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Bilateral Femoral Neuropathy Following Psoas Muscle Hematomas Caused by Enoxaparin Therapy

Authors' Contribution:
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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Female, 64
Final Diagnosis: Bilateral femoral neuropathy
Symptoms: Inability to walk
Medication: —
Clinical Procedure: None
Specialty: Critical Care Medicine

Objective: Adverse events of drug therapy

Background: Femoral neuropathy as a result of retroperitoneal hemorrhage most commonly occurs following pelvic and lower extremity trauma, but has been described to develop as a less frequent complication of anticoagulation.

Case Report: We present the case of a 64-year-old white woman who was being treated for pulmonary embolism and deep venous thrombosis with enoxaparin. In the course of her treatment, she was noted to be hypotensive, with a sudden drop in hematocrit. She had been previously ambulatory, but noted an inability to move her bilateral lower extremities. A diagnosis of bilateral femoral neuropathy as a result of psoas hematomas caused by enoxaparin was made. Anticoagulation was discontinued and she was treated conservatively, with an excellent outcome. At the time of discharge to a rehabilitation center, she had regained most of the motor strength in her lower extremities.

Conclusions: We believe this is the first reported case of bilateral femoral nerve neuropathy following use of enoxaparin. A full neurological examination should always be performed when there is sudden loss of function. The constellation of bilateral groin pain, loss of lower extremity mobility, and decreased hematocrit raised the suspicion of massive blood loss into the cavity/compartments. Thus, a high index of suspicion should be maintained by clinicians when presented with such symptoms and signs, as there can be significant morbidity and mortality when prompt diagnosis is not made.

MeSH Keywords: Enoxaparin • Femoral Neuropathy • Hemorrhage • Psoas Muscles

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Background

The use of anticoagulation has increased over the years, ranging from its use in the treatment of deep venous thrombosis (DVT) and acute coronary syndromes (ACS) to primary prevention against thromboembolic events in atrial fibrillation [1]. Hemorrhage remains the most common and feared complication of anticoagulation therapy. However, when compared to unfractionated heparin (UFH), low molecular weight heparin (LMWH), such as enoxaparin, poses less bleeding risk among its other advantages, which include a superior efficacy and longer half-life, allowing for an easier dosing schedule [1–3]. The most common sites of bleeding are the oropharynx, soft tissues, gastrointestinal tract, and the genitourinary tract, in descending order of frequency [1]. The retroperitoneum is an uncommon location for spontaneous hemorrhage due to anticoagulation and occurs in about 1.3–6% of patients receiving anticoagulation [4,5]. Bilateral psoas muscle hematomas as a result of anticoagulation are an even rarer event, with few reports of UFH and dalteparin being the culprit agents. There has never been a report of enoxaparin causing bilateral psoas muscle hematoma [6, 7]. We present a case of an elderly woman with bilateral femoral nerve neuropathy following bilateral psoas muscle hematomas caused by enoxaparin.

Case Report

A 64-year-old woman with a history of hypertension, type 2 diabetes mellitus, and chronic obstructive pulmonary disease (COPD) presented with complaints of shortness of breath at rest. A computed tomography angiogram (CTA) of the chest revealed acute bilateral pulmonary emboli (PE) and a deep vein thrombosis (DVT) in the left popliteal vein, and she was started on enoxaparin at 1 mg/kg twice a day for treatment of acute DVT/PE. On day 10 of admission, the patient became hypotensive and was found to have a drop in hematocrit from 14 g/dL on admission to 5.8 g/dL. Prothrombin time was mildly increased to 15.0 (reference upper limit of normal is 12.8), INR was 1.3, and activated partial thromboplastin time was within normal limits. CT abdomen and pelvis showed bilateral psoas hematomas without extravasation (Figure 1). Magnetic resonance imaging (MRI) of the lumbar spine confirmed this finding.

Enoxaparin was discontinued, crystalloids and 4 units of blood were given, and an IVC filter was placed.

On day 16, the patient reported inability to move bilateral lower extremities, but did not report pain. Her hemodynamics and hematocrit at this point had stabilized.

A neurologic exam revealed normal strength in her arms. She was unable to elevate her legs against gravity or to extend



Figure 1. CT image showing bilateral retroperitoneal hematomas, larger on the right than the left (arrows).

her knees. She had normal arm reflexes, but absent leg reflexes. Her sensation could not be consistently assessed, although she reported that her right medial calf had less sensation than her arm.

Electromyography (EMG) suggested bilateral femoral sciatic nerve neuropathy, worse on the right than left. The cause of this finding was ascribed to the bilateral psoas hematomas.

She was treated conservatively with daily physical therapy. Her hospital course was extended due to complications from her COPD exacerbation. Follow-up imaging of the retroperitoneum and repeat EMG was not performed because the patient was clinically improved. At the time of discharge to a rehabilitation center 4 weeks after initial diagnosis of neuropathy, motor strength was 4/5 in bilateral lower extremities and she could ambulate minimally with a walker.

Discussion

LMWH, such as enoxaparin, typically have favorable adverse effect profiles and less bleeding risk, possibly due to greater inhibition of factor Xa than thrombin and less platelet inhibition [3,6]. However, severe hemorrhagic complications can occur and there are reports of large abdominal wall hematomas, retroperitoneal hemorrhages, and epidural hematomas developing following enoxaparin use [3].

The femoral nerve is the most commonly affected nerve in cases of retroperitoneal masses causing compression, because it is the largest branch of the lumbar plexus originating from the dorsal branches of the L2–L4 ventral rami, running superficially in the groove between the iliacus and the psoas [2–4]. Clinically, femoral neuropathy presents with weakness of the lower extremities, inability to flex the hips or extend the knees,

patellar hyporeflexia, and decreased sensation in the anterior thigh; in some cases, paresthesia precedes the motor symptoms [2,6–8]. There are 2 proposed mechanisms of nerve injury: decreased blood supply in the iliopsoas gutter causing ischemia and pressure-related damage from compression by the hematoma [4,8].

The etiology of the susceptibility of the iliopsoas to spontaneous hemorrhage is unclear, but a proposed theory is that micro-trauma from coughing, vomiting, and sports can, in the presence of a coagulopathy, result in hemorrhage. Another theory is that the iliopsoas is naturally predisposed to spontaneous intramuscular hemorrhage, as it is the strongest flexor in the body and is involved in numerous locomotive maneuvers [4,8].

Another important and life-threatening situation occurs when iliopsoas hematoma(s) result in hypotension and hypovolemic shock, depending on the extent of intramuscular hemorrhage [1,9]. Goodfellow et al., in an experiment on a fresh cadaver, found that the psoas muscle can hold large amounts of injected water (up to 10 times its capacity), whereas the iliacus was barely distensible. This demonstrates that a significant amount of the cardiac output (blood volume) can be lost in the retroperitoneum, leading to shock [10].

CT and MRI are the imaging modalities of choice for making the diagnosis once clinical suspicion is raised. The advantage of CT includes allowing prompt diagnosis to be made, particularly in the case of hemodynamic instability, and there is less discomfort to a patient who may be in pain from nerve ischemia. However, MRI can give more details of nerve root compression [5–6,8]. Ultrasonography (US) studies are limited because the psoas muscle is deep-seated, with overlying bowel [8,9].

Management is largely centered on resuscitative measures, blood transfusions, and crystalloid infusion for hemodynamic instability, in addition to discontinuation of anticoagulation and reversal of any coagulopathy [8,9]. In our patient, there were no coagulopathic parameters to be corrected. A review of case reports shows that both operative and conservative

approaches have been advocated, with similar outcomes [3–7]. The decision to intervene either surgically or percutaneously depends on the severity of the femoral nerve neuropathy with the aim of reversing pressure-induced nerve ischemia. Another indication is refractory hemodynamic instability failing to respond to conservative management [9]. Percutaneous decompression by either US or CT guidance has increasingly been utilized and provides a minimally invasive approach, particularly in those patients deemed to be poor surgical candidates, but has to be performed prior to the organization of the hematoma(s) [11]. An evolving strategy is trans-catheter arterial embolization in patients found to have active bleeding demonstrated by spiral CT. An adverse effect is the potential for spinal cord or peripheral nerve ischemia [7,9].

The prognosis is generally good, with up to 70% of patients regaining almost complete functionality with or without surgical intervention [3–9,11].

Conclusions

There have been case reports of spontaneous retroperitoneal hemorrhage causing femoral nerve compression in anticoagulated patients; however, enoxaparin has infrequently been reported to cause retroperitoneal hemorrhage. To the best of our knowledge, this is the only case report in which spontaneous psoas hematomas resulted in bilateral femoral neuropathy as a complication of enoxaparin anticoagulation. The constellation of bilateral groin pain, loss of lower extremity mobility, and decreased hematocrit raised the suspicion of massive blood loss into the cavity/compartments. Thus, a high index of suspicion should be maintained by clinicians when presented with such symptoms and signs, as there can be significant morbidity and mortality when prompt diagnosis is not made.

Conflicts of interest

None.

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