Unpredictable progression of osteolysis following cementless hip arthroplasty

24 femoral components followed for 6–10 years

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We reviewed 24 hips that developed femoral osteolytic lesions following cementless total hip replacement with a Porous-Coated Anatomic prosthesis after a mean of 8 (6–10) years. 15 of the hips showed hardly any radiographic deterioration in the osteolysis. 2 became much worse with dramatic loss of bone stock. Both of these hips required revision of the acetabular component as well as bone grafting of the otherwise well-fixed femoral component. The remaining 7 hips showed mild-to-moderate enlargement of the lesions. All 24 hips were asymptomatic.

Blood tests and bone scintigraphy were of no predictive value in assessing the probable progression of the lesions. Serial radiography remains the cornerstone of the monitoring of osteolysis, following cementless hip replacement, the extent of bone loss being usually far greater than indicated by radiographic examination.

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Osteolysis following cementless total hip replacement has been reported by Maloney et al. (1990), Santavirta et al. (1990) and Smith and Harris (1995). However, the location of the lesions and the time for their appearance differ and the association with prosthetic loosening is not clear. The natural history of these lesions has not been documented and there are no guidelines for the timing of surgical intervention.

We have previously reported on localized femoral osteolysis occurring in 24 of 104 hips 3 or more years after cementless hip replacement (Learmonth et al. 1995). All these patients were asymptomatic and were therefore treated expectantly. This paper reports on the review after a further 3-year interval.

Patients and methods

We reviewed 24 hips in 21 patients that had developed femoral osteolytic lesions following replacement with a Porous-Coated Anatomic (PCA) prosthesis (Howmedica, Rutherford, New Jersey) (Table 1).

All hips had a 32-mm chrome cobalt head and a single assembly acetabular component with an outer diameter of 52 mm or less. The mean follow-up after the primary replacement was 8 (6–10) years. All patients were assessed clinically according to the Charnley modification of the D'Aubigné and Postel scoring system (Charnley 1979). Anteroposterior and "turned" lateral radiographs taken annually and at the time of review were analyzed. Progression of granulomas was broadly classified, according to the percentage increase in the size of the granuloma, as mild (< 100%), moderate (> 100%) and aggressive (> 500%). An attempt was made to assess polyethylene wear by radiographically determining the ratio of the thickness of the plastic, superior and inferior to the head. A full blood count, sedimentation rate and a technetium bone scan were carried out on the 2 hips that exhibited markedly expanding lesions.

Results (Table 1)

Femoral lesions occurred in Gruen zone 7 (Gruen et al. 1979) in all hips, while zone 1 was also involved in 3 patients. There was no radiographic evidence of loosening of any femoral component. All but one of the hips showed evidence of progression of the osteolytic process over the 3-year period. 7 of the granulomas exhibited moderate progression: in 5 of these, the outer diameter of the cup was 46 mm, corresponding to a plastic thickness of 3.5 mm or less.

In 2 hips, aggressive progression necessitated revision of the acetabular component and additional bone grafting of the proximal femur (Figure 1). The full blood count, sedimentation rate and bone scintigraphy in these 2 patients were unremarkable. Bacterial

Table 1. Observations

No	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X
1	50	M	R	A	7.2	B	6	5	6	32	46	32	3	7	10x5	14x8	124	+					82	
2	41	F	L	O	5.7	A	6	6	6	48	49	32	6	7	2x2	3x2	50	(+)					89	
3	38	M	R	O	8.8	A	6	6	6	36	49	32	6	7	12x5	16x4	3	(+)					87	+
4	42	M	R	A	7.5	B	5	4	5	44	46	32	3	7	15x8	33x8	120	+					88	
5	36	F	R	A	6.3	B	5	5	6	45	49	32	6	7	20x5.5	27x9	120	+					76	
6	36	F	L	A	5	B	5	6	6	58	52	32	9	7	8x4	12x4	50	(+)					80	
7	49	F	R	R	6.9	C	6	3	3	28	49	32	6	7	4x2	6.5x2	63	(+)					84	
8	89	F	R	R	7.6	B	6	5	6	40	46	32	3	7	10x6	25x10	316	+					74	+
9	25	M	R	A	7.1	B	6	5	6	43	49	32	6	7	8x5	13x5	63	(+)					87	
10	50	M	L	R	6.6	B	6	5	5	42	52	32	9	7+6	8x8	10x9	40	(+)					80	
11	43	F	L	R	7.6	B	3	3	3	44	49	32	6	7	25x12	100x25	671	++					84	
12	43	F	R	R	7.9	B	6	5	5	40	49	32	6	7	8x4	9x4	13	(+)					85	
13	28	M	R	A	6.8	B	6	5	5	42	49	32	6	7	12x4	13x4	13	(+)					85	
14	28	M	L	A	6.6	B	5	4	5	38	52	32	9	7	7x4	7x5	25	(+)					89	
15	50	F	L	O	8.9	A	5	6	6	56	52	32	9	7	7x4	8x4	14	(+)					82	
16	23	M	R	T	9.6	A	6	6	6	35	49	32	6	7+1	8x5	18x20	800	++	1	24x12	55x22	320	87	+
17	60	F	R	O	9.8	B	5	6	6	45	46	32	3	7+1	8x5	18x5	125	+	1	40x10	60x20	200	80	+
18	54	F	R	A	7.2	B	6	6	6	42	46	32	3	7+1	20x8	33x10	106	+	1	25x15	36x22	164	81	
19	63	F	L	O	6.8	B	6	4	5	40	49	32	6	7	16x9	17x10	18	(+)					77	
20	51	F	L	O	8.8	A	5	6	5	47	52	32	9	7	15x4	20x8	167	+					86	+
21	48	F	R	O	8.2	A	6	5	5	50	49	32	6	7	4x3	4x2	–33	(+)					82	
22	46	M	L	O	7.9	A	6	5	6	34	52	32	9	7	2x2	3x2	50	(+)					83	
23	29	M	L	A	6.8	B	5	5	6	50	52	32	9	7	10x4	13x3	3	(+)					77	
24	53	M	R	O	8	A	6	5	6	50	52	32	9	7	15x5	15x6	20	(+)					90	

A Age
B Gender
C Side
D Primary diagnosis
  R Rheumatoid arthritis
  A Avascular necrosis
  O Osteoarthrosis
  T Trauma
E Follow-up (years)
F Charnley grouping
Charnley Hip Scores
G Pain
H Movement
I Walking
Acetabulum
J Inclination (degrees)
K Outer diameter (mm)
L Inner diameter (mm)
M HDP thickness (mm)
  (approximate mean)
Granulomas
N Gruen zone
O Original measured size (mm x mm)
P Size after 3 years (mm x mm)
Q % increase in size over 3 years
R Classification
  (+) mild
  + moderate
  ++ aggressive
S Gruen zone 1
T Original size
U Size after 3 years
V % increase in size
Femur
W % fit and fill in femoral metaphysis
X HDP measurable wear

cultures taken at the time of revision were negative. Histology revealed a foreign body granuloma with refractile particles in one patient. The other (Figure 1) showed no birefringence under polarized light, but revealed eosinophilic debris and hemosiderin deposits suggestive of hemarthrosis. This lesion had breached the medial cortex to form a tense encapsulated cyst invading the adjacent soft tissues.

Discussion

The pathogenesis of periprosthetic osteolysis following cementless total hip replacement remains obscure. The pseudosynovial membrane invariably present at the bone-prosthesis interface has been shown to have bone-resorbing activity (Ohlin et al. 1990). The cytochemical response culminating in bone loss is thought to be activated by the ingestion of particulate debris (Murray and Rushton 1990, Santavirta et al. 1990). The precise load of wear debris generated clearly differs in each patient, but in many the volume must be similar. Therefore this does not explain why some patients develop osteolysis, while others do not. Indeed, in our series the amount of polyethylene wear did not correlate with the progression of osteolysis. 5 hips had radiographically demonstrable wear with eccentricity of the head within the cup. 3 of these showed moderate and 1 minimal progression of the osteolytic lesion. It is unlikely that hypersensitivity plays a role, as the sedimentation rate and the differential white blood count were normal in all our patients, and the contralateral hip of the one patient who displayed aggressive progression had a non-progressive lesion in zone 7 (Figure 1) in the contralateral hip.

It thus seems likely that other factors, such as genetic predisposition, may make an important contribution to the development of osteolysis. The clinical problem is to predict whether a seemingly benign lesion may become aggressive and rapidly destructive, as occurred in 2 of our patients.

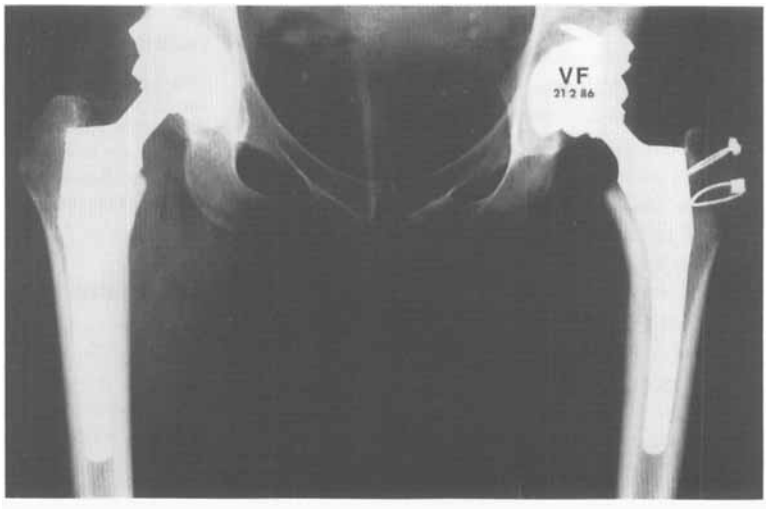
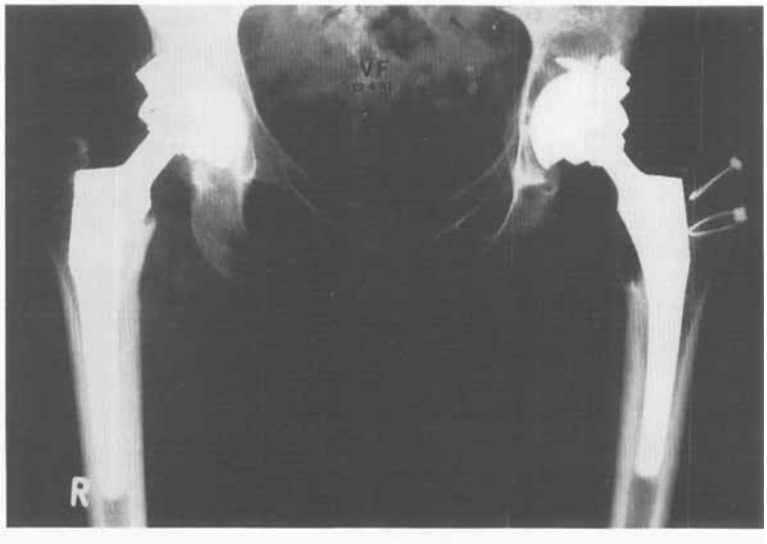
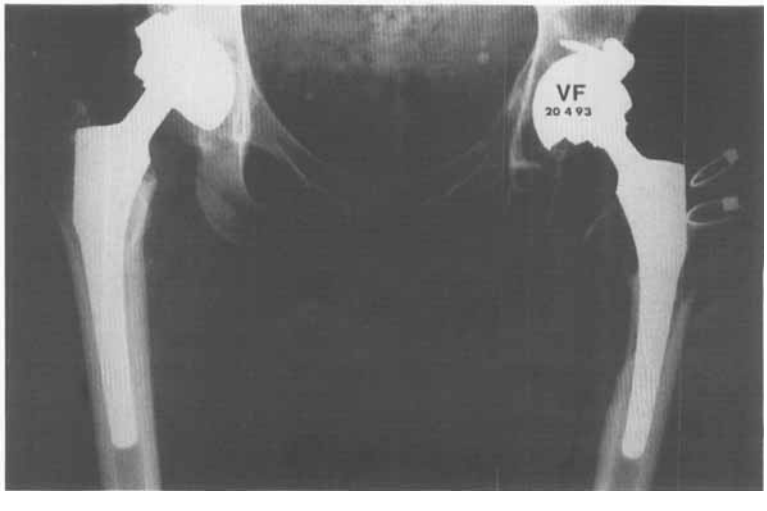


Figure 1. 36-year-old woman with systemic lupus erythematosis, who had bilateral Porous-Coated Anatomic total hip replacements for avascular necrosis.

Early post-operatively



Large osteolytic lesion present in Zone 7 of the left hip 5 years later. This had remained static for 2 years. Much smaller lesion noted on the right.



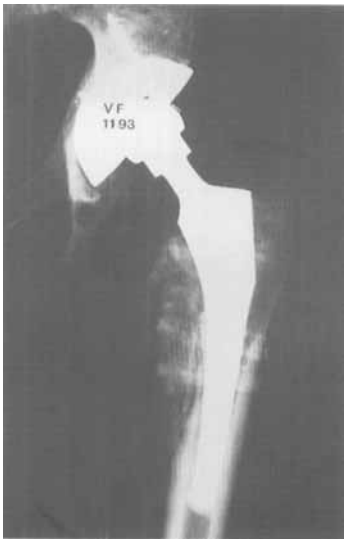
Aggressive progression of the lesion on the left, with considerable loss of bone stock. The lesion on the right has remained unchanged.

The technetium bone scans of both hips that converted to rapidly progressive destruction were normal. This could, perhaps, have been predicted since these are essentially osteoclastic lesions.

Tanzer et al. (1992) reported on 20 hips that had "femoral endosteal cortical erosions" following cementless total hip replacement that were followed up from 20 to 53 months. 12 of the femoral components were shown to be loose. None of the femoral components in our series was clinically or radiographically loose. Smith and Harris (1995) reported progression of osteolysis in 15 of 29 hips. They also noted that the clinical ratings were not sensitive to the presence of osteolysis.

It is disturbing that osteolysis can occur and progress, while remaining clinically silent. It is economically impossible to regularly and indefinitely monitor all patients, following total hip replacement. However, we suggest biennial radiographs, if a prosthesis has been used that does not have well documented long-term follow-up studies. Regular radiographic monitoring is then mandatory, if an osteolytic lesion is identified.

Localized osteolysis and loss of bone stock remain a problem following uncemented total hip arthroplasty. At present, there are no markers to identify lesions which will become markedly progressive. Once osteolysis has been identified, all hips must be regularly monitored by serial radiography at 6-month intervals. Excessive or rapid loss of bone stock warrants early revision, even in the asymptomatic patient.



The cup has been revised and a segmental onlay graft has been applied to the otherwise well fixed femoral component. 2 years postoperatively there was good radiographic incorporation of the segmental onlay graft.

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References

Charnley J. Low-friction arthroplasty of the hip. Springer, Berlin 1979: 20.

Gruen T A, McNeice G M, Amstutz H C. "Modes of failure" of cemented stem-type femoral components. A radiographic analysis of loosening. Clin Orthop 1979; 141: 17-27.

Learmonth I D, Grobler G P, Dall D M. Loss of bone stock with cementless hip arthroplasty. J Arthroplasty 1995; 10 (3): 257-63.

Maloney W J, Jasty M, Harris W H, Galante J O, Callaghan J J. Endosteal erosion in association with stable uncemented femoral components. J Bone Joint Surg (Am) 1990; 72 (7): 1025-34.

Murray D W, Rushton N. Macrophages stimulate bone resorption when they phagocytose particles. J Bone Joint Surg (Br) 1990; 72 (6): 988-92.

Ohlin A, Johnell O, Leiner U H. The pathogenesis of loosening of total hip arthroplasties. Clin Orthop 1990; 253: 287-96.

Santavirta S, Hoikka V, Eskola A, Konttinen Y T, Paavilainen T, Tallroth K. Aggressive granulomatous lesions in cementless total hip arthroplasty. J Bone Joint Surg (Br) 1990; 72 (6): 980-4.

Smith E, Harris W H. Increasing prevalence of femoral lysis in cementless total hip arthroplasty. J Arthroplasty 1995; 10 (4): 407-12.

Tanzer M, Maloney W J, Jasty M, Harris W H. The progression of femoral cortical osteolysis in association with total hip arthroplasty, without cement. J Bone Joint Surg (Am) 1992; 74 (3): 404-10.