Bilateral femoral nerve compression and compartment syndrome resulting from influenza A–induced rhabdomyolysis: a case report

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ABSTRACT

Bilateral femoral nerve compression neuropathy caused by primary iliopsoas muscle pathology is rare. We report a case of extensive compartment syndrome of the right arm and both legs associated with bilateral femoral nerve palsy resulting from severe muscle swelling secondary to influenza A infection. Our objective is to alert physicians to the possible development of compartment syndrome in patients with influenza and severe myalgia. We also reviewed the literature on the pathophysiology and management of femoral nerve compression neuropathy.

Key words: compartment syndromes; influenza A virus; myoglobinuria; nerve compression syndromes; rhabdomyolysis

CASE REPORT

At 2 am, 1 January 2006, an 18-year-old man collapsed at home and was immediately sent to the hospital by his mother. He regained consciousness upon arrival. He had a history of allergic rhinitis and chronic otitis media. He presented with productive cough and left ear discharge for a week, and generalised myalgia and weakness for 2 days. He had no recent travel and denied illicit drug abuse. His immunisations were current and he had no family history of myopathy or clotting disorders.

Physical examination demonstrated that he was febrile (39.4°C), tachycardic (108 beats/minute), and hypotensive (90/40 mm Hg). Fluid resuscitation was started immediately and the patient was transferred to the intensive care unit. His vital signs improved after fluid resuscitation (blood pressure, 120/70 mm Hg; heart rate, 90 beats/minute).

At around 11 am, the patient complained of increasing swelling and pain in all 4 limbs. Initial laboratory tests revealed an elevated white cell count (17 x10^9/l), hyperkalaemia (K=7.3 mm/l), and metabolic acidosis (pH=7.15). Other pertinent laboratory studies included serum creatine phosphokinase (538 000 iu/l), lactate dehydrogenase (16 660 iu/l), aspartate aminotransferase (2840 iu/l), and alanine aminotransferase (693 iu/l). His renal function was impaired (urea, 9.8 mmol/l; creatinine, 369 μmol/l) and his urine was tea-coloured. Urinalysis demonstrated myoglobin, a large amount of albumin and red blood cells.

The patient had severe pain over the right forearm and both legs, which were grossly swollen and tense.
The right hand and both feet were paraesthetic and cool; capillary refill was approximately 3 seconds. Passive stretching of the fingers and ankles produced excruciating pain at the right forearm and both calves, respectively. The left arm and both thighs were relatively spared.

At around 12:30 pm, the intra-compartment pressures of all 4 limbs were measured. They were markedly increased in the right forearm and both legs; the left forearm was normal (Table 1).

Neurological examination demonstrated weakness in ankle dorsiflexion secondary to peroneal nerve entrapment, in hip flexion and adduction, and in knee extension bilaterally. The femoral stretch test was positive, both knee jerks were absent and sensation in the L2 to L4 dermatomes was diminished. Knee, ankle, and toe flexion was relatively preserved (Table 2).

The abdomen was soft and not distended; the back, buttock and both thighs were soft and non-tender, with no clinical evidence for compartment syndrome. Urgent computed tomography was arranged to look for an intra-abdominal or pelvic haematoma or abscess that may have caused compression of the femoral nerves.

The patient’s general condition deteriorated drastically. He became so tachypnoeic that endotracheal intubation was necessary. His renal function deteriorated rapidly and he developed a coagulopathy.

At 2:30 pm, haemodialysis was started and emergency fasciotomy of the right upper limb and both lower limbs was performed at bedside with patient’s condition being closely monitored by the anaesthetist and intensive care unit staff. Intra-operatively severe muscle engorgement was noted upon incision of the fascia; part of the muscles in the anterior compartment of the left lower limb was found to be necrotic and was excised. Continuous muscle swelling was observed after compartmental release (Fig. 1).

At 5:20 pm, computed tomographic scanning of the abdomen and pelvis revealed severe swelling of the iliopsoas and iliacus (Fig. 2). Generalised swelling of the intercostal and paraspinal muscles was noted. There was no evidence for intra-abdominal haematoma/abscess or intra-spinal pathology. The patient’s renal function did not improve and he remained dependent on mechanical ventilation and haemodialysis. He also remained anaemic and coagulopathic. Multiple blood and fresh frozen plasma transfusions were needed during the postoperative period. Because of his critical condition, surgical exploration to release the femoral nerve was not performed. Leg support was given to keep his hips in flexion to alleviate femoral nerve tension.

<table>
<thead>
<tr>
<th>Extremities</th>
<th>Intra-compartmental pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mobile wad</td>
</tr>
<tr>
<td>Right forearm</td>
<td>25</td>
</tr>
<tr>
<td>Left forearm</td>
<td>24</td>
</tr>
<tr>
<td>Right leg</td>
<td>52</td>
</tr>
<tr>
<td>Left leg</td>
<td>90</td>
</tr>
</tbody>
</table>

### Table 1
Intra-compartmental pressure in the 4 extremities

<table>
<thead>
<tr>
<th>Neurology</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strength</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip flexion</td>
<td>Grade 1</td>
<td>Grade 1</td>
</tr>
<tr>
<td>Hip adduction</td>
<td>Grade 2</td>
<td>Grade 2</td>
</tr>
<tr>
<td>Knee extension</td>
<td>Grade 1</td>
<td>Grade 1</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
<td>Grade 0</td>
<td>Grade 0</td>
</tr>
<tr>
<td>Ankle eversion</td>
<td>Grade 0</td>
<td>Grade 0</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>Grade 0</td>
<td>Grade 0</td>
</tr>
<tr>
<td>Ankle plantar flexion</td>
<td>Grade 3</td>
<td>Grade 3</td>
</tr>
<tr>
<td>Flexor hallucis longus</td>
<td>Grade 4-</td>
<td>Grade 4-</td>
</tr>
<tr>
<td>Sensation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light touch and pinprick</td>
<td>Diminished</td>
<td>Diminished</td>
</tr>
<tr>
<td>S1 to S3</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Reflex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Ankle</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

### Table 2
Neurological examination

![Figure 1](image.png)
Severe muscle swelling after fasciotomy of the forearm.
Wound debridement was performed repeatedly. Muscle swelling gradually subsided and split skin grafts were used to cover the fasciotomy wounds on day 9. However, part of the skin graft failed and 2 more operations on days 13 and 16 were needed to provide complete wound coverage.

The patient’s respiratory function gradually improved after wound coverage and he was extubated on day 19. Daily haemodialysis was stopped on day 20 as his renal function returned to normal. His lower limb power improved slowly. On day 25, strength of hip flexion and knee extension was assessed as grade 2 and grade 4, respectively. Strength of ankle and great toe extension remained grade 0. On day 35, strength of hip flexion and knee extension was grade 4+; ankle dorsiflexion and eversion were grade 3 and great toe extension was grade 2. He was able to walk independently despite mild bilateral foot drop. An anti-foot drop orthosis was prescribed and he was discharged on day 45.

Eight months after the onset of illness, the patient had fully regained strength in his extremities, though he had persistent weakness (grade 4–5) in the left great toe and in dorsiflexing the ankle. This may have resulted from removal of necrotic muscle in the anterior compartment during debridement. Range of movement in all limbs was normal (Fig. 3).

DISCUSSION

Compartment syndrome caused by influenza-induced rhabdomyolysis has been reported.\(^1\)\(^-\)\(^3\) Its clinical presentation is similar to those caused by fracture, soft tissue injury, arterial injury, and prolonged limb compression. Symptoms include excruciating pain, paraesthesias, limb paralysis, decreased distal circulation, and fever, although they may not be apparent, especially in unconscious patients. In the evaluation of a patient with influenza and severe myalgia, clinicians must have a high index of suspicion for compartment syndrome. If in doubt, measurements of compartment pressures may suggest the diagnosis.

Screening for infective cause of rhabdomyolysis was performed on the first day of presentation. Blood culture and toxicology analyses were negative. Urine legionella antigen was negative. Atypical pneumonia titre was not elevated. Only the initial tracheal aspirate was positive for the influenza A antigen. The source of the influenza A remains undetermined. None of

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**Figure 2** Magnetic resonance imaging demonstrates swelling of the psoas and iliacus muscles.

**Figure 3** Recovery of limb strength.
the patient’s contacts had similar upper respiratory symptoms nor myolysis.

Once the diagnosis of compartment syndrome is made, urgent fasciotomy for compartmental release is suggested to prevent further rhabdomyolysis which may self-perpetuate a cycle of additional muscle damage. In cases of influenza-induced rhabdomyolysis, a distinctive feature of continued muscle swelling after the release suggests a primary muscle pathologic process,¹ as opposed to other more common causes of compartment syndrome in which muscle damage is secondary to elevated external pressures. Taking into account the possibility of further muscle swelling, careful and continuous wound monitoring is needed to ensure adequate compartmental release and debridement of necrotic tissues. Skin grafting to cover the fasciotomy wound should be delayed until muscle swelling has subsided.

Extensive compartmental involvement is another distinctive feature of compartment syndrome caused by influenza infection.¹,² In our case, there was generalised muscle swelling resulting in not only compartment syndrome in the extremities, but also femoral nerve compression as a result of iliopsoas muscle swelling, which made the diagnosis difficult.

Femoral compression neuropathy secondary to psoas or iliacus haematoma has been frequently reported. It may be associated with haemophilia or other blood dyscrasias, use of anti-coagulants and anti-thrombotic agents, traumatic haematomas, or rarely, rupture of an abdominal aortic aneurysm.⁴⁻¹⁵ Bilateral femoral compression neuropathy secondary to primary muscle pathology induced by influenza A infection has not been reported.

This patient presented with septic shock, compartment syndrome of the right arm and both legs, and femoral nerve palsy, which was not apparent during the initial clinical examination. At the time of presentation, our differential diagnosis included spinal infection or iliopsoas haematoma/abscess; we assessed the compartment syndrome secondary to prolonged compression during the period when the patient was unconscious. Contrast-enhanced computed tomography demonstrated that swelling of the iliopsoas muscle, not an intra-abdominal or intraspinal fluid collection, was the cause of the femoral nerve compression.

Management remains controversial, even in cases of femoral neuropathy secondary to iliopsoas haematoma with a localised pathology.¹⁶,¹⁷ Some authors recommend operative decompression of the haematoma if the patient is fit for surgery.¹⁸⁻²⁴ Others advocate conservative management at least initially,²⁵⁻²⁷ because meta-analyses failed to demonstrate significant differences in outcomes for surgical versus conservative approaches.²⁸ The decision was even more complicated in our case because there was diffuse iliopsoas swelling, but no localised pathology. Where and how extensive should the compartmental release be?

The femoral nerve comprises fibres from the posterior divisions of the anterior rami of the second through fourth lumbar roots. The nerve is formed from the roots in the substance of the psoas muscle and emerges from the lateral border of the muscle to run in a gutter between the iliac and psoas muscles. It passes through the femoral canal behind the inguinal ligament. Cadaveric experiments have shown that there is a sheet of thick iliacus fascia bridging the sulcus between the iliacus and psoas muscles, which is penetrated by the femoral nerve.²⁶²⁹ It is the rigidity of this distal part of the ‘fascial funnel’ which results in compression of femoral nerve in this anatomic region. One infusion experiment demonstrated that in the iliac fossa as many as 3 distinct laminae reinforce the distal portion of the iliac fascia. These laminae were either fused or separated to produce pouches of various sizes. Latex injected into these space spread from mid-lumbar levels to below the lesser trochanter, encircling, compressing and stretching the femoral nerve in its abdominal and sub-inguinal course.³⁰ Therefore, some authors suggest that femoral nerve compression is caused only when the iliacus is involved,²¹²⁶,³¹ although this is not always the case.³²

There is a report of lumbar plexopathy resulting from a retroperitoneal haemorrhage. In this case, haemorrhage caused distension of the psoas muscle and consequent compression of the entire lumbar plexus.³³ In addition to weakness of the hip flexors, there was weakness of the hip adductors, which were innervated by the obturator nerve.

In our case, both the psoas and iliacus muscles were grossly swollen and both hip flexors and adductors were weak. If fasciotomy were performed, extensive release from proximal psoas down to lesser trochanter may have been needed to ensure adequate decompression. Considering that the patient was in critical condition and coagulopathic, non-surgical therapy was preferred. Leg support was used to keep patient’s hips in flexion. Fortunately, the patient made a full neurological recovery.

CONCLUSION

Influenza A may cause rhabdomyolysis and result in an extensive compartment syndrome affecting all
4 extremities and other parts of the body. Thorough physical examination to look for evidence of compartment syndrome is indicated, particularly in unconscious patients. Prompt compartmental release can salvage affected limbs. Femoral nerve compression neuropathy can be managed conservatively.

REFERENCES