Failed hip prostheses in hemodialysis patients

Amyloid deposition at the bone-implant interface in 4 cases

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4 hemodialysis patients with failed bipolar hip prostheses underwent 6 revision arthroplasties. Their average age was 55 years, the average duration of hemodialysis was 14 years and the average interval from the primary arthroplasty to revision was 7 years. The interface membranes revealed amyloid deposits in all specimens. There were few polyethylene wear particles, and cement debris or foreign body reactions were rare. It appears that amyloid may cause osteolysis and early prosthetic loosening.

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Avascular necrosis of the femoral head associated with steroid therapy and pathological fractures of the femoral neck due to metabolic bone disease are common in patients on hemodialysis. There is also a high incidence of prosthetic loosening when compared to patients who are operated on for arthrosis for unclear reasons (Naito et al. 1994). The exact mechanism is unclear.

Amyloidosis is a common complication of hemodialysis which may aggravate conditions such as destructive spondyloarthropathy (Kuntz et al. 1984) and cause pathological fractures of the femoral neck (Campistol et al. 1990). The relationship between amyloid deposition and loosening of prostheses in hemodialysis patients has not been investigated. We examined the interface membranes in hemodialysis patients with failed hip arthroplasties.

Patients and methods

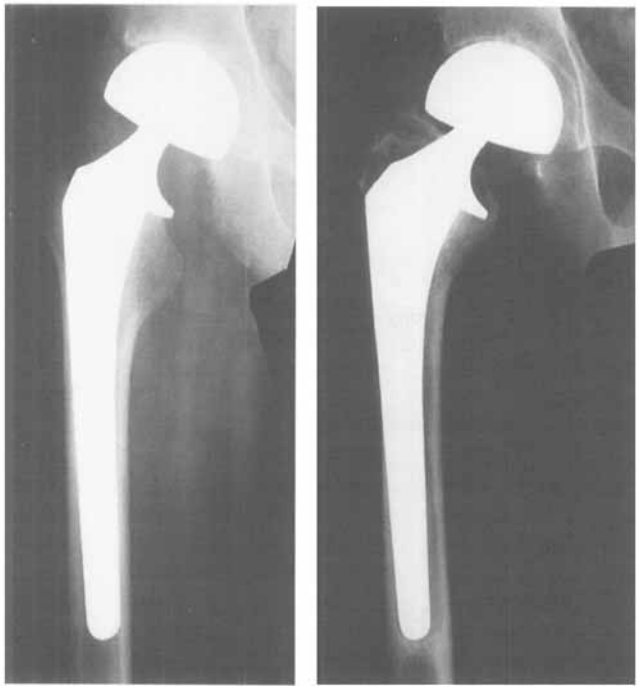
Between 1991 and 1995, 4 patients who were on hemodialysis for chronic renal failure underwent 6 revision arthroplasties because of femoral component loosening after insertion of bipolar hip endoprostheses (Table). All of the patients were men, aged 34–65 years, who had been on hemodialysis for 12–17 years. The interval from the primary arthroplasty to revision ranged from 4 to 8.5 years. The primary diagnosis was avascular necrosis of the femoral head in 4 hips (2 patients) and femoral neck fracture in 2 hips (2 patients). 5 femoral components were implanted without cement and 1 with cement (Table).

The indications for revision surgery included pain, component migration and severe osteolysis around the prosthesis. Septic loosening was ruled out by CRP, ESR and cultures of aspirates at revision. All 5 cementless prostheses subsided more than 2 mm within 2 years of the primary arthroplasty (Figure 1).

Clinical data on 4 patients

Case	Age	Sex	Years of hemodialysis	Years between primary arthroplasty and revision	Primary diagnosis	Type of fixation, Femoral component
1	59	M	17	7.5	Femoral neck fracture	Cemented, Harris precoated
2	34	M	17	8.5	Avascular necrosis	Cementless, Omnifit
	34	M	17	8	Avascular necrosis	Cementless, Omnifit
3	63	M	12	8	Femoral neck fracture	Cementless, Bateman
4	65	M	15	4	Avascular necrosis	Cementless, Omnifit
	65	M	15	4	Avascular necrosis	Cementless, Omnifit

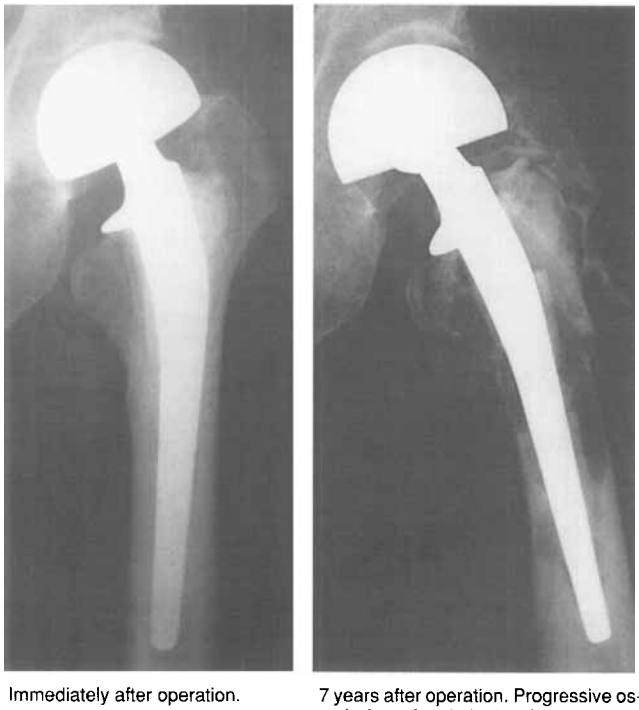
Figure 1. Radiographs showing aseptic loosening after insertion of a cementless bipolar endoprosthesis.



Immediately after operation.

8 years after operation. Subsidence and linear osteolysis.

Figure 2. Radiographs showing aseptic loosening after insertion of a cemented bipolar endoprosthesis.



Immediately after operation.

7 years after operation. Progressive osteolysis and pathological fracture.

In the patient with a cemented component, noticeable osteolysis of the proximal femur developed 6 years after the primary arthroplasty (Figure 2). All patients showed proximal migration of the prosthetic head or osteolysis in the acetabulum.

Interface membranes in the femur, pseudocapsule and synovial tissue proliferating in the acetabulum were collected at revision arthroplasty, fixed in 10% formalin and embedded in paraffin. Sections were stained with hematoxylin-eosin and with Congo red to identify amyloid. In case 2, sections were stained with daylon instead of Congo red and were also examined immunohistochemically by the streptavidin-biotin complex (SABC) technique (Hsu et al. 1981, Milios and Leong 1988) to identify beta-2-microglobulin. The surface of the loose components was also examined under a scanning electron microscope (SEM) for evidence of wear and corrosion.

Results

In all patients, light microscopy showed that the interface membrane in the femur contained a homogeneous acellular substance which stained with Congo red (or daylon), indicating the presence of amyloid (Figure 3). Most of the interface membranes were composed of hyalinized fibrous tissue and contained only a few histiocytes or foreign-body giant cells showing phagocytosis. Cement debris and wear particles were rare. In case 2, immunohistochemistry revealed the presence of beta-2-microglobulin in the interface membrane, which is the major protein constituent of amyloid deposits caused by hemodialysis (Figure 4).

Macroscopically, there was no surface damage to any of the retrieved components, including the polyethylene-bearing insert, outer surface of the head or femoral component. In addition, SEM showed no evidence of wear caused by polyethylene particles of the surface of the components.

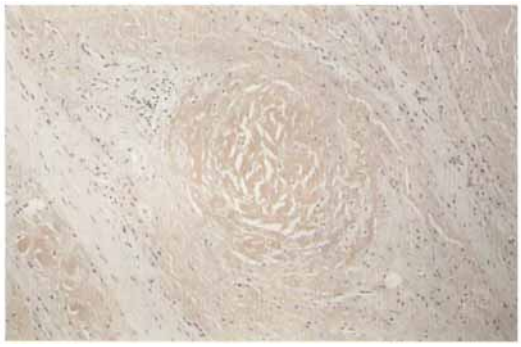


Figure 3. Bone-implant interface membrane showing amyloid deposits (daylon ×40).

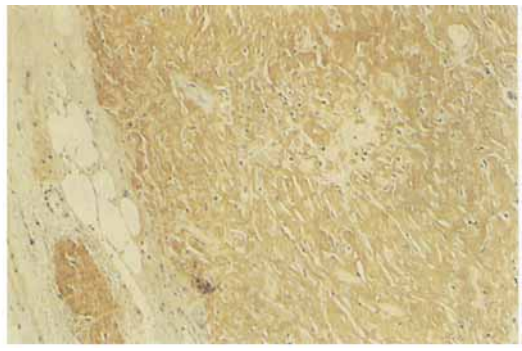


Figure 4. Bone-implant interface membrane. Amyloid was found to be consistent with beta-2-microglobulin (SABC technique ×100).

Discussion

The outcome of hip arthroplasty in patients on hemodialysis is worse than that for other patients. Naito et al. (1994) reported that the loosening rate was one third at an average of 5 years after the operation. Naito et al. (1994) and Radford et al. (1989) attributed the high incidence of loosening to a decrease in bone quality caused by systemic metabolic abnormalities, but the exact cause remains unknown.

We found amyloid deposits in the interface membrane in all our 6 cases, while there were few histiocytes and giant cells containing particulate debris. SEM also showed little evidence of wear. These findings suggest that there may be a biological cause of loosening associated with amyloid deposition, which is special to hemodialysis patients.

There are reports on an association between amyloid and osteoarthropathy in hemodialysis patients (Kurer et al. 1991, Ayers et al. 1993). Campistol et al. (1990) hypothesized that amyloid deposits in the bone matrix weaken the bone structure and predispose to pathological femoral neck fractures.

Normally, amyloid deposition is seen in joints or in the juxtaarticular area,while deposition in the trabeculae or the marrow of tubular bones is an uncommon finding (Onishi et al. 1991). According to Ayers et al. (1993) and Campistol et al. (1990), amyloid deposition occurs by contiguous progression from the synovial fluid to the synovial membrane, the cartilage, and then the subchondral bone. Schmalzreid et al. (1992) suggested that joint fluid may penetrate along the interface between a prosthesis and the bone, and may enter the periprosthetic tissues. We suggest that amyloid deposits may progress from the periarticular area to the distal femur via the bone-implant interface.

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