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Case Report

Bilateral iliopsoas hematoma: Case report and literature review

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Abstract

Background: Clinically significant spontaneous bilateral iliopsoas hematoma is a rare complication of anticoagulation therapy. Definitive treatment of spontaneous iliopsoas hematomas is not well-established and varies between observation and surgical intervention. The intramuscular hematoma causes severe pain, muscle dysfunction, and occasionally nerve palsy with the femoral nerve most commonly affected. Most patients are neurologically normal but when a significant neurological deficit is associated with iliopsoas hematoma, optimal treatment recommendations vary. We report a case of spontaneous bilateral iliopsoas hematomas causing significant bilateral femoral nerve dysfunction.

Case Description: The authors present the case of a 63-year-old female who developed bilateral femoral nerve palsy due to anticoagulation bleeding complication. Magnetic resonance imaging demonstrated large bilateral intramuscular psoas hematomas causing femoral nerve compression. Surgical evacuation and decompression of the femoral nerves was performed with rapid neurological improvement.

Conclusion: Management recommendations depend on the volume and cause of the hematoma, timing of diagnosis, and the degree of neurological impairment. A conservative approach with bed rest and correction of bleeding abnormalities to allow the hematoma to spontaneously resorb has been utilized for patients with small hematomas and little to no neurological symptoms. In contrast, more aggressive recommendations have been made for patients with large hematomas, severe motor function deficits, or hemodynamic instability.

Key Words: Anticoagulation, femoral nerve, hematoma, iliopsoas, peripheral nerve neurosurgery

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INTRODUCTION

The indications for anticoagulation or antiplatelet therapy are diverse. Given the increasing use of anticoagulants, the occurrence of bleeding complications is expected to rise. It has been reported that 1-7% of patients taking anticoagulants suffer a bleeding complication each year.[4] Iliopsoas hemorrhage is a rare and occasionally serious complication of bleeding disorders that occurs most commonly in patients with hemophilia but can be encountered in patients on anticoagulation therapy for other disorders. The intramuscular hematoma causes

severe pain, muscle dysfunction, and occasionally nerve palsy with the femoral nerve most commonly affected. Most patients are neurologically normal but when a significant neurological deficit is associated with iliopsoas hematoma, optimal treatment recommendations vary. We report a case of spontaneous bilateral iliopsoas hematomas causing significant bilateral femoral nerve dysfunction.

CASE REPORT

A 63-year-old female presented to the emergency room with acute chest pain, discomfort, and breathing difficulties. A computed tomography (CT) angiogram of the chest revealed a sub-segmental pulmonary embolism in the right lower lung lobe. The patient was subsequently admitted and started on intravenous heparin. She also received a single dose of clopidogrel (Plavix®) at the time of admission. The next day, she was started on warfarin (Coumadin®) 7.5 mg at bedtime with heparin bridging. Partial thromboplastin time was monitored but poorly controlled, showing variations from 29 to 180 seconds over the next several days. Five days later, the patient's International Normalized Ratio reached a therapeutic range at 2.0 and heparin was discontinued. That same day, she began complaining of abdominal and flank pain.

On day 7 of hospitalization, the patient developed moderate weakness in the proximal lower extremities. A CT scan of the abdomen was performed and demonstrated large, bilateral iliopsoas muscle hematomas. Magnetic resonance imaging (MRI) of the lumbar spine demonstrated acute hematoma formation in the iliopsoas bilaterally [Figure 1a-c]. Neurosurgery was consulted on day 8. On physical examination, the patient was in no apparent distress and stable hemodynamically. She had normal tone, but moderate bilateral proximal lower extremity weakness with 3/5 strength in bilateral iliopsoas and 2/5 strength in quadriceps. Extension of the hip elicited pain in the back and groin. The patient also exhibited hyperalgesia and numbness over the L2-L4 dermatomes, bilaterally.

Conservative management using vitamin K and fresh frozen plasma for Coumadin® reversal was recommended, with the intention of monitoring and following the patient's clinical examination. Over the next 2 days, the patient reported symptomatic improvement of her abdominal pain and thigh numbness, but persistent weakness. On day 11 of hospitalization, it was noted that the patient's strength had deteriorated further with iliopsoas and quadriceps strength of 1/5, bilaterally. Based on this neurological deterioration, she was taken for surgical decompression of the hematomas through bilateral flank incisions. The external oblique muscle and external oblique fascia were transected. The incision was carried down to the internal oblique muscle and preperitoneal space. A longitudinal incision was made over the psoas major muscles and hematoma material of 240 and 220 ml was drained from the right and left compartments, respectively. The femoral nerves were identified and separated from clot material. Despite bipolar stimulation of 1-4 μV to the femoral nerve, no significant electromyography response was elicited.

By the third postoperative day, the patient demonstrated marked improvement in strength and near complete resolution of her thigh numbness; both iliopsoas muscles had 4/5 power. The left quadriceps showed 4/5 strength while her right quadriceps remained 0/5. She was then discharged to a rehabilitation facility. Iliopsoas strength remained 4/5 bilaterally at 6-week follow-up. Her left quadriceps improved to 5/5 and her right quadriceps improved to 1/5. At 3- and 6-month follow-up visits, the patient was noted to have full strength in her lower extremities, but experienced continued mild dysesthesias in anterolateral thighs bilaterally.

DISCUSSION

The clinical presentation of iliopsoas hematomas is often that of sudden-onset low back pain. The differential diagnosis for back pain is extensive and includes pancreatitis, ureteric colic, lumbar spondylosis, aortic dissection, and musculoskeletal pain. The iliopsoas

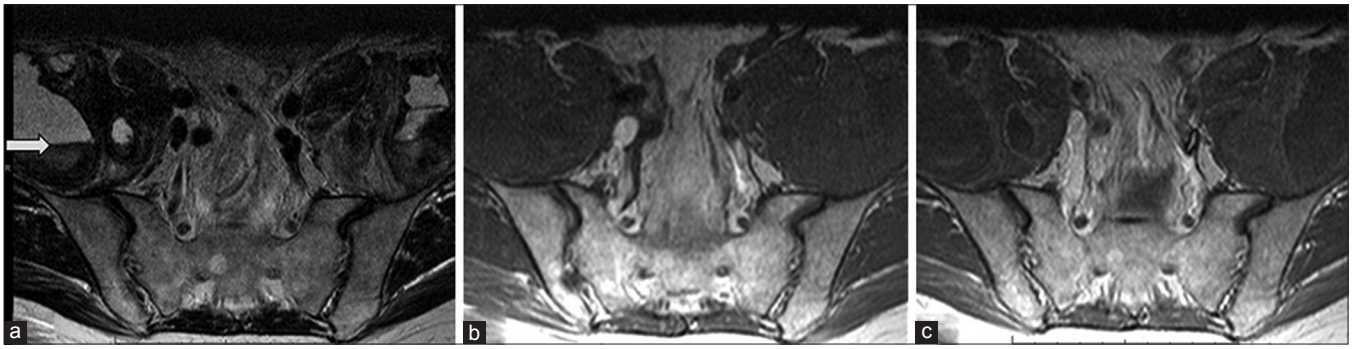

Figure 1: (a) Axial T2, (b) precontrast T1, and (c) postcontrast T1-weighted axial MR images of the lumbar spine showing markedly enlarged bilateral iliac muscles containing heterogeneous hematomas, which also show fluid–fluid level on axial T2-weighted images

muscles are retroperitoneal and may cause sudden severe back pain that radiates typically to the groin area and anterolateral thigh.[2-4,8-10,14] Typically the hip is flexed on the involved side, thereby relaxing the iliopsoas muscle, and pain is elicited on passive extension as the iliopsoas muscle is stretched (psoas sign).[2-4,9] In cases of large retroperitoneal hematomas, ecchymotic lesions in the flanks (Grey Turner's sign) or in the peri-umbilical area (Cullen's sign) may be noted. In extreme instances hypovolemic shock may ensue.[2,4,10,15]

Iliopsoas hematomas have also been associated with compressive femoral neuropathy. This occurs because of the long course of the femoral nerve, as it arises from the lumbar plexus formed by the posterior divisions of the L2-L4 nerve roots, descends though the fibers of the psoas muscle and emerges at its lower lateral border. The femoral nerve then travels down through the iliopsoas groove, formed by the lateral border of the psoas, medial border of the iliacus muscle and is covered by a tight, poorly distensible iliacus fascia.[3-5,8] The femoral nerve finally leaves the pelvis by passing beneath the medial inguinal ligament to enter the femoral triangle just lateral to the femoral artery and vein. The nerve can be compressed anywhere along its course but it is particularly susceptible to compression within the body of the psoas muscle, at the iliopsoas groove and at the inguinal ligament.[3,4,8-10]

Motor branches of the femoral nerve innervate the iliopsoas and the quadriceps. Its sensory branch, the saphenous nerve, innervates the skin on the anterior thigh and the anteromedial aspect of the calf.[3,4] Since the hematoma may compress the femoral nerve as it passes through the iliacus or psoas muscle, patients usually present with acute lower extremity paresthesias, and in severe cases, muscle weakness. The muscle weakness usually spares the adductor magnus and brevis, which are primarily innervated by the obturator and sciatic nerves. Furthermore, compression of the ureter can occur as it descends towards the bladder on the anterior aspect of the psoas major muscle, potentially leading to hydronephrosis and acute renal failure if the compression is bilateral.[3,4] Compression of the inferior vena cava causing deep vein thrombosis has also been reported.[3]

Besides clinical examination, there are several imaging modalities that can aid in establishing the diagnosis. Ultrasonography has been used to diagnose iliopsoas hematoma but its sensitivity and specificity are user-dependent, rendering the results potentially less reliable. In addition, obstruction of the ultrasonic waves by surrounding hollow organs may make it technically difficult to visualize the deep-seated iliopsoas muscle.[3,4,8,9] Plain X-ray films may also be useful, when an ill-defined iliopsoas shadow can suggest the diagnosis. However, MRI is the investigation modality of choice given its high sensitivity and specificity, but it remains expensive, time consuming, and contraindicated in patients with ferrogenic implants.[8] Therefore CT, which is readily accessible, fast, and has a high degree of sensitivity, remains the most commonly utilized test for this diagnosis.[3,4,8,9]

Even though iliopsoas hematomas are well-described in the literature, their incidence remains uncommon. Iliopsoas hematomas are typically caused by trauma in patients on anticoagulation/antiplatelet therapy or in those with hemophilia.[1,2,4] This entity was first reported by Tallroth in 1939 when he described an iliopsoas hemorrhage in a patient with hemophilia.[15] In 1966, Debolt reported the first two cases of femoral neuropathy due to anticoagulant (heparin) induced iliopsoas hematomas.[6] Since then, there have been multiple reports of iliopsoas hematoma in the literature that describe femoral neuropathy and weakness. To the best of our knowledge, ours is the first case report of bilateral spontaneous iliopsoas hematomas with significant bilateral femoral neuropathy.

Definitive treatment of spontaneous iliopsoas hematomas is not well-established and varies between observation and surgical intervention.[3-5,8-10,11-13] Management recommendations depend on the volume and cause of the hematoma, timing of diagnosis and the degree of neurological impairment. A conservative approach with bed rest and correction of bleeding abnormalities to allow the hematoma to spontaneously resorb has been utilized for patients with small hematomas and little to no neurological symptoms.

In contrast, more aggressive recommendations have been made for patients with large hematomas, severe motor function deficits, or hemodynamic instability. According to Butterfield, surgical decompression and neurolysis with identification of the femoral nerve will give superior outcomes compared with conservative management as only 2 of 10 of their patients experienced complete recovery with conservative management.[3] Also, ultrasound-guided percutaneous aspiration has been reported to be safe and beneficial in patients who are poor surgical candidates.[7,11]

Although our patient was first managed with observation, the neurological deterioration prompted surgical intervention, which led rapidly to improved neurological status. While it is possible that our patient would have recovered completely over time, we feel that surgical intervention allowed for a more rapid recovery and, possibly, avoided a poorer outcome.

CONCLUSIONS

The sudden onset of back pain in patients on anticoagulation therapy or those with bleeding disorders should trigger consideration of bleeding

complications. From the review of the literature and based on our experience it seems reasonable to pursue medical management with bed rest and the correction of bleeding abnormalities in patients who have little to no neurological complications. However, when a patient presents with significant and especially profound neurological deficits, surgical exploration and decompression may be the treatment of choice.

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