

## ■ CASE REPORT

# Non-operative management of femoral neuropathy secondary to a traumatic iliacus haematoma in an adolescent

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**Iliacus haematoma is a relatively rare condition, which may cause a local compressive neuropathy. It is usually diagnosed in adults with haemophilia or those on anticoagulation treatment and may occur after trauma. We present the case of a healthy 15-year-old boy with a femoral neuropathy due to an iliacus haematoma which resolved following conservative treatment.**

Haematoma within the iliacus muscle causing a compressive neuropathy is extremely rare and surgical treatment is often recommended.<sup>1-8</sup> We present a case of neuropathy following iliacus haematoma formation which resolved satisfactorily following conservative treatment.

### Case report

A 15-year-old boy presented with a four-day history of pain in his left leg, paraesthesiae and weakness. This followed an incident during a basketball game in which he hyperextended his left hip. He described mild discomfort, but completed the game. There was no history of pain before the injury. The pain became more severe and he developed left-sided abdominal discomfort. While playing baseball two days later, there was increased pain in his left hip after batting. Again, he finished playing but the pain increased. That night, he noted progressive numbness and weakness of his left leg. The next day he presented to another hospital where he was diagnosed with a muscle strain and given crutches. He was prescribed a high dose of non-steroidal anti-inflammatory medication (naproxen sodium; 250 mg every 6 to 8 hours). However, his continuing paraesthesiae, weakness and pain compelled his family to seek further help.

Initial examination was normal with stable vital signs. However, he was only able to walk with crutches because of the weakness in the left leg. There was tenderness in the left lower quadrant on abdominal palpation and numbness on the anterior aspect of the thigh and medial border of the lower leg and foot. There was no spinal tenderness.

Passive extension and active flexion of his left hip were painful and the most comfortable position was slight flexion and external rotation. The power of the quadriceps was grade 3 or equal to that of gravity. Strength elsewhere was normal. The quadriceps reflex was absent. The haemoglobin level was 15 g/dl and coagulation times were normal.

Plain radiographs of the pelvis was normal. An abdominal CT scan demonstrated a 3 cm × 7 cm intramuscular iliacus haematoma (Fig. 1). He was admitted and after consultation with the paediatric surgery service, conservative treatment was advised. He was placed on strict bedrest and given a 24-hour course of intravenous steroids to decrease the inflammatory component of the neural compression. Naproxen was discontinued. There was an improvement in sensation, primarily in the saphenous nerve distribution, and an increase in the strength of the quadriceps after 48 hours, he could walk with crutches and was discharged. Strength in the quadriceps had increased to grade 4. The haemoglobin level was monitored without significant change.

When seen one week after discharge there was improved sensation in the femoral and saphenous nerve distributions and strength in his quadriceps was almost fully restored. After two months clinical examination was normal and he had returned to full activities. An MR scan was normal and eliminated an arterio-venous malformation as the cause of the haematoma.

### Discussion

Two distinct lumbosacral plexus compression syndromes are recognised.<sup>9</sup> In one, the entire plexus is compressed within the psoas muscle, resulting in weakness of those muscles supplied

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Fig. 1

CT scan showing left iliacus haematoma (arrow).

by the obturator and femoral nerves. In the other, the femoral nerve is compressed within the iliacus muscle, as is thought likely in this case. Goodfellow, Fearn and Matthews<sup>10</sup> demonstrated the fibrous iliacus compartment through which the femoral nerve passes. Injection of fluid into this closed compartment caused it to swell, thereby applying pressure on the femoral nerve. In this event, flexion and external rotation of the hip were the positions of greatest compartment volume.

A review of the English case reports of iliacus haematoma producing a femoral nerve palsy in a previously healthy paediatric patient showed fewer than ten cases.<sup>1-8</sup> The majority were managed operatively. All patients achieved at least a partial functional recovery which seems to be the natural course of the condition. It is, therefore, difficult to confirm when surgical decompression would be beneficial.

In our case, we chose non-operative treatment for several reasons. Exploration of the retroperitoneum carries the risk of further bleeding after removal of the clot. There is also the potential for iatrogenic nerve injury and infection. CT-guided drainage was also considered, however, adequate decompression was questionable due to the presumed viscosity of the clot. Surgical decompression would have been attempted if the neurological symptoms were progressive or if there were signs of haemodynamic instability.

The cause of the haematoma is unclear. The patient had normal clotting factors, no history of a coagulopathy and no significant family history. In this case, muscle trauma was the most likely cause of haematoma formation, possibly worsened by the use of naproxen.

In summary, iliacus haematoma is rare in adolescence. It should be suspected in patients presenting with hip pain and neurological findings. Non-steroidal anti-inflammatory medication should be avoided in the early phase of the condition. The investigation of choice is CT which allows rapid identification and measurement of the haematoma. In the absence of progressive neurological findings, we feel it is reasonable to undertake conservative treatment as an inpatient. Investigation for possible underlying causes should be performed and treatment with steroids may also be helpful. In our patient, there was a rapid initial improvement followed by a slow recovery to normal over several months. Vigilance must be maintained, however, as progressive symptoms require decompression to avoid permanent neurological deficit.

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