Delayed, transient sciatic nerve palsy after primary cementless hip arthroplasty

A REPORT OF TWO CASES

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CASE REPORT

Delayed sciatic nerve palsy is uncommon after primary hip replacement. Two kinds of sciatic palsy have been reported with regard to the time of onset: early palsy related to wound haematoma or lumbosacral nerve elongation which occurs between surgery and 18 days, is more frequent than delayed palsy, occurring between 10 and 32 months, which is usually caused by cement extrusion or heat produced by cement polymerisation.

We present two cases of delayed, transient sciatic nerve palsy arising at three weeks and four months after primary cementless arthroplasty, respectively, without haematoma and with a normal lumbar spine. These palsies were possibly caused by excessive tension from minor limb lengthening of 2 cm to 4 cm required to achieve leg-length equality. As the initial symptoms were limited to calf pain and mild numbness in the foot, surgeons should be aware of this mode of onset, particularly when it is delayed after hip replacement. Both patients recovered fully by 12 months after surgery so we did not undertake surgical exploration of the nerve in either patient.

Sciatic nerve palsy is a well-known but uncommon complication of total hip replacement (THR), with an overall prevalence of less than 0.5%\(^1\-^3\). While immediate post-operative sciatic nerve palsy may occur from time to time, delayed sciatic palsy is rare. Early palsies are usually caused by post-operative haematoma or direct nerve lesions\(^4\-^6\). Schmalzried et al\(^1\) described early sciatic nerve palsies which presented up to 12 days after surgery in eight of 3126 THRs. All were related to a wound haematoma. Delayed palsies at 10 weeks to 32 months were mainly related to cement extrusion or heat produced by cement polymerisation\(^7\-^9\). Very late palsies (7 to 18 years) were related to mechanical failure wear debris compressing the nerve,\(^10\-^11\) migration of a trochanteric wire,\(^12\) or intrapelvic migration of an acetabular component\(^13\).

We report two cases of delayed sciatic nerve palsy, with particular reference to the time of onset and the patient’s symptoms.

Case reports

Case 1. A 24-year-old woman presented with osteoarthritis secondary to Perthes’ disease with a leg-length discrepancy of 4 cm. She had no previous surgery. Her pre-operative Harris hip score\(^14\) was 41 points. The range of movement at the hip was 60° of flexion, 10° of abduction, 20° of adduction, 40° of external rotation, and -20° of internal rotation. Cementless THR was performed through a posterolateral approach. Limb-length equalisation was achieved. The post-operative period was uneventful until 21 days after surgery, when she developed severe pain in the ipsilateral calf, followed by mild numbness in the foot and a limp. She had no history of trauma. Haematoma was excluded by clinical examination. The hip was pain free and the range of movement 100° of flexion, 30° of abduction, 20° of adduction, 20° of external rotation, and 10° of internal rotation. Examination revealed hypoaesthesia of the right foot and paresis of the fibular division of the sciatic nerve. Electrophysiological assessment showed a sub-acute lesion of the proximal lumbosacral plexus (L5), without anomalies suggesting a spinal or radicular lesion. The patient received symptomatic treatment with analgesics and an ankle-foot orthosis. The palsy had recovered completely 12 months after surgery and her Harris hip score was 92 points. The range of movement at the hip was similar to that recorded post-operatively.

Case 2. A 29-year-old man presented with severe osteoarthritis of the left hip with a leg-length discrepancy of 2 cm secondary to neonatal osteomyelitis and septic arthritis (Fig. 1). He had a Harris hip score of 56 points pre-operatively. The range of movement at the left
The hip was 80° of flexion, 30° of abduction, 20° of adduction, 20° of external rotation, and 0° of internal rotation. Before the onset of osteoarthritis, he enjoyed cycling and windsurfing, and wished to return to these activities after hip replacement. Cementless THR was performed through a posterolateral approach (Fig. 1). The post-operative period was uneventful. Cultures of specimens harvested during surgery (bone and synovial tissue) were negative. Limb-length equalisation was also achieved. He was satisfied with the outcome and started windsurfing three months after surgery. His Harris hip score was 98 points and the range of hip movement 105° of flexion, 40° of abduction, 25° of adduction, 30° of external rotation, and 20° of internal rotation. During the fourth post-operative month, he returned with severe pain in the left calf, moderate numbness of the left foot and mild palsy of the foot extensors. Electrophysiological assessment confirmed partial palsy of the fibular division of the sciatic nerve. Fibrillations were evident in the tibialis anterior muscle, the extensor hallucis longus muscle and the short head of the biceps femoris muscle. There was no sensory deficit. Three weeks later, the extensor hallucis longus muscle was already showing improved power. After six months, the neurological symptoms had resolved.

Discussion
The post-operative sciatic palsy which developed in these two patients was of interest because of the delay and mode of onset. Calf pain was the main presenting symptom. Motor weakness and loss of sensation were less marked. There was no history of lumbar spine trauma or pain in either patient and as a result no spinal investigations were performed. Electrophysiological assessment showed clear evidence of a late onset sciatic palsy.

Apart from the possible effect of limb lengthening, we were unable to determine the aetiology of these delayed

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* THR, total hip replacement
neurological complications. There was no evidence of post-operative haematoma, or new lumbar spine symptoms. A cementless prosthesis was used in both patients, excluding thermal injury or compression by cement extrusion. Nerve trauma related to prosthetic failure such as migration or wear debris, usually associated with late palsy, was excluded.\textsuperscript{10,11,13} Direct neurological trauma at the time of surgery was also excluded because of the delay in the onset of symptoms. The posterolateral approach and cementless prostheses carry a risk of post-operative sciatic palsy,\textsuperscript{3,15} but the sciatic nerve was not exposed or sharply dissected during the operations. The probability of limb lengthening as the cause\textsuperscript{1,16,17} was slight as a correction of 4 cm and 2 cm, respectively in the two patients was near the threshold of 3 cm usually identified as a risk factor for post-operative sciatic nerve palsy.\textsuperscript{1,16} The increase of flexion obtained after arthroplasty may have played a role by increasing the effect of limb lengthening. Flexion increased from 60° to 100° in the first patient and 80° to 105° in the second, similar to that obtained in uneventful hip replacements.\textsuperscript{18} Hip disorders during childhood in both patients may have played a part in the onset of the delayed sciatic palsy. Nerve fixation by fibrosis may also be incriminated, but only the second patient had previous surgery. According to Farrell et al.,\textsuperscript{3} most risk factors for the development of motor nerve palsy can usually be identified, but the temporal relationship between minor leg lengthening in these two patients and the delayed onset of palsy is difficult to understand. We found no report in the literature that could explain this complication and no guidelines for the management of such patients. Electrophysiological assessment suggested that the sciatic palsy was caused by excessive traction on the lumbar plexus (L5 root) as the fibular division was injured in both patients. According to Seddon,\textsuperscript{19} complete recovery in six and 12 months suggests delayed axonotmesis. As both our patients recovered completely from their palsy, Sunderland’s classification\textsuperscript{20} indicates that the basement membrane, the perineurium and the epineurium of the axons, would have been spared, which would place the two cases of nerve injury in stage 2.

In the literature,\textsuperscript{4-9} delayed sciatic palsies are mainly related to neurological insult secondary to cement compression or heat produced by methacrylate polymerisation (Table I). The two patients reported here show that cementless hip replacement can also be complicated by delayed sciatic nerve palsy and that mild limb lengthening is a possible factor in such a delayed palsy.

As the initial symptoms were limited to calf pain and mild numbness in the foot surgeons should be aware of this mode of onset, particularly when it is delayed after hip replacement. As both patients recovered fully within 12 months after surgery we did not consider surgical exploration of the nerve.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References