An 18-year-old male presented to the Accident and Emergency Department at Aberdeen Royal Infirmary with a 48 hour history of right footdrop and paraesthesia, which had gradually developed towards the latter part of a training weekend in aikido. He recalled no direct injury, but described that he had repeatedly and forcibly inverted the ankle during the course of training. He further commented that a similar but milder episode of footdrop had occurred during a previous training weekend but that this had resolved spontaneously with rest.

On examination there was a full range of movement of both the knee and ankle joints, with no evidence of ligamentous laxity, bruising, tenderness or swelling.

There was a partial right footdrop with grade 3 power noted in the peroneal and anterior tibial muscle groups, which were otherwise normal. There was no abnormality of the posterior musculature. Paraesthesia was evident in the distribution of both the superficial and deep branches of the common peroneal nerve. Radiographs of the knee and ankle joints were normal. A diagnosis of neuropaxia of the common peroneal nerve was made, the patient managed expectantly and when seen for review two weeks later, there was complete resolution of symptoms.

The common peroneal nerve may be damaged as a result of compartmental compression or traction injury. We found no clinical evidence to indicate that the injury was a consequence of compression and accordingly, did not attempt to gauge compartmental pressures. Traction damage to the common peroneal nerve has been reported after adduction and hyper-extension injuries to the knee (Barbour and Levitt, 1983; White, 1988) and also following inversion injuries to the ankle (Meals, 1977). Traction to the nerve is generally thought to occur as it encircles the fibular head prior to passing underneath peroneus longus and dividing into its superficial and deep branches. Experiments with cadavers have demonstrated that by simulating an inversion injury to the ankle, up to 25 mm of movement can be demonstrated proximally in the common peroneal nerve (Nobel, 1968). A further aetiological factor was proposed by Hyslop in 1941 who noted that during the process of ankle inversion, the peroneal muscles contract in an attempt to counteract the inversion movement (Hyslop, 1941). In the course of this the nerve may be injured between the fibula and the contracting peroneus longus.

Operative exploration of patients with common peroneal nerve traction injuries has revealed multiple intraneural haematomata that develop at the sites of entry of the nutrient vessels (Nobel, 1966). These haematomata are not localised to the sites of traction, but have been found on occasions to extend above the knee joint.

Our patient described no single episode of trauma that could account for his symptoms and we therefore assume that the footdrop was the cumulative result of repeated ankle inversions whilst training. This footdrop and paraesthesia subsequently resolved with rest and the experience has not dampened his enthusiasm for the sport!

Rather as an afterthought, the patient volunteered the information that his aikido instructor complained of intermittent paraesthesia in what sounded like the distribution of the common peroneal nerve but had never reported any motor symptoms.

Yours faithfully,

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References