Sciatic Nerve Injury in a Renal-Transplanted Patient with Avascular Osteonecrosis

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Dear Sir,

Avascular osteonecrosis affecting weight-bearing bones has been recognized as a frequent complication, associated with corticosteroid therapy, in renal transplantation [1, 2]. We report on a renal-transplanted patient with avascular osteonecrosis of both femoral heads. The patient's condition was aggravated by a severe injury of the sciatic nerve.

A 33-year-old man with end-stage renal disease secondary to malignant hypertension received a renal transplant 9 years ago. He had previously been on haemodialysis for 1 year.

Immunosuppression was started with 1 g of methylprednisolone just before transplant surgery, 60 mg/day of prednisone on a tapering schedule and azathioprine at 125 mg/day. During the first month, the patient suffered an episode of acute rejection and was treated with 15 boluses of 250 mg of methylprednisolone each. The cumulative steroid dose during the two first months was approximately 2.6 mg/kg b.w./day.

Eighteen months after transplantation, bilateral avascular osteonecrosis of the hip was diagnosed, mainly affecting the left joint. The patient was repeatedly evaluated for surgical treatment and transiently put on a non-weight-bearing regimen and prudent anti-inflammatory agents.

Seven years after renal transplantation, the patient was hospitalized with a growing 4-week history of pain and hypoesthesia in the left sciatic nerve territory accompanied by foot-drop.

Detailed neurological and electro-myographic studies showed a severe left sciatic nerve injury at the intrapelvic or pelvic exit level (incomplete axonotmesis). Pelvic magnetic resonance imaging showed further important damage in both hip joints together

Fig. 1. Magnetic resonance imaging shows the emigrated mass (M) compressing the left sciatic nerve (Sc.N.L.).

with a tumoral mass at the sciatic notch level compressing and displacing the left sciatic nerve (fig. 1).

At surgery an orange-sized mass communicating with the very destroyed hip joint was removed. The sciatic nerve was released. The mass removed was composed of a thick fibrochondral wall with a bloody content and abundant necrosed osteochondral and osseous material emigrated from the left joint. The pain disappeared promptly after operation, but the improvement of

neurological deficits in sensitivity and motility remains slow and only partial to date (2 years after the operation).

The association of avascular osteonecrosis and steroid therapy in renal transplant recipients was recognised years ago. Its prevalence varies considerably among transplant centers but is generally around 10% [1]. It is generally accepted that the keystone to the development of avascular osteonecrosis is the

cumulative dose of steroid [2]. Nevertheless, reported series using immunosuppression with cyclosporin A and lower steroid doses have evidenced a sharp decrease in this complication [3, 4].

With established osteonecrosis, conservative management, including the veto on weight-bearing, only controls the symptoms in around 50% patients; the rest should preferably undergo surgery, generally joint

replacement [5, 6]. The poor compliance to non-weight-bearing in our patient and, perhaps, the delay in surgery were obviously the factors that governed the progress of hip destruction, retro-emigration of necrotic material, and the severe injury to the sciatic nerve due to compression and dislocation by the emigrated mass. We have found no reports of similar complications in transplanted patients. Several similar cases of sciatic nerve injury due to the cement used in hip replacement in nontransplanted patients have been described [7]. The neurological improvement in such cases depended on the time elapsed until sciatic neurolysis.

We conclude that when avascular osteonecrosis appears in a transplanted patient, surgery should not be delayed too much, especially in patients whose compliance to rest cannot be expected.

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Book Review

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New Insights in Vertebrate Kidney Function

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This is an excellent, well-edited, well-written adequately illustrated, fascinating book. It contains 14 chapters by well-known authorities dealing with all aspects of vertebral renal physiology, from

reptiles via amphibians to birds. There are also chapters on the comparative physiology of the mammalian nephron, the renal angio-tensin system and volume hemostasis and natriuretic factors in non-mammalian vertebrates. The authors are a 'Who's Who' of renal physiology, and it is difficult to say which chapter is the best, as I am interested in all the vertebrates. However, I personally learned the most from E.J. Braun's chapter on renal function in birds, reflecting my personal interest.

For the curious, the true biologist, this book is a bargain. The editors should be complemented on their excellent editorial work, clearly a labor of love.

G. M. Berlyne, New York

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