CASE REPORT

Spontaneous iliopsoas haematoma: a complication of hypertensive urgency

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SUMMARY

Iliopsoas haematoma is a rare clinical entity which can be life threatening in extreme cases. We are reporting a case of iliopsoas haematoma as a complication of hypertensive urgency. A 67-year old woman presented to emergency room with hypertensive urgency and hip pain. During hospitalisation, her haemoglobin was decreasing and on further evaluation, she did not have any signs of external bleeding and laboratory results were not suggestive of haemolysis. CT scan of abdomen and pelvis revealed a spontaneous iliopsoas haematoma. A likely explanation for this presentation in the absence of coagulopathy and trauma is very high blood pressure. Patient was on low-dose aspirin at home which could have further aggravated her bleeding due to platelet dysfunction. She was managed conservatively with blood transfusions and blood pressure was reduced to target after which she recovered.

BACKGROUND

Iliopsoas haematoma is a rare clinical entity which can be life threatening in extreme cases. The muscle is deep seated and usually does not sustain trauma and the bleeding is mostly spontaneous due to a coagulopathy. The clinical symptoms can be non-specific hip, groin or back pain with no external evidence of mass on examination and can be overlooked as a musculoskeletal pain. Moreover, hypertensive urgency is not thought to be an aetiology of spontaneous iliopsoas haematoma, though it could be associated with other target organ damage. We report a case of iliopsoas haematoma secondary to hypertensive urgency. Aspirin could have further aggravated the bleeding due to platelet dysfunction. The chance of missing an iliopsoas haematoma is high in this clinical scenario because the very high blood pressure usually does not suggest active bleeding. Delay in diagnosis of this condition can be fatal. This case is written in the intention to enlighten physicians and increase awareness of spontaneous iliopsoas haematoma as an unknown possible complication of hypertensive urgency.

CASE PRESENTATION

A 67-year old woman presented to emergency room with a dull pain in the left hip and thigh, non-radiating and with an intensity of 9/10 for 3 days duration. Her pain was exacerbated with movement of her left hip on ambulation. She denied any trauma to her hip and stated she never had any pain to her joints before this. The pain was increasing in intensity over 3 days and she decided to seek medical attention. She also stated to the

emergency room physician that her blood pressure was uncontrolled for the past 2 weeks and her anti-hypertensive medication was increased by her primary care physician. Despite the increase in her medications, her blood pressure remained uncontrolled with most of her blood pressure readings more than 200/100 mm Hg at home. She denied abdominal pain, flank pain, haematuria, blurred vision, chest pain and shortness of breath. She has a history of hypertension, type 2 diabetes mellitus and chronic kidney disease stage 3. She did not have any history of spontaneous or prolonged bleeding. Her family history did not reveal any bleeding disorders. Her home medications were repaglinide, ergocalciferol, aspirin, esomeprazole, nifedipine, atorvastatin and furosemide. She was in distress due to pain in her left hip during physical examination. Her blood pressure was 222/94 mm Hg and her pulse rate was 77/min. Physical examination revealed tenderness of the left thigh. The left hip was in a flexed position and extension was limited by pain. In contrast her right hip had normal range of motion without any tenderness. There was no external bruise noted over the thigh. There were no other positive findings.

INVESTIGATIONS

A complete blood count showed a white count of 4.2 K/ μ L and a platelet count of 159 000 which was within normal limits. However, she had a normochromic, normocytic anaemia with haemoglobin of 10 g/dL due to her chronic kidney disease. Her creatinine had increased from 2.9 mg/dL (blood urea nitrogen (BUN) 39 mg/dL) at her baseline 2 months ago to 3.8 mg/dL (BUN 55 mg/dL). A urine analysis showed five red blood cells with no active sediments. An ultrasoundogram of her kidneys showed reduced size of both kidneys with increased echogenicity and without any evidence of obstruction. Owing to severe left hip pain, an X-ray of the hip was carried out and no fracture or osteoporosis was noted. The patient was started on analgesics to control pain. However, since admission over 4 days, a progressive drop in haemoglobin was noted, from 10.1 to 7.3 g/dL, with no haemodynamic compromise. On further evaluation, she did not have any signs of external bleeding, stool occult blood test was negative, laboratory results were not suggestive of haemolysis and a CT scan of abdomen and pelvis without intravenous contrast, that was carried out due to her chronic kidney disease to look for internal bleeding, revealed a spontaneous iliopsoas haematoma (figures 1 and 2).

Her baseline coagulation profile was normal. She was not on anticoagulation at home. After the CT

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Figure 1 CT of abdomen and pelvis axial view showing left iliopsoas haematoma.

imaging showed iliopsoas haematoma, a coagulation profile was performed, which was within normal range.

DIFFERENTIAL DIAGNOSIS

- ▶ Hip fracture;
- ▶ Hip arthritis;
- ▶ Muscular strain;
- ▶ Spontaneous iliopsoas haematoma (was not thought about in the initial evaluation).

TREATMENT

Patient was managed conservatively with blood transfusions and her blood pressure was controlled.

OUTCOME AND FOLLOW-UP

A CT was repeated and did not show expansion of the haematoma and haemoglobin remained stable (figures 3 and 4). The patient received physiotherapy and had remarkable improvement in her hip and thigh pain on discharge. Her renal function returned to her baseline before discharge.

Figure 2 CT of abdomen and pelvis coronal view showing left iliopsoas haematoma.

Figure 3 Follow-up CT of abdomen and pelvis axial view showing stable left iliopsoas haematoma.

DISCUSSION

Iliopsoas haematoma is a rare clinical entity which has been well described in literature in the context of coagulopathy. It was first described by Tallroth¹ in a patient with haemophilia. In 1966, Debolt and Jordan reported the first two cases of heparin-induced iliopsoas haematomas presenting with femoral neuropathy.²

The aetiology of iliopsoas haematoma is classified into spontaneous and traumatic haematoma. Spontaneous iliopsoas haematoma could be unilateral or occasionally bilateral,³ and commonly occurs as a consequence of coagulopathy secondary to anticoagulation medications⁴ and haemophilia.^{5,6} There have been few reported cases of iliopsoas haematoma associated with alcoholic liver cirrhosis.⁷ Coronary angiography and percutaneous coronary intervention can be complicated with retroperitoneal hematomas^{8,9} as a consequence of access site-related bleeding and the concurrent use of anticoagulation and potent antiplatelet drugs and rarely have there been reported cases of iliopsoas haematoma.¹⁰ Spontaneous iliopsoas haematoma has been reported after cardiac surgeries but is an effect of the perioperative use of anticoagulants and antiplatelets, and the diagnosis in these patients could be challenging in the setting of sedation.¹¹

Figure 4 Follow-up CT of abdomen and pelvis coronal view showing stable left iliopsoas haematoma.

Significantly elevated blood pressure could be associated with acute ongoing target organ damage involving multiple systems. Acute left ventricular dysfunction, myocardial infarction, ischaemic stroke, haemorrhagic stroke, hypertensive encephalopathy, hypertensive retinopathy, aortic dissection and acute kidney injury are some of the complications associated with elevated blood pressure. This possibly explains the likelihood of an unknown complication of spontaneous iliopsoas haematoma. In fact, the high blood pressure delayed the diagnosis of ongoing bleeding. Our patient also had deterioration of her renal function as a complication of elevated blood pressure. There have been no reported cases of iliopsoas haematoma associated with hypertensive urgency.

Our patient was on aspirin, which could have further aggravated the bleeding due to platelet dysfunction. Platelet dysfunction is usually associated with spontaneous skin, mucosal and central nervous system bleeding but is not associated with spontaneous deep visceral haematoma and haemarthrosis in the absence of coagulopathy. Spontaneous retroperitoneal haematoma with the use of ticlopidine, an antiplatelet agent, has been described in a case report; however, that patient also had concurrent coagulopathy.¹² We believe that the elevated blood pressure would have been the trigger for spontaneous iliopsoas haematoma and aspirin would have further aggravated the ongoing bleeding.

The clinical symptoms of iliopsoas haematoma vary from hip pain, thigh pain, back pain radiating to the groin and neurological symptoms secondary to compression of the femoral nerve. Some patients may demonstrate a psoas sign with exaggeration of the pain on passive extension and the affected hip would be maintained in a flexed position. Massive retroperitoneal haematoma may cause ecchymotic lesions in the flanks (Grey Turner's sign) or in the periumbilical area (Cullen's sign). Rarely, patient could have hypovolemic shock.

Diagnosis is made by imaging. The choice of initial imaging for diagnosis would be CT scan due to its free availability and therefore, is utilised frequently. MRI is more sensitive and specific but its use is restricted as it is time consuming, expensive and cannot be used in patients with metallic implants. X-rays and ultrasonogram could be useful but are limited by the lack of specificity and sensitivity. This patient had an X-ray of the hip which ruled out fractures; however, further imaging was not done initially as the possibility of haematoma was not considered in the context of absence of trauma and coagulopathy. When the patient's haemoglobin started dropping, CT scan was carried out and revealed a haematoma.

The management options can be conservative or surgical depending on the haemodynamic stability, the size of the haematoma, ongoing bleeding and extent of neurological impairment. Conservative management is recommended for mild bleeding and surgical decompression is the choice for severe haemorrhage or significant compressive femoral neuropathy. Various surgical techniques have been explored, such as open surgical decompression or percutaneous decompression.¹³ Transarterial embolisation¹⁴ has been utilised to control the bleeding of iliopsoas haematomas secondary to anticoagulant therapy, and has been found to be safe and successful in patients with high risk from surgery. Our patient had no haemodynamic instability or evidence of femoral nerve compression. Repeat imaging did not show expansion of the haematoma. So observation with control of her blood pressure was the therapeutic measure used.

In conclusion, iliopsoas haematoma is a rare clinical entity which could present as a hip pain and can be fatal if not diagnosed in a timely manner. High blood pressure can be a risk factor, though so far this is not reported in the literature. We report here a case who presented with hypertensive urgency and spontaneous iliopsoas haematoma.

Learning points

- ▶ New onset worsening hip and thigh pain should raise the suspicion for iliopsoas haematoma.
- ▶ Absence of coagulopathy, not on anticoagulants and absence of trauma does not preclude the possibility iliopsoas haematoma.
- ▶ Iliopsoas haematoma should be considered in any patient with uncontrolled hypertension and new onset hip pain, and early CT imaging is mandatory and can be life-saving.

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Competing interests None.

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REFERENCES

- 1 Tallroth A. Hemophilia with spontaneous hemorrhage in the iliopsoas muscle followed by injury to the femoral nerve: report of a case. *Acta Chir Scand* 1939;92:1–9.
- 2 DeBolt WL, Jordan JC. Femoral neuropathy from heparin hematoma. Report of two cases. *Bull Los Angeles Neurol Soc* 1966;31:45–50.
- 3 Wada Y, Yanagihara C, Nishimura Y. Bilateral iliopsoas hematomas complicating anticoagulant therapy. *Intern Med* 2005;44:641–3.
- 4 Sasson Z, Mangat I, Peckham KA. Spontaneous iliopsoas hematoma in patients with unstable coronary syndromes receiving intravenous heparin in therapeutic doses. *Can J Cardiol* 1996;12:490–4.
- 5 Balkan C, Kavakli K, Karapinar D. Iliopsoas haemorrhage in patients with haemophilia: results from one centre. *Haemophilia* 2005;11:463–7.
- 6 Dauty M, Sigaud M, Trossaert M, et al. Iliopsoas hematoma in patients with hemophilia: a single-center study. *Joint Bone Spine* 2007;74:179–83.
- 7 Sugiyama C, Akai A, Yamakita N, et al. Muscle hematoma: A critically important complication of alcoholic liver cirrhosis. *World J Gastroenterol* 2009;15:4457–60.
- 8 Farouque HM, Tremmel JA, Raissi Shabari F, et al. Risk factors for the development of retroperitoneal hematoma after percutaneous coronary intervention in the era of glycoprotein IIb/IIIa inhibitors and vascular closure devices. *J Am Coll Cardiol* 2005;45:363–8.
- 9 Smilowitz NR, Kirtane AJ, Guiry M, et al. Practices and complications of vascular closure devices and manual compression in patients undergoing elective transfemoral coronary procedures. *Am J Cardiol* 2012;110:177–82.
- 10 Raja Y, Lo TS, Townend JN. Don't rule out retroperitoneal bleeding just because the angiogram was done from the radial artery. *J Invasive Cardiol* 2010;22:E3–4.
- 11 Une D, Shimizu S, Nakanishi K. Bilateral iliopsoas hematomas under sedation: a complication of postoperative therapy after coronary artery bypass grafting. *Acta Med Okayama* 2010;64:71–3.
- 12 Nakao A, Sakagami K, Mitsuoka S, et al. Retroperitoneal hematoma associated with femoral neuropathy: a complication under antiplatelets therapy. *Acta Med Okayama* 2001;55:363–6.
- 13 Holscher RS, Leyten FS, Oudenhoven LF, et al. Percutaneous decompression of an iliopsoas hematoma. *Abdom Imaging* 1997;22:114–16.
- 14 Zissin R, Gayer G, Kots E, et al. Transcatheter arterial embolisation in anticoagulant-related haematoma—a current therapeutic option: a report of four patients and review of the literature. *Int J Clin Pract* 2007;61:1321–7.

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