Autoantibodies to red cells associated with metallosis—a case report

Seigo Nakamura, Yuji Yasunaga, Yoshikazu Ikuta, Kouichi Shimogaki, Norikazu Hamada and Noboru Takata

Department of Orthopaedics and Division of Blood Transfusion Services, Hiroshima University School of Medicine. 1-2-3 Kasumi, Minami-ku, Hiroshima, 734 Japan. Tel +81 82 257-5233. Fax -5234
Submitted 97-02-22. Accepted 97-05-12

A 60-year-old woman had a total hip arthroplasty for arthrosis in 1990, using a dual-bearing hip prosthesis (Omniflex, 52mm outer head, 22mm inner head; Osteonics, Allendale, New Jersey, USA) with acetabular reaming and bone autografting from the femoral head which was fixed with ceramic screws. The femoral stem was of Ti-6Al-4V alloy with a cobalt-chrome-molybdenum femoral head (62–68% cobalt, 27–30% chromium, 5–7% molybdenum and 0–1% nickel). In 1993, radiographs showed proximal migration of the outer head, a radioopaque shadow around it and iliac bone scalloping. The shadow gradually became wider and deeper. In 1994, the patient had severe hip pain and could hardly walk (Figure 1). The hip was revised in June 1995. The blood concentration of chromium was 0.9 μg/dL (normal value<1.0 μg/dL) and that of nickel was 1.9 μg/dL (normal value 0.2~0.8 μg/dL). Hemoglobin was low, 10.4 g/dL. Autoantibodies of unknown origin to red blood cells were detected in serum, although no antibodies to red blood cells had been found in the routine examination at the first operation. During the reoperation, much periprosthetic metallic debris was found. The ceramic screws were broken by direct contact with the outer head of the prosthesis; the inner head was severely abraded and had a cone-like shape, but the outer head was slightly abraded and rounded (Figure 2). The polyethylene in contact with the inner head was severely abraded. We reinforced the lateral acetabulum with another bone autograft from the iliac crest, and a 48 mm Ti-6Al-4V

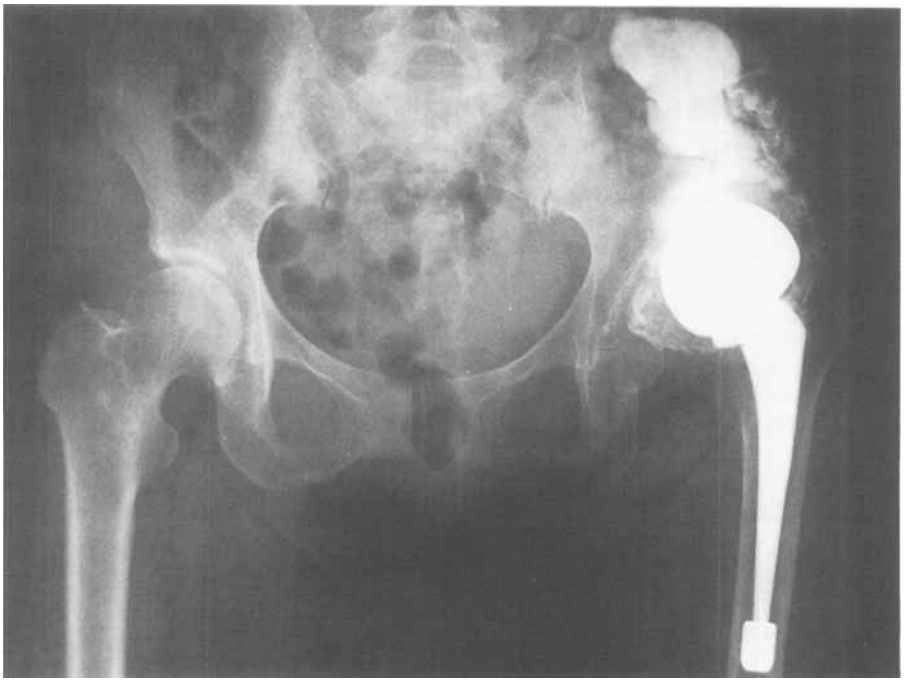

Figure 1. Before revision surgery. There is a radioopaque shadow caused by severe metallosis around the prosthesis.

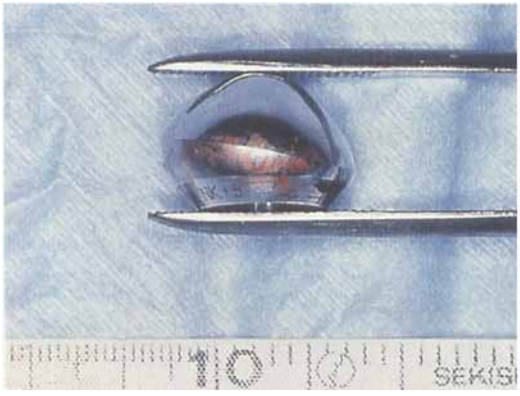

Figure 2. Cobalt-chromium alloy inner head of the prosthesis with severe abrasive wear.

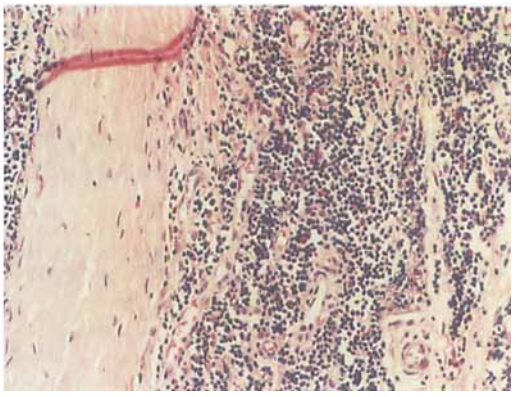

Figure 3. Lymphocytes in the tissue around the prosthesis. (HE ×66).

alloy press-fit cup was fixed with 3 titanium screws to the acetabulum. In the removed tissues, we found metallic debris and necrotic collagen; lymphocytes and eosinophilic polymorphonuclear cells had aggregated near the necrotic tissue (Figure 3). The metal concentration in the tissue was almost identical with the ratio of metals found in the inner head, measured with Inductively Coupled Plasma Atomic Emission Spectroscopy. The titers of haptoglobin were always within normal limits, except immediately after operation. The blood concentration of chromium was 0.8 μg/dL, and that of nickel had fallen to 0.1 μg/dL after operation. Although the autoantibodies to red blood cells had not decreased, the hemoglobin level had increased to 12.5 μg/dL.

Discussion

Matsuda et al. (1992) reported metallosis due to broken ceramic screws in dual-bearing hip prosthesis. Interestingly, the inner head was mainly abraded and this was the main cause of metallosis. Our case showed the same severe metallosis and abrasion of the inner head, which confirms Matsuda's report. The main complication occurred in the space between the inner head and polyethlene, where the ceramic debris could be buried. This space should maintain a perfectly smooth surface for contact lubrication. The patient had a high blood concentration of nickel and a high titer of autoantibodies, which had not been detected at the first operation. Autoimmune hemolytic anemia caused by a metallic implant has not been reported, whereas dermatitis and vasculitis (Types I, III and IV allergies) due to metallic implants have been described (Evans et al. 1974, Elves et al. 1975, Rostoker et al. 1987). Intermediate metallic debris, especially nickel, can act as a hapten and induce immunization (Rostoker et al. 1987). On the other hand, autoimmune hemolytic anemia can be caused by hapten-like activity of penicillin (Foerster 1993). In our case, metallic ions may have induced the autoimmune hemolytic anemia. If so, this is the first report of such a phenomenon.

Acknowledgements

We thank Professor Peter A. Revell, Department of Histopathology, Royal Free Hospital School of Medicine, for his valuable suggestions about this manuscript.

References

Elves M W, Wilson J M, Scales J T. Incidence of metal sensitivity in patients with total hip replacement. BMJ 1975;15 (4): 376-8.

Evans E M, Freeman M, Miller A J. Metal sensitivity as a cause of bone necrosis and loosening of the prosthesis in total joint replacement. J Bone Joint Surg (Br) 1974; 56 (4): 626-42.

Foerster J. Autoimmune hemolytic anaemia. In: Clinical hematology (Ed. Wintrobe M M). Lea & Febiger, Philadelphia 1993; 1170-96.

Matsuda Y, Yamamuro T, Kasai R. Severe metallosis due to abnormal abrasion of the femoral head in a dual-bearing hip prosthesis. J Arthroplasty 1992 (Suppl 7); 7: 439-45.

Rostoker G, Robin J, Binet O. Dermatitis due to orthopedic implants. J Bone Joint Surg (Am) 1987; 69 (9): 1408-12.