

Bilateral Femoral Nerve Compression by Iliopsoas Hematoma in Anticoagulated Patient of Coronavirus Disease-19 Pneumonia

ABSTRACT

A 64-year-old obese (body mass index [BMI] – 38) female, a known hypertensive with type 2 diabetes and hyperlipidemia, admitted to our hospital with fever and sore throat, diagnosed to have COVID-19. She needed invasive mechanical ventilation from hypoxia due to acute lung injury from severe COVID-19. She received IV Remdesivir for 10 days, IV methyl prednisolone 40 mg for 15 days, and enoxaparin 60 mg BID early in the course of the illness as her D-dimers were considerably elevated, as per hospital protocol. There was a significant drop in hemoglobin level during the course of admission. Incidentally detected was her unable to move both lower limbs. Weakness was sudden in onset; there was no associated back pain or abdominal pain. She had bilateral femoral nerve compression due to iliopsoas hematoma secondary to anticoagulation. Weakness gradually showed improvement with physiotherapy.

Key words: Anticoagulation, COVID-19, Femoral nerve compression, Iliopsoas hematoma

INTRODUCTION

The coronavirus disease (COVID)-19 pandemic originated in China in December 2019 spreading rapidly to many countries. Severe COVID-19 may lead to respiratory failure and acute respiratory distress syndrome, systemic thrombosis culminating in cardiovascular complications which are an important feature in the pathophysiology of the disease.^[1]

Hematoma in the iliopsoas muscle is a rare manifestation seen probably in patients receiving anticoagulation therapy or in patients with coagulopathy. It may present with severe pain in the affected groin, hip, and thigh and may lead to weakness in the quadriceps muscles.^[2]

We report here with a case of iliopsoas muscle hematoma that leads to bilateral (left more than right) femoral neuropathy in a patient receiving anticoagulation for COVID-19 illness.

CASE

A 64-year-old obese (body mass index [BMI] – 38) female, a known hypertensive with type 2 diabetes and hyperlipidemia, on therapy for 10 years, with metformin, glimepiride, telmisartan, clopidogrel, and atorvastatin. She was admitted to our hospital with fever and sore throat, for which she was treated for 7 days in another hospital.

On presentation, she was afebrile, tachypneic, and hypoxic on a non-rebreathing mask. All her vital parameters were relatively well maintained. She needed non-invasive ventilation support and was eventually shifted to invasive mechanical ventilation as she continued to be hypoxic due to acute lung injury from severe COVID-19.

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She was continued on mechanical ventilation with lung protective strategy for 9 days, following which she was weaned off. She was hemodynamically stable and was fully conscious.

She received IV Remdesivir for 10 days, IV methyl prednisolone 40 mg for 15 days, and enoxaparin 60 mg BID early in the course of the illness as her D-dimers were considerably elevated, as per hospital protocol. She continued to receive her antihypertensive and antidiabetic medications.

She developed mild hematuria on the 8th day of admission. Her enoxaparin and clopidogrel were discontinued which led to clearing of hematuria in a couple of days. On day 10 of intensive care unit (ICU) stay, her hemoglobin dropped from 9.7 g/dl to 7.5 g/dl with no evidence of blood loss. Her coagulation studies revealed the APTT to be twice the normal levels. There was no obvious occult blood seen in her stools. Her hemoglobin improved to 9.8 g/dl following transfusion of 2 units of packed cell; subsequently, no further fall in hemoglobin was seen. A prophylactic dose of enoxaparin 60 mg was reintroduced. A careful monitoring of the hemoglobin did not reveal a further drop in levels.

By the 11th day of admission, she was unable to move both her lower limbs. Weakness was sudden in onset; there was no associated back pain or abdominal pain. On examination, in the upper limbs, the power demonstrated was 4/5 in all muscle groups. Lower limbs power revealed left HIP flexor 2/5 right hip flexors 3/5, left hip abductor 2/5, right hip abductor 3/5, right knee extensors 3/5, and left knee extensor 2/5. Sensory system was essentially normal. Her total creatine phosphokinase and electrolytes were normal. Reflexes were absent in both lower limbs and proximal muscle weakness was more profound as compared to distal group.

Neurology opinion was sought. They suggested a differential diagnosis of (1) critical illness neuromyopathy, (2) acute inflammatory demyelinating polyradiculopathy, and (3) lower motor neurone, left more than right weakness.

On day 14, her repeat reverse transcription polymerase chain reaction for COVID was negative and she was shifted to regular ward.

An electromyography (EMG)/NCV was done.

EMG/NCV revealed that there was evidence of mild chronic motor axon degeneration affecting the muscles of lower limbs which could be at root level; decreased amplitude of left more than right femoral nerve – could not be properly evaluated due to obesity. There was no evidence of any active or ongoing motor axon degeneration and no evidence of any muscle disease.

Magnetic resonance imaging spine was done suggestive of large focal hematomas in the left iliopsoas and right iliacus muscles showing early subacute stages of bleeding as shown in Figures 1 and 2.

There was desiccation, diffuse bulge along with moderate postcentral protrusion of L4-L5 disc causing severe focal canal stenosis and bilateral neural foramina narrowing which was further accentuated by severe degenerative facet arthropathy and ligamentum flavum hypertrophy.

It was decided to restart enoxaparin for deep vein thrombosis prophylaxis following 2 days of cessation of anticoagulation therapy.

A repeat computed tomography (CT) scan done after 5 days of enoxaparin therapy did not suggest any further increase in the size of the hematoma. There was no further increase in muscle weakness or pain in the abdomen. She was then mobilized and was subsequently discharged from hospital and was asked to repeat CT scan after a month.

On follow-up after a month, she was able to move with support of a walking cane. CT scan showed a reduction in the size of hematoma. Physiotherapy was continued.

DISCUSSION

Spontaneous hemorrhage of the iliopsoas muscle followed by femoral nerve neuropathy is a rare complication possibly seen in patients on anticoagulation. The neuropathy is a result of the decreased blood flow to the surrounding epineurium

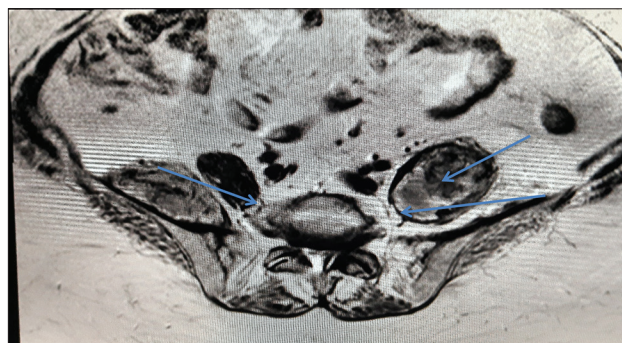


Figure 1: Femoral nerve compression and iliopsoas hematoma

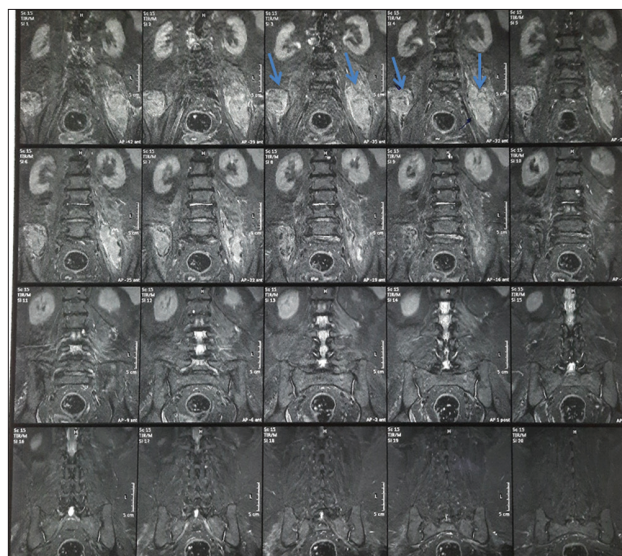


Figure 2: Left iliopsoas hematoma and right iliacus hematoma

or may be secondary to the pressure-related ischemia or neuropraxia.^[3]

In a study, the incidence of iliopsoas hematoma was 3.8 cases for 1000 admissions, during ICU stay. Only 10% of the patients in this study were above the therapeutic range of anticoagulation. Age, anticoagulation, a high BMI, and dialysis were independent risk factors for developing an iliopsoas hematoma in the ICU. The most frequent presentation was a drop of hemoglobin by >2 g/dl which was demonstrated in 75% of the patients and followed by shock in 50% of patients.^[4]

Some physicians suggest that the hematoma should be percutaneously drained under CT or ultrasound guidance and is against conservative therapy. Others advocate surgical decompression in the presence of significant femoral neuropathy compounded by the size of the hematoma. Blood transfusion and component therapy may be required.

The current patient probably had bilateral femoral nerve compression due to iliopsoas hematoma secondary to

anticoagulation. The possibility of aspiration of hematoma was discussed with the interventional radiologist but he was of the opinion that the hematoma had already organized and the CT aspiration would, probably be ineffective. Conservative approach was deemed appropriate for this patient.

CONCLUSION

This patient was diagnosed to have COVID-19 pneumonia and developed anticoagulation-induced iliopsoas hematomas, which may have led to femoral nerve compression and muscle weakness.

Iliopsoas hematomas may be missed in the ICU. Patients who develop sudden onset weakness in the lower limbs, associated with a drop in hematocrit with or without hypotension, should be screened for iliopsoas hematomas, more so if they are on anticoagulants. Factor Xa activity should be monitored while on anticoagulation with low-molecular-weight heparin, especially when patients are obese or have renal failure.

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How to cite this article: Amin PR, Jhaveri BN. Bilateral Femoral Nerve Compression by Iliopsoas Hematoma in Anticoagulated Patient of Coronavirus Disease-19 Pneumonia. *Bombay Hosp J* 2021;63(3):138-140

Source of support: Nil, **Conflicts of interest:** None

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