

Femoral Neuropathy Following Anticoagulation Therapy in a Young Patient, A Case Report

Genç Bir Hastada Antikoagülasyon Tedavisini Takiben Oluşan Femoral Nöropati: Bir Olgu Sunumu

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ABSTRACT

Clinical report of a young patient with femoral neuropathy on anticoagulation therapy is presented. He was referred to the electromyography laboratory for evaluation of right thigh weakness. Femoral neuropathy secondary to iliopsoas hematoma is not an uncommon presentation in persons with bleeding disorders especially in persons with haemophilia. But this clinical situation is rarely reported in young patients on anticoagulation therapy. The clinical presentation and extent of nerve injury will depend on the site of hemorrhage.

Keywords: Iliopsoas hematoma, femoral neuropathy, electromyography, musculoskeletal rehabilitation

ÖZET

Antikoagülasyon tedavisi almakta iken femoral nöropati gelişen bir genç hastanın klinik raporu sunuldu. Hasta sağ uylukta güçsüzlük nedeniyle araştırmak amacıyla elektromiyografi laboratuvarına yönlendirilmişti. İliopsoas hematomuna ikincil femoral nöropati kanama diatezi olan, özellikle hemofilili hastalarda nadir değildir. Ancak bu klinik durum antikoagülasyon tedavisi alan genç hastalarda nadiren bildirilmektedir. Sinir hasarının boyutları ve klinik tablo hemorajinin yerine bağlı olacaktır.

Anahtar sözcükler: İliopsoas hematomu, femoral nöropati, elektromiyografi, muskuloskeletal rehabilitasyon

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Introduction

Nerve lesions are frequent causes for referral to electromyography laboratory. Femoral neuropathy secondary to iliopsoas hematoma is not an uncommon presentation in persons with bleeding disorders especially in persons with haemophilia. It has also been reported in older individuals on anticoagulation therapy (1-4). But this clinical situation is rarely reported in young patients on anticoagulation therapy. A high index of clinical suspicion is needed for the diagnosis especially when seen after referral for electro diagnostic studies.

Case report

15 years old male patient was referred for electro physiologic evaluation of right thigh wasting and weakness. He gave history of sudden onset right hip pain after playing football. There was similar history of right hip pain 3 months back following which he noticed mild wasting of right thigh. He was walking with support, poor weight bearing on right side and right hip held in flexion. Clinical examination revealed 30 - 40 degrees of flexion deformity of right hip, further extension was painful. Power of right knee extension was 0/5, further hip flexion 2/5 and hip adduction 4/5. Hip extensors were

acting but grading of muscle power could not be done because of pain. Sensory impairment was present in right anteromedial thigh and medial leg. Knee jerk was absent on the right side. Further exploration of history revealed that he was on warfarin for mitral valve disease. Iliopsoas haemorrhage with secondary femoral neuropathy was suspected. MRI of both hips (Figure 1) revealed bulky right iliacus muscle with well defined intramuscular mixed signal running the course of the muscle. The central

part of muscle was mostly cystic with multiple septa signifying localised organising muscular hematoma. The right psoas muscle appeared unremarkable. The hematoma was surgically evacuated. Follow up nerve conduction study (Figure 2) showed absent compound muscle action potential over right quadriceps muscle signifying severe axon loss of right femoral nerve. Left femoral nerve stimulation (Figure 3) elicited normal compound muscle action potential. Saphenous sensory

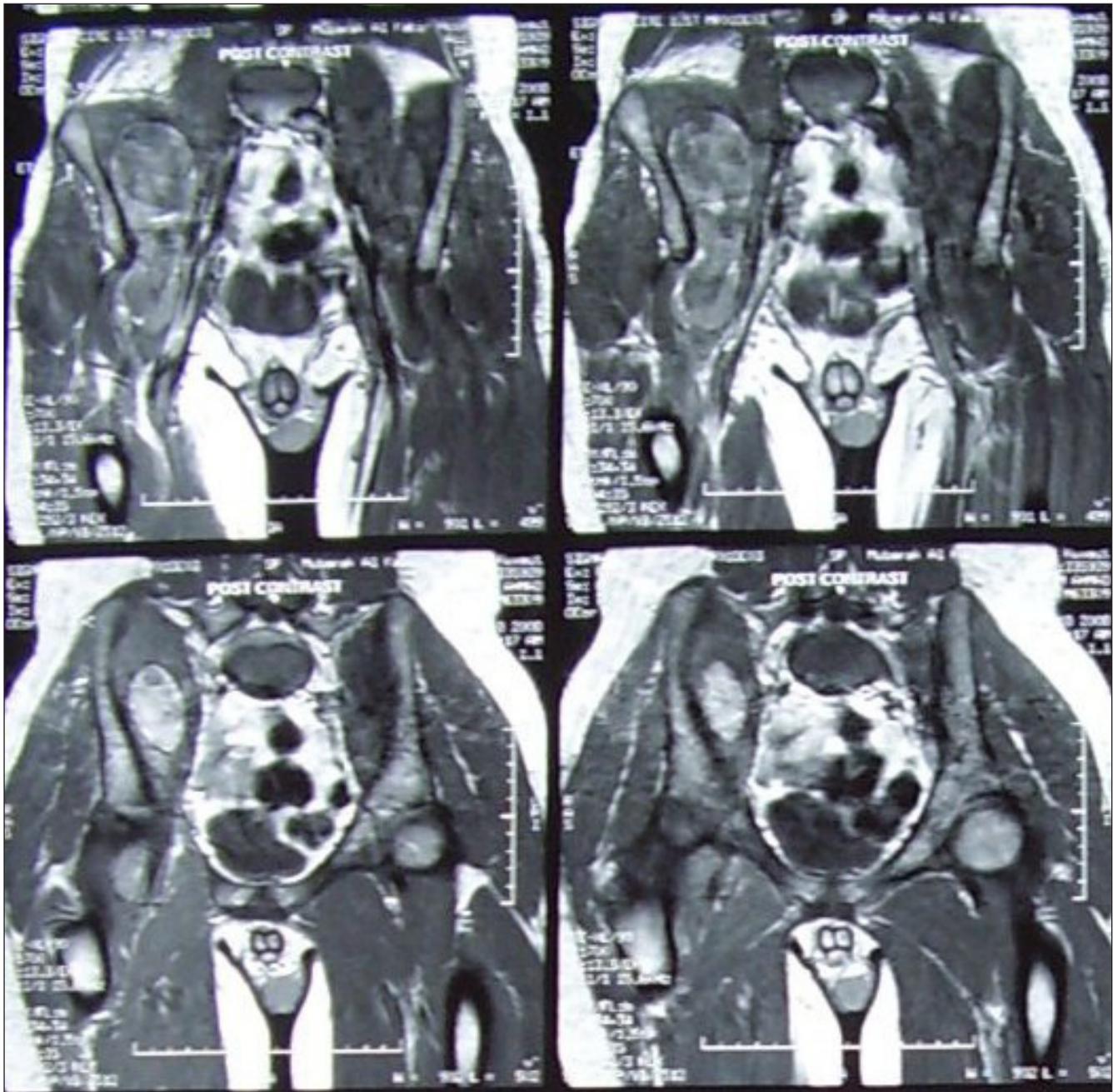


Figure 1. MRI of both hips showing bulky right iliacus muscle due to well defined intramuscular mixed signal intensity with cystic central part.

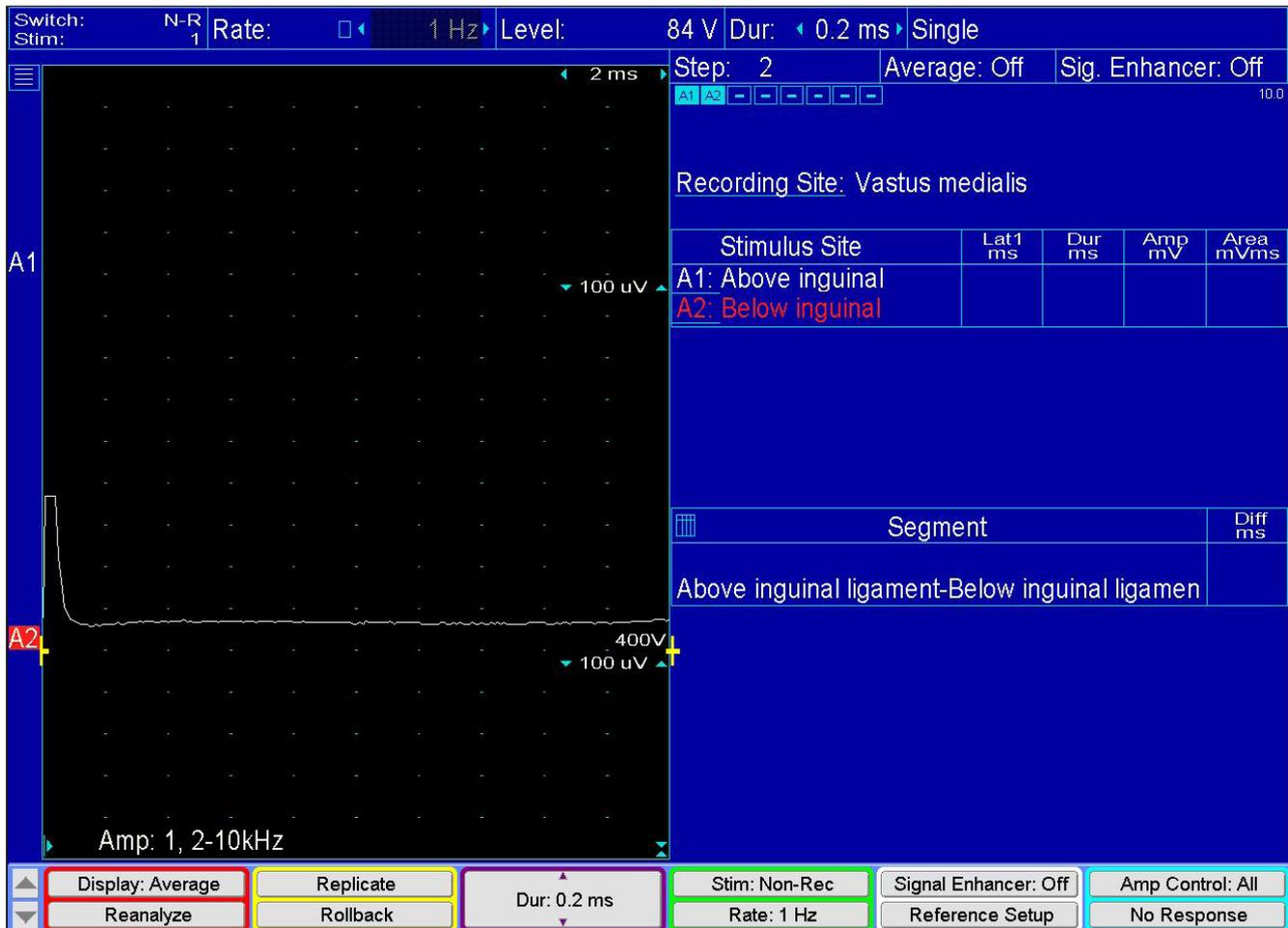


Figure 2. Nerve conduction study of right femoral nerve failed to elicit compound muscle action potential.

nerve action potential was also absent on the right side. Other nerve conduction data were unremarkable. Needle electrode examination was deferred as the patient was on Warfarin.

On follow up after six months he was walking without aids. The muscle power of right hip flexor and knee extensor has improved. Hip flexion was 4/5 and knee extension was 3-5. Sensory impairment persisted in right anteromedial thigh and medial leg. Knee jerk was absent on the right side.

Discussion

The lumbar plexus is formed from the ventral primary rami of first three lumbar nerves (L1, L2, L3), with a branch from the ventral primary ramus of T12 and major portion of ventral primary ramus of L4 nerve. Ventral primary rami from the above nerves emerge from the spinal column and lie within the substance of psoas muscle. The quadratus lumborum and psoas muscles are

supplied by direct muscular branches from the above rami. The lumbar plexus' various branches or peripheral nerves form in the psoas muscle's substance to then transverse separate paths supplying innervation to the inferior aspect of abdominal region and lower limb's anteromedial portion (5).

The femoral nerve is formed by the posterior division of L2, L3 and L4 ventral primary rami. It emerges through the fibres comprising the psoas major muscle's lateral margin coursing inferiorly in the intermuscular groove between this and the iliacus muscle (5). Covering these structures is the iliacus fascia. The retroperitoneal compartment containing femoral nerve and the two muscles continues inferiorly to the attachment of iliopsoas muscle in the thigh. After passing posterior to the inguinal ligament and lateral to the femoral artery, the femoral nerve divides into multiple branches. The cutaneous branches – intermediate femoral cutaneous nerve, medial femoral cutaneous nerve and saphenous nerve supply the anteromedial thigh and medial leg. The motor branch to iliacus muscle is supplied in the retro



Figure 3. Nerve conduction study of left femoral nerve shows normal compound nerve action potential on stimulating above and below inguinal ligament.

peritoneal region. The pectineus muscle and sartorius muscle are supplied after the nerve emerges from below the inguinal ligament. Final terminal muscular branches supply the quadriceps muscle (5).

It has been demonstrated anatomically that the iliac fascia forms separate compartments for the iliacus and psoas muscles except for a communication beneath the inguinal ligament where both muscles are enclosed by a dense and indistensible funnel (6). Large quantities of fluid can be injected beneath the sheath of the psoas muscles which is easily distended in its upper part. Fluid injected beneath the less distensible sheath of the iliacus muscle forms a tense globular swelling similar to the iliacus hematomas palpated clinically (6).

Hemorrhage into the iliacus, psoas and iliopsoas muscles is an infrequent complication of anticoagulant therapy and is usually unilateral (7). Retroperitoneal haemorrhage as a complication of anticoagulant therapy was first noted in 1953 by Russek, who found it twice among 122 fatal cases of myocardial infarction receiving anticoagulants (8). The fascial compartment about the

psoas muscle has the potential to be quite voluminous and patients with bleeding here can go into hypovolemic shock secondary to severe blood loss (5). Hemorrhage into the iliacus compartment is much less voluminous, but tends to develop high pressure (5). It is postulated that blood, arising from minor trauma to the iliacus muscle, fills the iliacus compartment and is forced into the funnel formed by the fascia covering iliacus and psoas muscles below the inguinal ligament, where the femoral nerve, lying between the two muscles is compressed (1).

Most of the reported cases in literature with femoral neuropathy following anticoagulant therapy occurred in adults or old patients (1-4). The low incidence in the young may be related to the lower prevalence of anticoagulation therapy in this age group. However a high index of clinical suspicion is needed for reaching the diagnosis.

MRI images of the patient described here revealed hematoma in the iliacus muscle. Based on the discussion above the following can be inferred for the present

case. Limited space within the iliacus fascia might have helped to contain the hematoma and prevented further bleeding. This may also explain the clinical presentation as femoral neuropathy rather than a lumbar plexopathy. High pressure developed because of limited space beneath the iliacus fascia may have produced severe compression neuropathy resulting in axon loss and longer recovery time.

It is a matter of controversy whether such hematomas should be surgically evacuated or conservatively treated (9). Conservative management is sufficient for cases with small hematomas and mild symptoms of femoral neuropathy, while some authors have recommended immediate surgical decompression in cases of life-threatening complications of iliopsoas hemorrhage (2). Transcatheter arterial embolisation of acute life threatening psoas hematoma is also described (9) especially in patients who are high risk candidates for surgery (2).

Conclusion

Femoral neuropathy secondary to iliopsoas hematoma is rarely seen in young patients on anticoagulation therapy. Bleeding into the psoas muscle can result in profound blood loss and hypovolemic shock. Iliacus hematoma tends to be localised but severity of neurologic insult may be more.

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