Acute compartment syndrome of the anterior thigh following quadriceps strain in a footballer

B J Burns, J Sproule, H Smyth


A rare case is presented of acute anterior compartment syndrome in the thigh of a footballer caused by an acute quadriceps strain, exacerbated by poor first aid and alcohol ingestion. Decompressive fasciotomy with subsequent split skin grafting of the wound defect resulted in a satisfactory outcome. The diagnosis requires a high index of suspicion lest it be overlooked with inevitably disastrous consequences.

A
cute compartment syndrome of the thigh in an athlete can be both limb threatening and life threatening and requires urgent diagnosis. The causal factor is usually a blow/direct trauma to the thigh (with or without fracture) or acute muscle overuse.1 Other precipitating factors include burns, arterial ischaemia, compression, or intoxication.2

CASE REPORT

A 21 year old man was referred to the emergency department 17 hours after taking part in a club game of soccer, complaining of severe constant pain in his right thigh. He recalled jumping vertically upwards from standing to compete for a header when he experienced acute pain in his right thigh. He denied direct trauma from either the ball or the competing player. He received no attention and continued to play for a further 10 minutes without further incident before limping off the pitch. He received no immediate medical/paramedical attention and did not apply any remedial measures. He consumed about 8 units of alcohol that evening. He took no analgesia.

He was unable to sleep properly, and, after consulting his primary care doctor, presented to the emergency department. He was non-weight bearing on the affected leg.

On examination his right thigh was swollen with a small area of ecchymosis on the anterolateral aspect of the distal thigh. The circumference of the right thigh at a level 10 cm proximal to the insertion of the quadriceps tendon measured 52 cm compared with 46 cm on the left side. His quadriceps muscle was tense and exquisitely tender. Active quadriceps function, as evidenced by his capacity to straight leg raise, was restricted and painful. Passive flexion of his knee was limited by subjective pain. Peripheral pulses were normal and all motor units were intact. There was an area of hypeaesthesia in the distribution of the lateral cutaneous nerve of the thigh.

Intracompartmental pressure was measured using a hand held monitoring system (Stryker Surgical, Kalamazoo, Michigan, USA). Anterior compartment pressure measured 29 mm Hg, medial compartment pressure measured 25 mm Hg, and posterior compartment pressure measured 27 mm Hg. Routine blood tests yielded: urea, 3.1 mmol/l; creatinine, 79 mmol/l; K+, 3.8 mmol/l; Na+, 136 mmol/l; bicarbonate, 31 mmol/l; white cell count, 10.6; haemoglobin concentration, 109 g/l; packed cell volume, 0.322. Urinalysis was normal. Radiology confirmed no fracture (figs 1 and 2).

A diagnosis of acute anterior compartment syndrome of the right thigh was made, and the patient gave consent for immediate fasciotomy. The surgical approach was through a lateral skin incision as described by Tarlow et al,3 extending from just distal to the intertrochanteric line inferiorly to the lateral epicondyle. The iliotibial band was incised in line with the skin incision. The vastus lateralis immediately herniated through the incision in the iliotibial band. All four muscles of the anterior compartment were considerably oedematous and were released individually. There was a 2 cm tear in the belly of the rectus femoris. There were no focal collections and no obvious areas of ischaemic or necrotic muscle. The compartment was soft after release. The vastus lateralis was carefully reflected off the lateral intermuscular septum. Releasing the septum along the length of the skin incision decompressed the posterior compartment. The hamstring muscles were normal. The wound was packed open, and sterile dressing was applied. Decompression of the adductor muscles through

Figure 1 Anteroposterior radiograph of lower femur.

Figure 2 Lateral radiograph of lower femur.
a separate skin incision was deemed not to be indicated. Four
days later the defect was covered in theatre by a split skin
graft (fig 3), and the patient was immobilised for five days.
He did not develop any complications. Rehabilitation entailed
initially passive and then gradual active motion exercises. At
three months, he was fully mobile and had full range of
motion. At five months, he returned to playing soccer.

DISCUSSION

Richard von Volkmann first described the theory of compart-
ment syndrome in 1881. He described ischaemic palsies and
muscle contractures after forearm and supracondylar hum-
eral fractures in children.4

In 1926 Jepson showed ischaemia resulting from an
increase in pressure in a subfascial space.5 However, the
definition was finally elucidated in 1976 by Mubarak et al.,6
who defined the syndrome as “an elevation of the interstitial
pressure in a closed osseofascial compartment that results in
microvascular compromise.”

In a search of the English literature, we could only find two
cases of acute thigh compartment syndrome in relation to
soccer, and both were related to definite contusion.7 To our
knowledge this is the only reported case of compartment
syndrome following an acute thigh muscle strain. Compartment syndrome of the thigh is, in general, rare
possibly because there is a large potential space to allow
swelling and hence increase in interstitial pressure before
endangering the circulation.8–10 Furthermore, the fascial
compartments of the thigh blend anatomically with the
muscles of the hips, potentially allowing blood to exit the
compartment under pressure.

Prompt recognition and surgical intervention in this
syndrome are imperative to prevent irreversible ischaemic insult.11 12 In many cases the diagnosis can be made from
clinical symptoms and signs alone. The cardinal features are:
(a) pain out of proportion to the clinical situation; (b)
weakness and pain on passive stretch of the muscles of that
compartment; (c) hypeaesthesia in the distribution of the
nerves running through that compartment; (d) tenseness of
the fascial envelope surrounding the compartment.13 14

Close monitoring of the limb at heart level and regular
examination of the patient at risk is essential once it has been
decided that there is the possibility of compartment
syndrome. If the above symptoms and signs worsen or do
not resolve, then prompt fasciotomy is indicated. However,
clinical findings may be equivocal if the patient has a head
injury with a lowered Glasgow coma score causing a lack of
communication. Furthermore, spinal cord injury and periph-
eral nerve injury may cloud the issue, as will the unco opera-
tive patient. In these cases, the clinician may not rely on
examination alone and involve scientific compartmental
pressure monitoring.

Pressure monitoring

The normal intracompartamental pressure ranges between 0
and 8 mm Hg.15 16 Gelberman et al17 suggested that ischaemia
of muscles and nerves occurs at about 30 mm Hg.17

Matsen et al18 used Whiteside’s continuous pressure
monitoring by way of an infusion technique catheter.
Although there may be a role for this method in the settings
mentioned above, we feel this may cause the clinician to
misleadingly rely on this method alone. Matsen et al observed
that, with this technique, there was a large range of
intracompartamental pressures in which some alert patients
developed neuromuscular deficits while others did not. They
showed that all patients who had maximum intracompart-
mental pressures of 45 mm Hg did not require fasciotomy.
All patients with maximum intracompartamental pressures of
55 mm Hg displayed the signs and symptoms of compart-
ment syndrome. Importantly, they showed variability in
pressure tolerance of a limb depending on its position relative
to the heart.19

Schwartz et al20 reported on a series of 21 thigh compart-
ment syndromes in 17 patients referred to their trauma
centre. They considered pressures of 30–40 mm Hg poten-
tially critical for the development of myoneural necrosis. A
pressure exceeding 40 mm Hg was considered diagnostic.
Thus there is obviously a low sensitivity and specificity
attached to intracompartamental pressure monitoring.

Nerve stimulation

Direct nerve stimulation may be used when one is trying to
ascertain why the patient is unable to voluntarily contract the
muscles of a compartment. Is this because of the compart-
ment pressures causing neuropraxia or primary nerve injury
proximal to the compartment? This is useful in the patient
with proximal nerve injury, spinal cord injury, and head
injury. This can be done at the bedside with a hand held
nerve stimulator such as the NS-2A peripheral nerve
stimulator (Professional Instruments Company, Houston,
Texas, USA). The electrical impulse is given subcutaneously
in approximation to where the nerve in question enters the
compartment. Failure of the muscle to contract would
suggest myoneural necrosis and hence compartment syn-
drome. However, if the muscle produces a normal response to
stimulation, it can be concluded that there is a nerve injury
proximal to the compartment. This is not useful as a
prospective monitoring aid, as clinical grounds should pre-
empt the development of paralysis secondary to compartment
syndrome. We believe this form of investigation is open to
operator disparity caused by incorrect placement of the
stimulation needles. Furthermore, in severe trauma it is
possible to have concomitant acute nerve injury to a
compartment and compartment syndrome, thus rendering
this investigation unhelpful.

Our patient represents that small group of patients in
whom compartment syndrome develops insidiously.
Although this patient cannot recall blunt trauma to the
thigh, it is possible that he did in fact sustain direct trauma,
as evidenced by the ecchymosis and swelling. However, there
is also the possibility that the pathophysiology was related to
an acute strain of the quadriceps muscle causing muscle
oedema secondary to inflammation. Oedema is usually
proportional to muscle damage.20 The mechanism of injury
and lack of recall of trauma would support this. Furthermore,
this patient took no measures to lessen the swelling and in
fact aggravated it by not maintaining the leg at heart level
and consuming alcohol. Alcohol has a clear association with
increasing oedema.

There have been a few reports of compartment syndrome
occurring after intense exercise. We do not associate this
patient’s case with compartment syndrome related to intense

Figure 3 Fasciotomy with split skin graft in situ.
exercise. Nau et al described such a case in the right thigh of a 51 year old man which developed three days after intensive weight training. This patient had gross muscle oedema visualised after fasciotomy and raised creatine kinase (1800 U/l) and lactate dehydrogenase (411 U/l) activities, indicating muscle damage. Kahan et al described a similar case in a 27 year old man who presented with reddish/brown urine, fever, and cramping pain in his thighs. He had performed 200 squats 24 hours before in response to a challenge and was not used to high intensity endurance exercise. His creatine kinase (116-460 U/l) and lactate dehydrogenase (4230 U/l) activities were grossly elevated. Intracompartmental pressures confirmed bilateral compartment syndrome. Bilateral fasciotomies were performed and necrotic muscle was debrided. Reneman described a series of 52 patients who had acute compartment syndrome caused by intensive use of the leg muscles. He postulated that the primary contributing factor causing the increase in intramuscular pressure (hence compartment pressure) was acute muscle hypertrophy. He proposed that there was a 20% increase in muscle volume caused by an increase in transcapillary filtration resulting from an increase in capillary pressure and surface area not compensated for by drainage.

**Conclusion**

We conclude that our patient developed an acute quadriceps strain causing inflammation and oedema in the muscle. This common and usually non-threatening injury developed into a compartment syndrome because of increased capillary pressure and increased surface area secondary to acute muscle strain, aggravated by the vasodilatory effects of alcohol, coupled with the effect of gravity to decrease venous return. Inappropriate immediate measures for decreasing swelling and actions that aggravate swelling of an injury can result in a potentially life threatening event. Compartment pressure measurements should only be used as a helpful guide in what is essentially, in the majority of instances, a clinical diagnosis. The clinician must remain vigilant and maintain a high index of suspicion lest an acute compartment syndrome be overlooked with inevitably disastrous consequences.

**Correspondence to:** Dr Burns, Emergency Department, St Vincent’s Hospital, Elm Park, Dublin 4, Republic of Ireland or 52 Beechpark Drive, Foxrock, Dublin 18, Republic of Ireland; bburns_2000@yahoo.com

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