Acute compartment syndrome of the dorsal forearm following noncontact injury

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INTRODUCTION

Acute compartment syndrome is a condition in which increased pressure within a closed tissue space compromises blood flow to muscles and nerves.1–4 Early diagnosis, often done by emergency physicians,4 can avoid limb-threatening sequelae, which include permanent neurologic deficit, muscle necrosis, ischemic contracture, infection, delayed healing of underlying fractures and rhabdomyolysis.5 The diagnosis is typically considered in traumatic mechanisms such as a direct blow or crush injury to a compartment, particularly when these are accompanied by a fracture in the same limb.6,7 However, the diagnosis is easily missed when caused by exertional and noncontact mechanisms.8–11 The following case illustrates that acute compartment syndrome must also be included in the differential diagnosis of injuries to the forearm from noncontact mechanisms.

CASE REPORT

A previously well 26-year-old man was evaluated by the trauma team at a rural surgical centre 1 hour after a single-vehicle rollover motor vehicle crash that occurred at low velocity. The patient reported a sudden onset of pain in the dorsal left forearm that began while forcefully grasping the steering wheel in an attempt to prevent himself from being ejected from the cab of the truck as it rolled over. After life-threatening injuries had been excluded, a secondary survey revealed a visibly swollen forearm with exquisite pain to palpation over the dorsal aspect. This pain was aggravated by passive wrist flexion. The dorsal forearm felt tense compared with the uninjured side, and had a palpable defect at the
junction of the middle and proximal third. The patient’s wrist extension was weak compared with the contralateral side, particularly involving the second, third and fourth digits. Sensation, distal pulses, capillary refill and limb colour were normal and there were no defects in skin integrity. Hemoglobin and hematocrit levels and the international normalized ratio were within normal limits. Radiographs of the radius and ulna, elbow and wrist were normal.

The patient was felt to have a rupture of the extensor digitorum muscle, and the orthopedic service was consulted. At this time, approximately 2 hours after the motor vehicle crash, the patient continued to have pain out of proportion to what would be expected given the physical findings and negative radiographs, and pain on passive stretch of the muscles of the dorsal forearm compartment. He did not have paresthesia or paresis. Despite the temptation to attribute all the signs and symptoms to the muscle rupture, a dorsal compartment pressure was obtained by the orthopedic consultant using a Stryker intracompartmental monitoring system and was found to be 80 mm Hg. With the confirmed diagnosis of acute compartment syndrome, the patient was immediately taken to the operating room for a dorsal compartment fasciotomy approximately 10 cm in length (Fig. 1). A complete rupture of the extensor digitorum muscle was confirmed and a nonexpanding large hematoma found within the muscle defect (Fig. 2).

The patient’s course in hospital was unremarkable. A washout, approximation of the muscle belly and closure of the skin was performed on postoperative day 2. The patient was advised to maintain the wrist splinted in extension for 2 weeks and was subsequently discharged from hospital with follow-up care.

DISCUSSION

The key to successful treatment of acute compartment syndrome is early diagnosis in the emergency department. This begins with appropriate suspicion and with considering the possibility of the diagnosis based on the mechanism of injury. Compartment syndrome can occur after virtually any injury, but is most commonly associated with mechanisms of direct contact or crush injury. In one series detailing the causes of acute compartment syndrome in 164 cases, 69% were associated with a fracture and 23% involved soft tissue injury without an associated fracture. The majority of cases without a fracture involved a direct blow to the affected muscle compartment or an injury with a major crushing mechanism. In 13% of cases without fracture, the etiology was deemed to be “spontaneous” secondary to exertional and noncontact mechanisms. This represented 3% of all cases of acute compartment syndrome seen. Interestingly, within the group of acute compartment syndrome due to soft tissue injury, 10% were either taking anticoagulants or had a bleeding disorder. A number of case reports exist describing acute compartment syndrome secondary to muscular rupture in the leg, and many conclude that noncontact mechanism as a cause of acute compartment syndromes is rare and easily missed. Although we identified 3 case reports of exertional acute compartment syndrome of the dorsal forearm, to our knowledge, ours is the first such report associated with muscular rupture.

The clinical diagnosis of acute compartment syndrome is based on pain “out of proportion to stimulus,” pain on passive stretch of the muscles of the involved
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compartment, paresthesia, decreased sensation to pin-prick, light touch or 2-point discrimination, and eventually paresis. Individually, the sensitivity of these clinical findings ranges between 13% and 19%, but the reported specificity and negative predictive values are 97% and 98%, respectively, suggesting that clinical features are more useful to exclude than confirm the diagnosis. The pain of a coexisting injury, particularly a fracture, can mask that of an impending compartment syndrome undermining the diagnostic utility of pain and pain from passive stretch. Hypoesthesia is the last clinical sign to develop before paresis, and only 13% of patients recover function when diagnosis is delayed until paralysis occurs. Diagnosis based on clinical signs and symptoms is difficult in patients who cannot provide a reliable history or participate in a physical examination. This includes patients with concomitant neurologic injury, critical illness or altered level of consciousness, and children.

Measurement of intracompartmental pressure is the definitive test for compartment syndrome. Urgent decompression is advised for measurements exceeding a threshold between 30 mm Hg and 50 mm Hg, with 30 mm Hg being the value most commonly cited. Other treatment triggers have also been suggested, such as a difference of 30 mm Hg between either mean arterial pressure or diastolic blood pressure and the measured intracompartmental pressure.

The only effective therapy for acute compartment syndrome is surgical decompression of the affected compartments by fasciotomy. To preserve muscle function, fasciotomy should be performed within 6 hours of injury; however, even 3 hours from injury 37% of patients develop muscle necrosis. Catastrophic results are virtually inevitable if fasciotomy is delayed beyond 12 hours after the injury was sustained.

CONCLUSION

Early diagnosis, based on appropriate suspicion from the mechanism of injury, signs and symptoms is the key to effective treatment of acute compartment syndrome. Soft tissue trauma that does not involve a direct blow, crushing forces or associated fracture is an uncommon but documented cause of acute compartment syndrome. The traditional diagnostic criteria of pain out of proportion to stimulus, pain on passive muscle stretch, paresthesia and paresis are individually insensitive and unreliable with certain patient groups. Such clinical criteria may be most useful when they are absent, to help exclude compartment syndrome. Measurement of intracompartmental pressure is the definitive test for compartment syndrome, and should be performed whenever the diagnosis is considered a possibility. A compartment pressure of 30 mm Hg is most commonly cited as the threshold to initiate treatment. Fasciotomy should be performed as soon as possible after diagnosis, and ideally within 6 hours of injury, to preserve functional outcome.

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REFERENCES


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