Case report

Unusual presentation of acute atraumatic peroneal compartment syndrome – A case report and review of literature

S.N. Qasim *, R. Rachha, M. Sood

Department of Trauma & Orthopaedics, Bedford Hospital NHS Trust, Kempston Road, Bedford MK42 9DJ, United Kingdom

1. Introduction

Acute traumatic peroneal compartment syndrome is a well known condition. It usually is a consequence of direct trauma, excessive muscular activity or as a complication of medical or surgical treatment. Acute atraumatic peroneal compartment syndrome is rare and poses a diagnostic challenge. Here we describe an unusual case that developed as a result of driving a car for 7 h and present a detailed review of literature of this phenomenon.

2. Case report

A 49 year old man, who was otherwise medically fit and well with BMI of 34, presented to the medical team with a three day history of right leg pain. Three days prior to his visit to our hospital, he presented to the emergency department in another city with sudden onset pain in his right leg after driving a car for 7 h. There was no history of trauma. He was initially treated with analgesia and was sent home. He presented again the next day to the same emergency department with worse pain and with some redness around the peroneal compartment and was treated as a case of cellulitis with analgesics, oral antibiotics and limb elevation. Three days after initial presentation he was referred to us after initial evaluation by the medical and vascular surgeons. He described unrelenting pain which got worse on walking or elevation of leg and felt much better with the foot dependant.

Physical examination revealed a tense, swollen and tender anterolateral compartment with mild erythema over the peroneal compartment (Fig. 1). There was no significant exacerbation of pain on passive stretching of the involved compartments. He had hypoaesthesia in the deep and superficial peroneal nerve distribution. He had a right foot drop with MRC Grade II power of dorsiflexion and Grade I power of eversion of the right foot and Grade V power of plantar flexion. He had palpable pedal pulses with peripheral capillary refill time of less than 2 s. Radiographs of lower limb did not reveal any fractures. His blood investigations revealed a normal White Cell Count and Urea and Creatinine but a Creatinine-Kinase (CK) of 9990 iu/L. His D-Dimer values were <100 ng/ml. Doppler ultrasound examination of the lower leg excluded deep venous thrombosis but interestingly revealed lateral compartment muscle oedema and reduced blood flow in the compartment.

A clinical diagnosis of anterolateral compartment syndrome with common peroneal nerve involvement was made and an emergency fasciotomy of the anterolateral compartments was done. The anterior compartment muscles showed no ischaemic damage. However the peroneal compartment was severely affected with necrosis of peroneus longus (Fig. 2). Initially about 50% of the peroneus longus was debrided and the wound was left open. Peroneus brevis and tertius were viable. Further debridements resulted in loss of about 80% of the peroneus longus. The wound was left open after the fasciotomy and was closed in a staged manner over ten days using a shoe lace technique1,14 (Fig. 3).

Supportive treatment in the form of fluids and prophylactic antibiotics was provided during the post-operative stay in the hospital and no additional renal support was required. He recovered well with no systemic compromise and was discharged home with an ankle foot orthosis (AFO) for his foot drop.

At six weeks follow-up there was hypoaesthesia over the superficial and deep peroneal nerve distribution with motor power of Grade III in the ankle dorsiflexors and grade II in the evertors. At one year follow up, there was no sensory deficit. The power in the ankle dorsiflexors was the same as the contra-lateral ankle dorsiflexors, but there was no further improvement in the motor power of the ankle evertors. There was no pain, instability or any varus deformity of the foot and so surgical treatment to address the weak evertors was not deemed necessary.

3. Discussion

Compartment syndrome can be caused by anything that increases the contents or restricts the volume of the compartment. Trauma is the most common cause. Different mechanisms of atraumatic compartment syndrome have been reported in the literature. Stollsteimer et al. described exercise induced acute compartment syndrome in a previously healthy athlete16 while
Ebenezer et al. reported in an athlete with a history of ‘shin splints’. There are some reports of acute compartment syndrome in football players resulting from ankle twisting or inversion injuries while in others it has been reported without any apparent injury to the calf. This phenomenon appears to be prevalent among military recruits. Cara et al. reported three cases each developing spontaneous intra-compartmental bleeding with haematoma formation during excessive physical activity that includes excessive walking (for 10 h) and climbing leading to acute compartment syndrome. Vanneste reported two cases; one after horse riding and other after a significant period of walking in an exhibition followed by prolonged immobilisation. Gershuni et al. reported that a flexed and abducted hip with a flexed knee and passively dorsiflexed ankle, the typical position adopted whilst driving, increases compartment pressure. He demonstrated that the greatest increase in compartment pressure to be in the deep posterior compartment followed by the anterior compartment. Interestingly, however, he demonstrated that there was no significant difference in pressure in the peroneal compartment.

A rise of pressure within a compartment above the closing pressure leads to ischemia of muscles and nerves. Rhabdomyolysis can lead to acute renal failure. Williams et al. and the extensive work of Finkelstein et al. shows that delayed fasciotomy can lead to infection, septicaemia, multiorgan failure, limb amputation and even death.

Given the potential morbidity it is important to recognise and treat compartment syndrome early. The diagnosis is primarily a clinical one. Unrelenting pain out of proportion to the inciting event is the most significant clue. Physical findings may include tense, tender swelling of the involved compartment with exacerbation of pain on passive stretching of the involved compartment muscles. There may be sensory deficit as well as weakness in muscle power. The diagnosis can be assisted by different modalities that include compartment pressure measurement, magnetic resonance imaging and ultrasound examination. Finkelstein suggested that compartment pressure recordings are not of much benefit after the crucial 8-h period, hence the importance of clinical diagnosis especially in delayed cases.

Surgical decompression by fasciotomy is the treatment of compartment syndrome. Functional outcome and the morbidity are related to timing of definitive treatment. The extent of muscle debridement depends on the surgeon’s experience as it can be very difficult to assess the muscle viability at the time of operation. Gabisan does suggest in his report to thoroughly decompress the muscle and possibly limit the debridement at the first operation which is what was done in this case. Supportive treatment is equally important to avoid any systemic complications.

4. Conclusion

This case report emphasises that compartment syndrome can present atypically. Isolated atraumatic peroneal compartment syndrome is very unusual and is very difficult to diagnose. One feature of note in this case is that in established compartment syndrome the blood supply to the muscles is compromised hence elevating the limb will further reduce the capillary pressure and worsen the ischaemia and therefore the pain. To our knowledge,
peroneal compartment syndrome due to prolonged driving of a car has never been reported in the literature and should be a part of differential diagnosis in a patient presenting with leg pain.

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