansion of the cystic lesion. Varus angulation was noted at five years and plain radiographs showed progressive lucency around the tibial component, subsidence of the femoral prosthesis and further expansion of the subchondral cystic lesion into the lateral tibial condyle (Fig. 2). By early 1995, ten years after the arthroplasty, the cyst had increased further in size (Fig. 3).

MRI showed a large cyst within the lateral tibial condyle, extending to the subchondral cortex posteriorly and to the proximal tibiofibular joint. There were some small cysts in the head of the fibula. Radiographs showed the cyst to have
the appearance of a giant-cell tumour or an aneurysmal bone cyst. A fine-needle biopsy was performed and histological examination indicated that it contained mucinous material mixed with a few inflammatory cells. Reactive cartilage and bone were seen in the cyst wall which was lined by fibrous tissue. There was no evidence of pyrophosphate crystals in the articular tissues or the subchondral cyst.

In March 1995, the patient had a revision to a total knee replacement. The femoral component was found to be loose but the tibial implant, despite its proximity to the cyst, was secure. The cystic lesion was curetted and the defect filled with cement. Histological examination of the synovium and the pseudomembranes from the femur and tibia showed a heavy foreign-body reaction, with the presence of numerous histiocytes and giant cells in response to polyethylene particles. Examination of the cyst lining also showed a histiocytic response associated with the presence of polyethylene particles which were identified by their strong birefringence under polarised light (Fig. 4).

Discussion

There are two main theories which attempt to explain the development of osteoarthritic cysts. The first is based on the idea that synovial fluid intrudes through the articular cartilage, resulting in hydraulic destruction of subchondral bone. Such intrusion is believed to be due to raised pressure of the intra-articular fluid which forces joint fluid into cancellous bone through gaps in degenerate articular cartilage. This theory is supported by the presence of defects in the articular cartilage over cysts, of fragments of articular
cartilage within cysts and the similarity of cyst fluid to synovial fluid.\textsuperscript{11}

The second view suggests that a localised area of subchondral necrosis of bone results from repetitive microtrauma which leads to cystic degeneration in bone with the articular cartilage being left intact.\textsuperscript{12,13} This is based on evidence of bony contusion, trabecular fracture and primary subchondral osteolysis which may, subsequently, communicate with the joint if the overlying articular cartilage and subchondral bone plate cracks.\textsuperscript{13}

In our case, the finding of polyethylene particles within the wall of the subchondral cyst supports the theory of intrusion of joint fluid. The popliteus bursa communicates with the knee and sometimes also with the proximal tibiofibular joint.\textsuperscript{14} This pathway may explain the migration of polyethylene particles into the cyst in our case and such communication with joints may provide a path for polyethylene particles to migrate large distances.

We postulate that, in a manner similar to aseptic loosening of joint prostheses, inflammatory mediators including cells and cytokines, are found within a bone cyst and induce an osteolytic reaction. This suggestion is supported by the work of Jiranek et al\textsuperscript{15} who showed that the process of bone resorption in osteoarthritic cysts involved a cellular profile and cytokine production similar to that seen in erosive osteolytic lesions which occur around failed joint prostheses. We presume that the cells of the cyst wall contribute to the osteolytic activity, either by direct release of soluble factors which stimulate bone resorption or by differentiating into bone-resorbing cells. Furthermore, this process is exaggerated by the presence of wear particles.

In our case, since polyethylene particles were found within the cyst, we conclude that there is direct communication between cysts and the joint space. These pathways are large enough to allow the migration of polyethylene particles of varying size. Our case illustrates how cysts can grow in the presence of wear particles and emphasises the importance of dealing with them at the time of the initial operation.

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References


