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Bone and muscle mass after hip rearthroplasty

Controlled CT study of 12 patients

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The cortical bone mineral density (BMD), bone volume, bone mass and muscle volume of the thigh and the cancellous BMD of the distal femur and proximal tibia were quantified by computed tomography (QCT) in 12 patients after hip rearthroplasty due to prosthetic loosening following arthroplasty for arthrosis. Both legs were scanned and the operated side was compared with the healthy side. A control group, 12 patients, had had unilateral hip arthroplasty without subsequent rearthroplasty. There was a 19 percent decrease in bone mass and 13 percent decrease in muscle volume in the middle femur, compared to the contralateral side, in the patients who had undergone rearthroplasty, and a reduction of 9 percent for the same variables in the control group. There was also a more marked osteopenia in the reoperated extremity in the distal femur and proximal tibia compared to the unoperated side

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Bone mineral loss after trauma is a constant phenomenon (Nilsson 1966, Nilsson and Westlin 1969, Ahl et al. 1988, Abbaszadegan et al. 1991). Lindberg and Nilsson (1985) examined 279 patients who had undergone arthroplasty for coxarthrosis and found an increase in fracture rate after the operation compared to matched controls for fractures distal to the femoral implant, but not for other fractures. In a study on femoral fractures after hip arthroplasty (Adolphson et al. 1987), we found an overrepresentation of fracture among those arthroplasty patients that had been operated on the same femur once or twice before. We interpreted this finding as a sign of an osteopenia caused by operative trauma, and we have confirmed that the bone mineral density (BMD) is indeed decreased in the femur and tibia on the arthroplasty side (Adolphson et al. 1992). We have now studied whether aseptic prosthetic loosening and the repetitive trauma of a rearthroplasty cause a more pronounced osteopenia in the reoperated leg, and if this osteopenia is more pronounced than the corresponding muscle hypotrophy.

Patients and methods

This study included 12, otherwise healthy, patients who had been operated on with a cemented total hip arthroplasty (Charnley or Stanmore) because of unilateral arthrosis 11 (6–22) years before the measurements, and who also had been reoperated with a cemented rearthroplasty (Charnley or Lubinus) due to aseptic prosthetic loosening 3 (2–3) years before the measurements (Table 1). Only patients without disability and earlier fractures of the contralateral leg and without hormonal therapy were chosen. Their clinical state was good with unlimited, pain-free walking ability and none of the patients had any radiographic signs of prosthetic loosening after the rearthroplasty.

To distinguish the changes of the rearthroplasty from the original operation or the arthrosis disease, we also investigated the same parameters in a control group; of all the patients who had had hip arthroplasty 11 years before, we randomly selected 12 patients with unilateral arthrosis, who had had an operation of the same type as the patients in the rearthroplasty group (Table 1). These patients had not been reoperated and none underwent hormonal therapy. Their clinical condition was good with unlimited, pain-free walking ability and none of the patients had any radiographic signs of prosthetic loosening.

Bone and muscle were examined by the techniques used in the virgin arthroplasty series (Adolphson et al. 1992). In no patient, neither in the rearthroplasty group nor in the control group, did the femoral prosthesis nor the cement extend to the vicinity of the CT scan of the middle femur.

Statistical analysis

The Wilcoxon signed-rank test and the Mann-Whitney U-test were used. The statistical analyses were performed on a Macintosh® II computer with the sta-

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Table 1. Data for the control leg in 12 patients with unilateral hip arthrosis who have been reoperated due to aseptic prosthetic loosening and 12 non-reoperated patients. Bone mass is calculated as BMD × bone volume. Median (25–75 percentiles)

	Reop group (n 12)		Non-reop group (n 12)	
	Male (n 9)	Female (n 3)	Male (n 6)	Female (n 6)
Age	69 (61–74)	79 (65–80)	73 (68–76)	77 (72–82)
Middle femur				
Bone mass	9.5 (8.5–11)	6.3 (5.5–8.5)	9.5 (8.5–11)	6.6 (6.2–6.8)
Bone volume	8.6 (7.5–8.8)	5.6 (5.5–7.3)	8.1 (7.6–8.9)	5.6 (5.5–6.1)
BMD	1.2 (1.1–1.3)	1.1 (0.9–1.2)	1.2 (1.2–1.2)	1.2 (1.1–1.2)
Muscle volume	90 (80–110)	60 (50–80)	80 (70–90)	60 (50–70)
Distal femur				
BMD	0.19 (0.16–0.24)	0.13 (0.10–0.15)	0.18 (0.15–0.25)	0.18 (0.13–0.24)
Proximal tibia				
BMD	0.15 (0.12–0.19)	0.10 (0.06–0.12)	0.14 (0.09–0.16)	0.12 (0.10–0.14)

Mass (g in a 1-cm thick slice), volume (cm^3) and BMD ($g \times cm^{-3}$).

Table 2. Bone and muscle differences between the operated leg and the control leg in 12 patients with unilateral hip arthrosis who have been reoperated due to aseptic prosthetic loosening and in 12 non-reoperated patients. Bone mass is calculated as BMD × bone volume. Median (25–75 percentiles)

	Reop group (n 12) diff percent	Non-reop group (n 12) diff percent	Difference between reop and non-reop groups percent
Middle femur			
Bone mass	−19[a] (10–26)	−9[a] (6–11)	−10[a]
Bone volume	−17[a] (14–18)	−6[a] (5–13)	−11[a]
BMD	−4 (0–13)	±0 (−3+5)	−4
Muscle volume	−13[a] (7–18)	−9[a] (1–21)	−4
Distal femur			
BMD	−28[a](10–38)	−7[a] (0–15)	−21[a]
Proximal tibia			
BMD	−31[a] (18–44)	−23[a] (8–28)	−8

[a] $P < 0.05$.

tistical package StatView SE + Graphics™. Differences were considered significant at P-values < 0.05.

Results

In the reoperated leg, compared to the contralateral, we found a decrease in cortical bone mass of 19 percent (Tables 1 and 2). Among the control patients there was a corresponding decrease in cortical bone mass of the middle femur of 9 percent. The decrease in bone mass was mainly of the bone volume, the difference in bone mineral density was small and not significant. The muscle volume in the thigh of the reoperated femur was decreased by 13 percent versus 9 percent in the non-reoperated group. The cancellous bone of the distal femur and proximal tibia had the greatest loss, most pronounced in the reoperated group who lost approximately 30 percent at these locations.

Discussion

The lean mass and fat component of the human thigh have been investigated by CT and planimetry in healthy subjects (Häggmark et al. 1978), after knee-ligament repair and plaster immobilization (Ingemann-Hansen and Halkjær-Kristensen 1980, Arvidsson et al. 1986), and in patients with various neuromuscular diseases (Termote et al. 1980). They found the method to be highly reliable and pointed out that measurement of the circumference of the thigh underestimates the true loss in muscle volume due to a substantial increase in subcutaneous fat.

Reports from bone mineral studies after total hip arthroplasty are sparse. Lindberg and Nilsson (1984) measured the bone mineral content in the distal end of the femoral shaft (mainly cortical bone) with single-photon γ-absorptiometry in 46 patients with coxarthrosis treated by total hip arthroplasty. They found an initial postoperative increase and thereafter a 10 percent net loss after a year in women; the men recovered their preoperative bone mineral content. However, they did not measure the BMD longer than 1.5 years after the operation. Steinberg et al. (1991) reported a 28 percent cortical bone loss around the prosthesis assessed by dual-energy x-ray densitometry 1–3 years after total hip arthroplasty. They demonstrated a progressive bone loss that was the result of the operation, not the disease process, and after 7–14 years they found a bone loss of 49 percent. Our measurements were made more distally and we could not confirm these high losses, as our patients in the control group after 11 years had a relative loss of only 9 percent in the middle femur, and not even the loss after rearthroplasty was more than 19 percent.

A possible explanation for the bone loss after hip arthroplasty may be the operative trauma. The greater loss after rearthroplasty may be induced by the extra operation. However, the bone loss in the proximal tibia was at least as high as in the femur which partly contradicts the importance of the operative trauma. Stress-shielding due to a limping walk pattern may be a more plausible cause of the ipsilateral bone loss after hip arthroplasty and especially the greater loss after rearthroplasty due to painful prosthetic loosening. A support for some unilateral stress-shielding still 11 years after arthroplasty is the smaller muscle volume at that time. The increased incidence of femoral fracture after arthroplasty (Lindberg and Nilsson 1985) may be explained by this greater bone loss.

It is interesting to note that the cortical bone loss in the femur is merely a loss of volume. If this occurs in accordance with senile osteopenia with mainly endosteal resorption (Frost 1985), it may have implications for the risk of prosthetic loosening. It may, however, be that the finding of a smaller bone mass among the reoperated patients just reflects an increased risk of prosthetic loosening among the patients who lose more after the original arthroplasty.

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