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Case Report

CASE REPORT

Progressive foot drop caused by below-knee compression stocking after spinal surgery

Karan Malhotra*, Joseph S. Butler, Adam Benton and Sean Molloy

Spinal Deformity Unit, Department of Spinal Surgery, Royal National Orthopaedic Hospital, Stanmore, UK

*Correspondence address. Spinal Deformity Unit, Department of Spinal Surgery, Royal National Orthopaedic Hospital, Brockley Hill, Stanmore HA7 4LP, UK. Tel: +44 7986 757570; E-mail: karan@doctors.org.uk

Abstract

Foot drop is a debilitating condition, which may take many months to recover. The most common cause of foot drop is a neuropathy of the common peroneal nerve (CPN). However, similar symptoms can be caused by proximal lesions of the sciatic nerve, lumbar plexus or L5 nerve root. We present a rare and unusual case of a patient undergoing spinal surgery at the level of L5/S1 and presenting 4 weeks postoperatively with progressive foot drop. Although the initial concern was a postoperative lesion at L5, the cause for this delayed presentation was extrinsic compression of the CPN at the level of the fibular head by a tight-fitting below-knee thromboembolic deterrent stocking. Compression stockings are widely used in all branches of medicine and in the community. It is important to recognize this potential cause of progressive foot drop early as it is preventable by simple measures, which can significantly reduce morbidity.

INTRODUCTION

Foot drop is a debilitating condition, which can take months to recover. One of the commonest causes for foot drop is a compressive neuropathy of the common peroneal nerve (CPN) at the level of the fibular neck [1]. We present the case of a patient who developed a progressive foot drop after spinal surgery due to a tight thromboembolic deterrent stocking (TEDS). This is a very rare occurrence but if recognized early the impact may be reduced. We discuss how to clinically distinguish a CPN lesion from an L5 lesion. Although CPN palsies have been reported in similar settings previously [2–4], these have all been sudden occurrences and this is the first reported case of progressive CPN palsy caused by a below-knee TEDS. Our aim is to raise awareness that a CPN palsy can develop over time and this should be recognized as a clinical entity to enable earlier intervention.

CASE PRESENTATION

A 57-year-old, Caucasian female was admitted for revision spinal surgery at the level of L5/S1. There were no intraoperative or immediate postoperative complications. At discharge 7 days after surgery she was noted to have normal power: Medical Research Council (MRC) grade 5/5, and sensation in both lower limbs and was given below-knee TEDS to wear for 6 weeks.

She re-presented to hospital 3 weeks later with a right-sided, painless foot drop. She first noticed weakness and altered sensation in her right foot 1 week prior and had attended her general practitioner who felt it was related to her spinal surgery, and was not concerned. She had a high stepping gait with reduced sensation along the distal two-third of her lateral lower leg and over the dorsum of her right foot, including the first webspace. Plantar sensation, ankle jerk and plantar reflexes were preserved. MRC power grade was one-fifth for ankle dorsiflexion, ankle eversion, toe and hallux extension, and four-fifth for ankle inversion. All other muscle groups had normal power. She had normal distal pulses and capillary refill.

Her back pain had not increased, she had no leg pain, and no gross leg swollen. On removing her TEDS on the right leg, she was noted to have a well-demarcated indentation in the

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skin at the proximal rim which had become tight. This was seen at the level of the fibular neck and was not present on the contralateral side (Fig. 1).

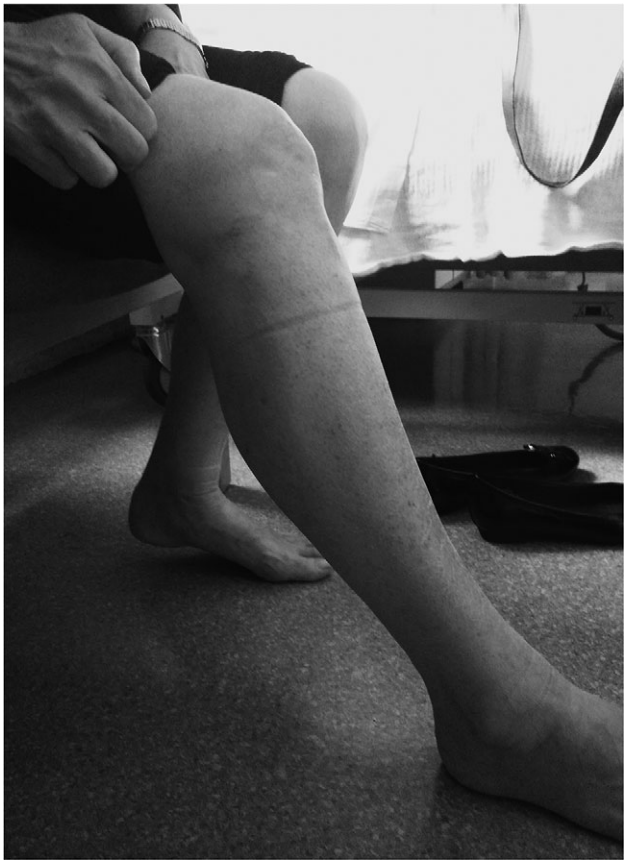

Figure 1: Clinical photograph of the patient's leg at presentation to our unit. A clear mark can be seen at the level of the fibular neck where the compression stocking had been tight.

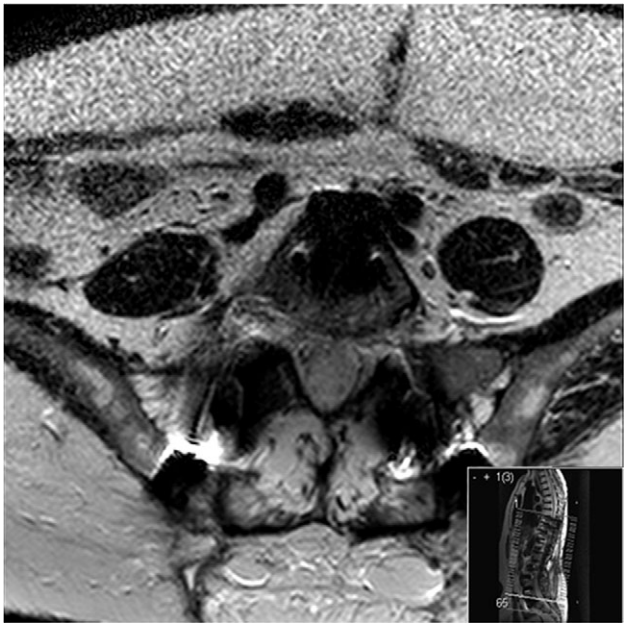

Figure 2: MRI (T1 weighted axial slice) of the lumbar spine at the level of L5/S1, demonstrating no compression of the cord or exiting L5 nerve root.

Differential diagnosis included a lesion of the right CPN or right L5 nerve root. A gadolinium-enhanced magnetic resonance imaging (MRI) scan of her lumbar spine demonstrated no signs of compression of the L5 nerve root (Fig. 2). MRI was performed of her right knee, which showed no extrinsic compression of the CPN, but demonstrated atrophy of the anterior muscles of the lower leg (Fig. 3). Nerve conduction studies demonstrated a severe right CPN lesion at the level of the fibular neck (Table 1).

The TEDS were removed. The patient was treated in a foot drop splint for 3 months and received physiotherapy (range of movement and strengthening exercises). At 6 months follow-up, complete recovery was noted with normal sensation and power in all muscle groups.

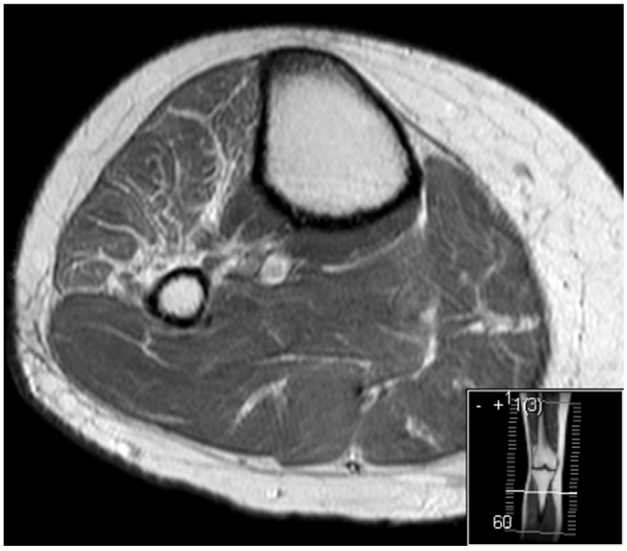

Figure 3: MRI (T2 weighted axial slice) of the leg at the level of the fibular neck, demonstrating no extrinsic compression of the peroneal nerves.

Table 1: Nerve conduction studies demonstrated increased latencies and reduced conduction velocities (20 m/s) from above the fibular neck (popliteal fossa), and normal latency and conduction velocity (48 m/s) below the fibular neck. These findings were consistent with a severe right CPN lesion at the level of the fibular neck with significant conduction block and a degree of axonal injury

Sensory—over superficial peroneal nerve (surface stimulation and recording—antidromic)	
Latency (ms)	3.8
Distance (cm)	15.5
Velocity (m/s)	41
Latency peak (ms)	4.6
Amplitude (μV)	4.5
Motor—over CPN (surface recordings at extensor digitorum brevis)	
Stimulation over ankle	
Latency (ms)	3.4
Stimulation below neck of fibula	
Latency (ms)	10.3
Distance (cm)	33.0
Velocity (m/s)	48
Stimulation over popliteal fossa	
Latency (ms)	16.4
Distance (cm)	12.0
Velocity (m/s)	20

Table 2: Chart showing the clinical differences and similarities between a lesion of the CPN and a lesion of the L5 nerve root. Italics denote a difference in clinical signs

	CPN lesion	L5 lesion
Ankle dorsiflexion	Weak	Weak
Ankle plantarflexion	Normal	Normal
Ankle eversion	Weak	Weak
Ankle inversion	*Normal*	*Weak*
Toe extension	Weak	Weak
Toe flexion	Normal	Normal/weak
Ankle jerk reflex	Normal	Normal/weak
Sensory loss	Dorsum of foot ± lateral distal two-third of leg	Dorsum of foot ± lateral distal two-third of leg
Pain	*Unusual*	*Common radicular pain/ lower back pain*

DISCUSSION

The L4, L5 and S1 nerve roots form the lateral trunk of the sciatic nerve via the lumbosacral plexus [1, 5–7]. The sciatic nerve divides into the tibial and CPNs just proximal to the posterior aspect of the knee. The CPN winds around the fibular head and neck, becoming subcutaneous and vulnerable to damage [1, 7, 8]. It then divides to form the superficial peroneal nerve (SPN) and deep peroneal nerve (DPN). The SPN travels in the lateral compartment of the lower leg supplying the peroneal muscles (everters of the ankle) and sensation over the dorsum of the foot and the lateral lower two-third of the leg [7]. The DPN travels in the anterior compartment of the lower leg and supplies the tibialis anterior (dorsiflexor of the ankle) long extensors to the toes, and sensation to the first webspace [5, 7, 9]. Damage to the CPN, therefore, results in a foot drop, with inability to evert the foot and loss of sensation in the areas described above.

The CPN does not supply the tibialis posterior muscle (inverter of the ankle) and so inversion is preserved [1, 7, 8]. As a predominant component of the CPN, fibers are derived from the sensory and motor fibers of the L4 and L5 nerve roots, an L5 lesion can mimic a CPN palsy. However, L4 and L5 also supply the tibialis posterior and so, in a lesion of L5, ankle inversion may be weak or absent [1, 7, 8, 10]. A lesion at L5 may also result in weakness of hip abduction, as L5 supplies the gluteus medius and minimus via the superior gluteal nerve [7]. It is difficult to reliably assess hip abduction in the setting of recent spinal surgery as it may be limited by pain; however, in this case, power in hip abduction was preserved which suggested a lesion at the CPN. Nerve root lesions are usually associated with radicular pain which was also absent in this case. Furthermore, the timing of the foot drop was delayed from surgery: complications from the metalwork or postoperative hematoma would be expected within the first week. Table 2 illustrates the clinical differences one may expect from between CPN and L5 lesions.

TEDS are commonly used in clinical practice in hospitals and the community. It is important that TEDS be carefully sized lest they roll down and become constrictive. CPN palsy due to intermittent pneumatic compression devices or ill-fitting above knee TEDS have been reported in the immediate postoperative period [2–4]. In our case, the onset of foot drop occurred 3 weeks following surgery and was not recognized. The patient, therefore, continued using an ill-fitting stocking, resulting in a progressive CPN neuropathy, eventually presenting as a dense right-sided foot drop. Although this is a very rare occurrence, it is imperative that clinicians be aware of this potential complication of ill-fitting TEDS. In such a setting, intrinsic nerve disorders predisposing to nerve palsies should be considered, such as hereditary neuropathy with liability to pressure palsies (HNPP). However, in this case, we did not perform genetic testing for the following reasons: our patient was 57 years of age and this was the first nerve palsy she has sustained; the neurophysiology findings did not suggest a neuropathy and would be expected to show a demyelinating polyneuropathy in the setting of HNPP [11]; the patient had a relatively quick and complete recovery.

Foot drop after spinal surgery may present a diagnostic dilemma for the clinician. It is important to rule out a surgical complication but the clinician should be mindful of the possibility of a CPN lesion as this is easily preventable and if missed, carries significant morbidity. Progressive lesions are rare but can occur and must be recognized. This case report is unique in that it describes such a progressive lesion occurring over a period of 4 weeks. Knowledge of the clinical signs of the possible lesions may help clinically differentiate the level of the lesion causing the foot drop. MRI and electrophysiological studies help confirm the diagnosis, more accurately localize the level of the lesion and help in assessing prognosis. TEDS should be appropriately sized and worn to prevent complications.

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There are no acknowledgements to be made.

CONFLICT OF INTEREST STATEMENT

None declared.

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ETHICAL APPROVAL

This is a case report and ethical approval is not required.

CONSENT

The patient has read the paper in its entirety and has consented to its publication.

GUARANTOR

Karan Malhotra.

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