■ CASE REPORT

Neuropathic pain following hip resurfacing due to a transneural suture

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A 60-year-old man developed severe neuropathic pain and foot-drop in his left leg following resurfacing arthroplasty of the left hip. The pain was refractory to all analgesics for 16 months. At exploration, a PDS suture was found passing through the sciatic nerve at several points over 6 cm and terminating in a large knot. After release of the suture and neurolysis there was dramatic and rapid improvement of the neuropathic pain and of motor function.

This case represents the human equivalent of previously described nerve ligation in an animal model of neuropathic pain. It emphasises that when neuropathic pain is present after an operation, the nerve related to the symptoms must be inspected, and that removal of a suture or irritant may lead to relief of pain, even after many months.

Neuropathic pain has many characteristics that differentiate it from other types of pain. Sensory signs may be manifest as negative and positive sensory phenomena, and there may also be motor or autonomic signs. Recent developments in our understanding of the mechanisms underlying the symptoms and signs, and the therapeutic effects of new drugs stem from the development of animal models of neuropathic pain, particularly nerve ligation models.

Case report

In 2004 a 54-year-old man underwent resurfacing arthroplasty of the right hip, with an uneventful post-operative recovery. In May 2008 he underwent left hip resurfacing. Post-operatively, he developed a foot-drop associated with neuropathic pain in his leg and foot. Morphine, paracetamol, gabapentin and tramadol were administered in combination for 16 months, but were ineffective. He was able to mobilise with crutches and the aid of an ankle-foot orthosis.

In September 2009, he was referred to our unit. He had severe disabling neuropathic pain in the distribution of the tibial and common peroneal nerves. The pain was a constant burning sensation, up to ten on a visual analogue scale (VAS). On examination, he walked with a high-stepping gait. Tinel’s sign was absent along the course of the sciatic nerve. He had an MRC grade 5/5 power in tibialis anterior, extensor hallucis longus, extensor digitorum longus and the peronei, with MRC grade 2/5 power in tibialis posterior and grade 3/5 in the long flexors and calf. He had altered sensation with hyperaesthesia in the distribution of the common peroneal nerve. There was diminished cotton-wool and pin-prick sensation in the lateral calf, on the dorsum of the foot and sole. Proprioception was absent in the left great toe, but preserved at the ankle, and vibration sense was absent in the great toe. Thermal thresholds were markedly elevated on the dorsum of the foot, the sural nerve territory and the sole, but normal in the medial calf. Nerve conduction studies showed moderate to severe abnormalities of motor and sensory conduction for the tibial, common peroneal and sural nerves.

We advised exploration of the sciatic nerve. Through a gluteus maximus splitting approach the nerve was identified and found to be tethered. The approach was therefore extended by reflecting the proximal attachment of gluteus maximus. The sciatic nerve was identified distally and followed proximally, where it appeared to be tethered and dragged anteriorly at the level of the greater trochanter. During dissection, a loop of monofilament absorbable (PDS) suture was identified transfixing the nerve. It appeared to weave in and out of the nerve for 6 cm and terminate in a large knot. The suture was carefully released (Fig. 1) and removed, where upon the nerve fell back into its normal anatomical position without ten-
sion. A complete neurolysis was carried out. The majority of the nerve was in continuity and an intra-operative nerve conduction test performed across the level of the lesion showed no response from the common peroneal component, and only a very small response from the tibial component. An indwelling local anaesthetic catheter was placed beside the sciatic nerve proximal to this lesion and left in situ for 24 hours, releasing levobupivacaine 0.125% at 3 ml/h. Following removal of the catheter and mobilisation, the patient was discharged from hospital.

Two weeks later his pain on the VAS had improved to a maximum of two (usually only one) and it was therefore suggested that he gradually reduce the dose of paracetamol, tramadol, and thereafter gabapentin. He was reviewed three months post-operatively, when his pain was one on the VAS and required minimal analgesia. Clinical examination revealed a dramatic improvement of the foot-drop, with MRC grade 3/5 power in tibialis anterior, extensor hallucis longus, extensor digitorum longus and the peroneii, and MRC grade 4/5 in tibialis posterior and the long flexors and calf. Sensory findings were similar to those pre-operatively. Six months post-operatively his pain was still described as one, but he no longer required any medication. He had MRC grade 4/5 power in tibialis anterior, extensor hallucis longus, extensor digitorum longus and the peroneii, with grade 5/5 power in tibialis posterior and the long flexors and calf.

Discussion
Peripheral nerve injury can generate chronic neuropathic pain characterised by a burning sensation, hyperalgesia and allodynia.6 In our case, the damage to the sciatic nerve was caused by a suture. This is an uncommon but recognised complication of hip replacement.7-10 with an overall prevalence of approximately 1% of total hip replacements, including primary and revision procedures.7-10 There are many ways in which the sciatic nerve can be injured during hip arthroplasty:7-10 during exposure; traction during dislocation or after insertion of the implant; by thermal injury from diathermy or polymerising cement; direct damage from a drill or misplaced reamer; pressure from forceps or a retractor; or by external compression, for example a haematoma.

It is difficult to identify the pathological mechanisms of neuropathic pain in the clinic, and in order to investigate these, a number of experimental models were developed.1-4 The Bennett, Chung and Seltzer rat models2-4 are the most commonly studied peripheral mononeuropathy animal models of pain. They mimic human clinical peripheral nerve injuries, and are therefore considered valuable.11 In the Chung model,2 the L5 and L6 spinal nerves are isolated and tightly ligated with a 6/0 silk suture. In the Bennett model2 the sciatic nerve is exposed at the mid-thigh level and four ligatures, spaced 1 mm apart, are tied loosely around the nerve. In the Seltzer model,4 the sciatic nerve is exposed at the level of the upper thigh and partially ligated with an 8/0 silk suture. All these rat models display signs of mechanically and thermally evoked hyperalgesia and ongoing pain.

Our case, with its similarities to these nerve-ligation models, could therefore be considered the human equivalent. Focal nerve injury causes a range of peripheral and central nervous system processes due to local inflammation, nerve degeneration and regeneration, ectopic impulse generation and ephaptic transmission reaction of adjacent tissues. These processes can lead to central changes, including sensitisation and disinhibition, which in some cases may persist and be highly resistant to treatment.

In our case, exploration allowed identification of the cause of the pain and release of the suture. The pattern of recovery in sensory and motor function shows that the majority of the nerve fibres had not degenerated. The underlying cause of the deficit must therefore be the constricting suture. Ochoa, Fowler and Gilliatt12 described prolonged conduction block in such a condition, with recovery of function within six months after release of the constriction, in which the myelin sheath is displaced but has not degenerated. We believe that in our patient the lack of recovery 16 months after hip resurfacing was due to the persistence of the constriction, so severe that the muscle perceived the lesion as deafferentation. As soon as the suture was removed, the condition described by Ochoa et al12 was applicable. The mechanism we describe is a conduction block where the cause is still present, hence its removal promises the possibility of recovery of those nerve fibres that have not yet degenerated.

In conclusion, the presence of continuing severe pain after an operation should raise the possibility of an ongoing neural injury created by tethering or irritation of the nerve by any cause. In this setting, when neuropathic pain persists, exploration and exposure of the affected nerve is indicated.
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References