Deposition of calcium pyrophosphate dihydrate (CPPD) crystals in the pseudocapsule, femoral and acetabular membranes and periprosthetic tissue at revision of 789 cases of failed total hip replacement. In 13, periprosthetic tissues were found to have deposits of CPPD crystals in areas of cartilaginous metaplasia; four also showed evidence of localised deposition of amyloid. None of the patients had a history of chondrocalcinosis in the hip or other joints. Cartilaginous metaplasia and other changes in periprosthetic tissues may predispose to the deposition of CPPD and associated localised amyloid.

Deposition of crystals of calcium pyrophosphate dihydrate (CPPD) occurs mainly in the hyaline cartilage and fibrocortilage of joints as well as in other articular and periarticular tissues. In the pseudomembrane, which lies between the prosthesis and bone in failed hip replacements, there is often abundant degenerate cellular and collagenous connective tissue including occasional areas of fibrocortilaginous metaplasia. Similar changes are seen in the pseudocapsule which surrounds a joint arthroplasty. These tissues also have a heavy foreign-body macrophage and giant-cell reaction to polymeric and metal wear particles. Deposition of crystals of CPPD has not previously been reported in these periprosthetic tissues.

We have therefore examined retrospectively specimens of tissues of hip revision arthroplasty collected over a three-year period for histological evidence of the deposition of CPPD crystals. We documented the clinical and implant details of those cases containing CPPD crystal deposits and determined histologically tissue changes associated with the deposition of CPPD crystals.

Patients and Methods

The periprosthetic tissues including pseudocapsules and femoral and acetabular pseudomembranes of 789 cases of failed hip replacement were available for examination. They had been collected over a three-year period.

The material was processed routinely to produce 5 µm paraffin-embedded tissue sections. All were stained with haematoxylin and eosin. In the three which contained CPPD crystals further staining with Toluidine Blue and Congo Red was carried out to detect cartilaginous metaplasia and deposition of amyloid, respectively. We reviewed the case notes, radiographs and previous histological findings of the CPPD-positive patients.

Results

Tissue from 13 of the 789 cases of failed hip replacement contained CPPD.

Clinical and radiological findings of the CPPD-positive patients. Table I gives the clinical details of each of these patients, the longevity of the implant and other details of the components and procedures of the arthroplasty. Ten of the implants were cemented and three were uncemented. Two patients had multiple revisions. The mean time to revision was 11.8 years (3 to 20). Ten of the 13 patients were female and the age at the time of primary arthroplasty was 25 to 85 years.

At the time of primary hip replacement none of the patients was known to have evidence of pyrophosphate arthropathy or conditions, other than osteoarthritis, known to be associated with CPPD deposition disease. In ten of the 13 patients the preoperative radiographs were available for review and six patients had radiographs of their knees taken within a year of revision. No radiological features of chondrocalcinosis were noted in any of these radiographs.

Histological findings of the CPPD-positive patients. In nine patients the original histological findings of the femo-
ral head and synovium, obtained at the time of primary hip arthroplasty, were available for review. They showed only the features of the primary joint disorder and there was no evidence of chondrocalcinosis.

All the specimens showed features of aseptic loosening with a heavy macrophage and giant-cell reaction to polymer and metal wear particles within cellular and collagenous connective tissue. Five cases showed deposits within both the pseudocapsule and the acetabular pseudomembrane, and one had crystals in the pseudocapsule as well as in both the acetabular and the femoral pseudomembranes. CPPD crystal deposits were present only in the acetabular pseudomembrane in two patients, only in the femoral pseudomembrane in three and only in the pseudocapsule in two. When a component was not revised no information was available on the membrane from the unrevised component. The exception to this was in the two cases of hemiarthroplasty which were revised (cases 3 and 8, see Table I) and in which acetabular and femoral membranes were studied. The deposits contained abundant positively birefringent short rod-shaped crystals when viewed under compensated polarised light (Fig. 1). With the exception of one case, in which the CPPD deposits were surrounded by macrophages and giant cells (Fig. 2), little or no cellular reaction was seen around these deposits. All periprosthetic tissues containing CPPD deposits showed histological evidence of cartilaginous metaplasia which was confirmed by metachromatic staining with Toluidine Blue. Cartilaginous metaplasia was not always seen in the areas containing CPPD deposits. The periprosthetic tissues of four cases also showed evidence of localised deposition of amyloid as shown by staining with Congo Red and Toluidine Blue. These deposits were generally small and were not found near CPPD crystals. Immunohistochemistry showed only staining for the amyloid P component and none for β2-microglobulin, kappa, lambda, amyloid A protein or prealbumin.
Discussion

Our study has shown that deposition of CPPD crystals occurs rarely in periprosthetic tissues. It was seen in only 13 of the 789 (1.6%) cases examined. None of the patients was known to have a history of CPPD crystal deposition disease in either the hip or other joints at the time of primary or revision arthroplasty. In addition, none had conditions known to be associated with CPPD crystal deposition, other than osteoarthritis. The mean duration of survival of the implant was 11.8 years.

The finding of such deposits in periprosthetic tissues is of interest in terms of the factors thought to promote the accumulation and deposition of these crystals. Previous hip surgery may be a major pathogenic factor in this process since it has been shown that deposition of CPPD crystals is associated with previous joint injury or surgery.\(^5\) This, in itself, could lead to changes in the extracellular matrix, particularly in the composition of the glycosaminoglycans of connective tissue which are thought to predispose to deposition of CPPD crystals. Since they are known to occur more commonly in cartilaginous tissues,\(^6\) it is not perhaps surprising that CPPD deposits were found in periprosthetic tissues containing evidence of fibrocartilaginous metaplasia. Cartilage cells have been shown in vitro to be capable of producing these crystals.\(^6\) It should be noted, however, that not all deposits of CPPD crystals were found in areas of cartilaginous metaplasia. Other features commonly seen in periprosthetic tissues which may have predisposed to the formulation and deposition of CPPD crystals include the presence of iron, associated with areas of haemorrhage or deposition of haemosiderin, and of metal ions within implant-derived metal wear particles.\(^5\)

Small localised amyloid deposits are commonly found in association with CPPD crystal deposits in the knee and elsewhere,\(^7\) but amyloid is an unusual finding in periprosthetic tissues, except in the context of B2-microglobulin dialysis-associated amyloid disease.\(^7\) As in other articular tissues in which localised amyloid and CPPD crystals are found together, it would appear that similar changes in articular connective tissues favour deposition of both of these components.

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References