

# Peroneal nerve palsy following an ankle sprain: case report

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## Abstract

Nerve damage as a result of torsional injuries to the ankle is uncommon. It does appear however, that subclinical injury to the common peroneal nerve following ankle injuries is more prevalent than previously thought. This case report highlights the importance of neurological assessment in all patients who present with ankle sprains no matter how benign the mechanism of injury may be. An excellent end result was obtained in this patient with conservative treatment alone.

## CASE REPORT

A forty year old gentleman presented to the emergency department having twisted his right ankle on rough ground one day earlier. His main complaint at the time of presentation was that of lateral ankle pain and swelling and inability to weight bear. In addition he was noted to have a complete foot drop which had progressed overnight following his injury. He also described altered sensation over the lateral aspect of the leg and dorsum of the foot.

His past history included low back pain with left sided sciatica which had responded to physiotherapy. He had also sustained two previous inversion type injuries to his right ankle from which he had made a complete recovery.

On examination of his right ankle he held his foot in plantar flexion and was found to have significant swelling and tenderness over the lateral ligament complex. There was also tenderness over the posterior aspect of the lateral malleolus. Ankle passive range of motion was uncomfortable and restricted. Distal pulses were palpable. Knee and hip examination revealed no focal swelling or tenderness with normal pain free range of motion. Neurological assessment showed altered sensation to pin prick in the distribution of the common peroneal nerve and MRC grade 1 power in the ankle dorsiflexors. Left lower limb neurology was normal.

Plain anteroposterior and lateral radiographs of the right ankle showed no fracture. Urgent MRI scan of the lumbar spine showed a small L4/L5 disc bulge on the left side which was not in keeping with the clinical findings. EMG studies, performed within two weeks of the injury, showed evidence of a severe demyelinating lesion of the right common

peroneal nerve around the fibula neck.

Motor conduction velocity was in the demyelinating range in the right common peroneal nerve around the fibula neck (velocity of 23 m/s on the right compared to 48 m/s on the left). Sensory nerve action potential was of significantly reduced amplitude in the right superficial peroneal nerve when compared to the left (amplitude of 7uV on the right compared to 21uV on the left).

Concentric needle EMG sampling showed evidence of marked chronic denervation with reinnervation in the right tibialis anterior and right peroneus brevis. The right tibialis anterior showed scanty fibrillation potential and positive sharp waves but on voluntary effort there were long duration low amplitude re-innervation motor units firing at abnormally rapid rates. The right peroneus brevis also showed evidence of re-innervation. The presence of re-innervation activity indicated continuity of the nerve. This study confirmed that the patients' lesion was peripheral in the common peroneal nerve and was not due to spinal pathology.

This patient was managed non-operatively with a foot drop splint and an intensive physiotherapy programme. He was reviewed regularly in the fracture clinic and by six weeks there was clinical evidence of nerve function recovery. He progressed well and at final follow up five months following his injury he was found to have a stable ankle with normal pain free range of motion. Sensation was improving with MRC grade 5 power in the ankle dorsiflexors.

## DISCUSSION

In the United Kingdom, an estimated 302,000 ankle sprains

are seen each year in Accident and Emergency Departments. This common injury is most frequently sustained during sporting activity usually as a result of forced ankle plantar flexion and inversion. Lateral ankle sprains account for 85% of all ankle sprains (1).

The lateral ankle ligament complex consists of anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL). ATFL is the weakest and most frequently injured element of the lateral ankle ligament complex. Lateral ankle ligament injuries are graded from I to III, based on increasing ligament damage and morbidity. In a grade I sprain, the ATFL is stretched with some of the ligament fibres torn, but no frank ligamentous disruption is present. A grade II sprain frequently involves complete tear of the ATFL and an additional partial tear of the CFL. A grade III injury implies complete disruption of both ATFL and CFL, possibly with a capsular tear. An accompanying tear of the PTFL can be present (1).

The complication of common peroneal nerve palsy following an ankle sprain has been reported in the English language medical literature (2,3,4,5,6). Isolated superficial peroneal nerve palsy has also been reported (7).

Although nerve injury is thought to be rare, a study by Nitz AJ et al (8) found that in their study group, 86% of patients with grade 3 ankle sprains had EMG evidence of injury to the peroneal nerve at two week follow up although clinical neurological examination remained normal. In addition 83% of patients with grade 3 sprains had EMG evidence of injury to the posterior tibial nerve. This would suggest that subclinical injury to peripheral nerves may be more common following severe ankle sprains than thought.

The aetiology of peroneal nerve paralysis following ankle sprain is not clearly understood. It is postulated that several mechanisms act alone or in combination to result in the clinical picture. Oppenheim (9) suggested nerve traction as a possible cause, as the nerve descends around the fibular neck.

Hyslop (10) postulated that the peroneal nerve is compressed against the fibular neck when the investing fibrous origin of the peroneus longus is drawn tight as the muscle resists ankle inversion. The effects of the nerve damage would be immediate in these situations. In Hyslop's three cases of peroneal-nerve palsy following inversion sprains of the ankle, paralysis occurred immediately following the injury in

only one. In another the palsy did not develop until the day after the inversion and planter flexion sprain; and in the third, not until one week after the sprain. In cases of delayed onset of paralysis, traction-compression mechanism does not fully explain the mode of injury. In these cases, it is more likely to be the result of a gradually expanding haematoma within the nerve sheath consequent to rupture of a nutrient vessel.

In two cases of common peroneal nerve injury following spiral fractures of the distal fibula, reported by Nobel (4), the dramatic disappearance of pain and paralysis after early evacuation of the haematoma strongly suggest that the haematoma in the nerve sheath was the sole cause of the paralysis.

In our patient neurological symptoms developed overnight resulting in foot drop. Leg pain was not significant at the time of presentation. The late onset of the paralysis in this case favours the diagnosis of paralysis by a haematoma which developed gradually and was subsequently resorbed without permanent sequelae.

Function of the peroneal nerve should be evaluated in all patients with the history of inversion ankle sprain. Manual muscle testing and neurological exams should be performed and these findings accurately documented. This examination may help in differentiating between overstretched nerve fibres and an expanding haematoma. Paralysis is usually immediate after overstretching, but if the paralysis has a gradual onset and causalgia-like pain develops, a haematoma within the sheath is more likely.

Early evaluation with EMG studies is helpful and provided the EMG studies show continuity of the nerve with evidence of re-innervation we feel that this unusual case can be managed safely by non-operative measures. There does not appear to be convincing evidence to suggest that early surgical exploration of the nerve is appropriate in all cases.

This case acts as a reminder of the importance of neurological assessment in patients who present with ankle sprains even if the mechanism of injury appears benign. An excellent recovery occurred in our patient with conservative treatment measures alone.

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