Case Series

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Spontaneous Iliopsoas Haematoma in Patients Undergoing Haemodialysis- An Enigma: A Case Series

Internal Medicine Section

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ABSTRACT

Chronic Kidney Disease (CKD) progression is associated with an increased risk of bleeding due to various reasons, some of which are iatrogenic. Bleeding at unusual sites, such as the iliopsoas muscle, is difficult to suspect as awareness about this condition is sparse, and the literature about the reasons for this condition is unclear. Iliopsoas bleed continues to be an enigma, and herein, the authors present a case series of three cases of spontaneous iliopsoas haematoma. The first case was a 64-year-old male with acute coronary syndrome and CKD on dual antiplatelet agents who had an iliopsoas bleed after dialysis. The second patient was a 51-year-old male with end-stage renal disease on Maintenance Haemodialysis (MHD) who bled into the iliopsoas after anticoagulation for Coronavirus Disease-2019 (COVID-19) related illness. The third patient was a 52-year-old female who had a Catheter-Related Bloodstream Infection (CRBSI) and Atrial Fibrillation (AF) requiring anticoagulation, and she developed an abdominal wall haematoma extending upto the iliopsoas. The authors highlighted the sudden occurrence, challenges in diagnosis and management of iliopsoas bleed in CKD, with a stress on the careful use of anticoagulation.

Keywords: Anticoagulation, Apixaban, Bleeding, Chronic kidney disease, Renal replacement therapy

INTRODUCTION

Iliopsoas haematoma is a rare complication associated with trauma, anticoagulation therapy, and haemorrhagic diathesis [1]. The reported incidence of retroperitoneal haematoma varies from 0.1 to 0.6% [2]. Old age, the use of anticoagulation, and CKD are known risk factors [2]. The clinical presentation may vary from mild pain to abrupt shock leading to cardiovascular collapse, which, if not diagnosed early, leads to adverse outcomes [3]. The exact mechanism is not known, but hypotheses include small vessel diffuse arteriosclerosis, forceful muscle strain, and heparin-induced immune microangiopathy [3]. Spontaneous iliopsoas haematoma in haemodialysis patients often goes unnoticed. The present series reports three cases of symptomatic spontaneous iliopsoas haematoma that required specific management.

Case 1

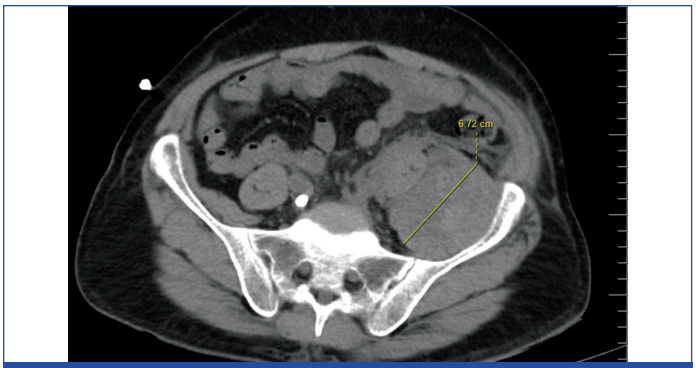
A 64-year-old male was admitted to the Department of Nephrology with complaints of bilateral pedal oedema lasting seven days and left-sided anginal chest pain for the past two days. The patient also had dyspnoea associated with a dry cough, which progressed to orthopnoea over the next two days. He has been diabetic for the past 20 years with a history of proliferative diabetic retinopathy. For the last year, he had been on dual antiplatelet agents (aspirin 75 mg and clopidogrel 75 mg once daily) for ischaemic heart disease (left ventricular ejection fraction 35%). The patient was also diagnosed with CKD-diabetic nephropathy during the same period, with a creatinine level of 2.5 mg/dL. Clinical examination revealed findings of accelerated hypertension with a blood pressure of 200/120 mmHg and acute pulmonary oedema. The patient was hypoxic and required oxygen support. Investigations revealed a haemoglobin level of 8.1 g/dL, serum creatinine of 3.2 mg/dL, platelet count of 1.75 lacs/cumm, and normal coagulation profile (prothrombin time 11.4 seconds, control 12 seconds, Activated Partial Thromboplastin Time (APTT) 29.4 seconds, control 30 seconds).

The patient was initiated on haemodialysis via a right femoral non tunneled catheter and underwent three sessions of haemodialysis. The patient's Electrocardiogram (ECG) showed T inversion in lateral leads with serial elevated troponin I. A diagnosis of acute coronary syndrome- non ST segment elevation myocardial infarction was made, and he was started on anticoagulation (heparin 5000 units subcutaneous every six hours). A coronary angiogram was planned; however, the patient developed severe pain in both thighs with a drop in haemoglobin of 2 g/dL. A Computed Tomography (CT) scan showed bilateral iliopsoas haematoma (left more than right, approximately 100 cc). Heparin was discontinued, and he received one unit of packed red cell transfusion, as well as cryoprecipitate. The patient underwent saline haemodialysis during the hospital stay and received analgesics for pain (tablet paracetamol 650 mg thrice daily for five days and tramadol 50 mg IV when required). Due to personal reasons, the patient was discharged against medical advice with a single antiplatelet agent as he had acute coronary syndrome, and he then expired at his residence four days after the discharge.

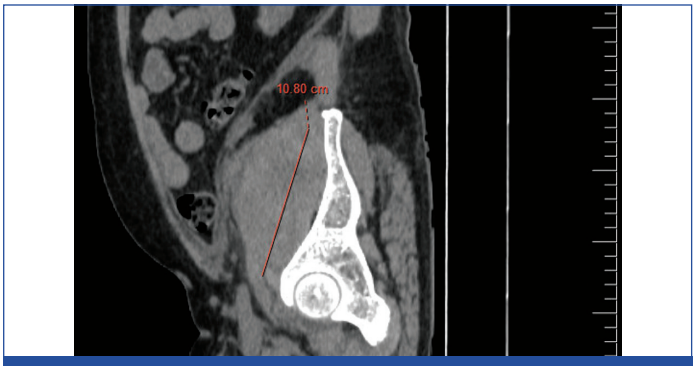
Case 2

A 51-year-old male was admitted to the Department of Nephrology with the chief complaint of intermittent low-grade fever for the past six days. He also experienced cough with scanty mucoid expectoration and dyspnoea. There was no history of chest pain. The patient had been on two classes of antihypertensive drugs for five years and was a known case of CKD on MHD for three years. Due to multiple access failures, the patient was undergoing haemodialysis through a right femoral tunneled catheter. He had been taking a single antiplatelet, aspirin 75 mg, once daily for three years (reason unknown). Physical examination revealed a blood pressure of 130/80 mmHg, pallor, oedema, tachypnoea, hypoxia, and bibasal crepitations. The patient tested positive for Coronavirus Disease-2019 (COVID-19) by Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) and was treated with nasal oxygen, anticoagulation (heparin 5000 units subcutaneous twice daily), and steroids (inj. hydrocortisone 100 mg twice daily). Aspirin was continued, and he recovered from COVID-19 illness. On the 19th day of admission, after a session of haemodialysis (with standard anticoagulation), the patient developed severe pain over the left lower abdomen, radiating to the lower limb, and experienced pain on flexion of the left hip. His haemoglobin had dropped by 1.5 g/dL (from 7.5 g/dL to 6 g/dL), platelet count was 1.92 lacs/cumm, and coagulation profile was normal (prothrombin time 12.5 seconds,

control 12 seconds, APTT 28.3 seconds, control 30 seconds). There was no haemodynamic instability. Ultrasound followed by Computed Tomography (CT) scan [Table/Fig-1,2] revealed a haematoma in the left ilacus muscle measuring approximately 11.7×7.8×6.8 cm (310 cc). Aspirin and heparin were stopped. The patient underwent saline haemodialysis and required three units of packed red cell transfusion. Haemoglobin and Packed Cell Volume (PCV) were monitored daily until the haemoglobin stabilised (haemoglobin at discharge was 8 g/dL). Pain was managed with analgesia (paracetamol 650 mg for five days and tramadol 50 mg as required). The patient responded well to this conservative management and had an uneventful recovery. On follow-up, he continued to remain on MHD with stable haemoglobin.



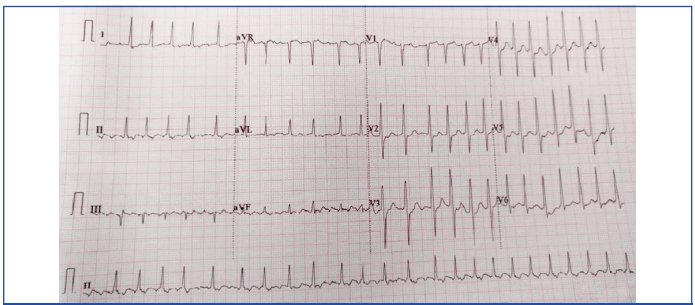
[Table/Fig-1]: CT axial section image showing left iliopsoas haematoma (case 2).



[Table/Fig-2]: CT saggital section showing left iliopsoas haematoma (case 2).

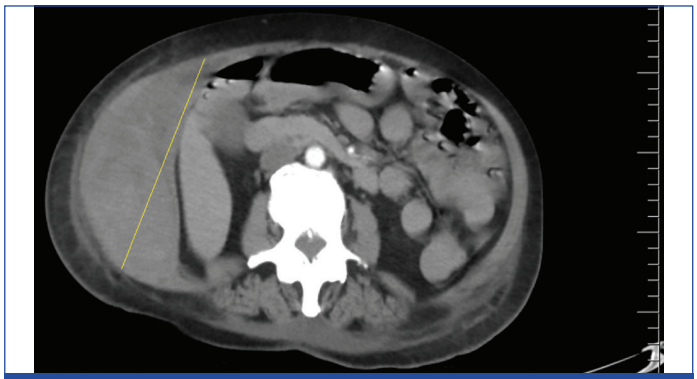
Case 3

A 52-year-old female was admitted to the Department of Nephrology with the chief complaint of high-grade fever with chills, cough with mucoid expectoration, and dyspnoea on exertion for the past two days. She also had a history of retrosternal chest pain for one day, associated with palpitations. The patient had been hypertensive for the past year and required three classes of antihypertensive drugs. She was recently diagnosed with CKD due to chronic interstitial nephritis and had initiated haemodialysis via a right temporary Internal Jugular Catheter (IJC) for the last month. On examination, the patient had an irregular pulse rate of 160/minute and BP of 160/100 mmHg. She was tachypneic with a respiratory rate of 22 breaths/minute. Respiratory examination showed bibasal crepitations. The ECG was suggestive of AF with a fast ventricular rate [Table/Fig-3].



[Table/Fig-3]: Electrocardiogram showing Atrial Fibrillation (AF) with fast ventricular rate (case 3).

Investigations revealed a haemoglobin level of 8 g/dL, total count of 16.8 cumm, serum creatinine of 10.1 mg/dL, serum sodium of 133 meq/L, serum potassium of 5.9 meq/L, and serum calcium of 10.4 mg/dL. The platelet count was 2.93 lacs/cumm, and the coagulation profile was normal (prothrombin time 13.2 sec, control 12 sec, APTT 28.7 sec, control 30 sec). The patient was treated with urgent haemodialysis via a right IJC. For AF, she was anticoagulated with heparin 5000 units subcutaneous for five days, followed by apixaban 2.5 mg twice daily, and received amiodarone infusion for two days, after which the AF reverted. The patient was also diagnosed with CRBSI as the blood culture from the peripheral line and catheter was positive. Hence, the right IJC was removed after a session of haemodialysis, and she was treated with intravenous antibiotics (inj. vancomycin 1gm i.v. every 72 hours and inj. amikacin 500 mg i.v. every 48 hours, total of 5 doses each), after which the patient became afebrile. On the third day of admission, the left brachiocephalic AV Fistula (AVF) was cannulated; however, she developed pain and swelling at the AVF site suggestive of haematoma. Therefore, a left femoral non tunneled catheter was secured (dose of heparin was withheld preprocedure). On the seventh day (two days after starting apixaban-patch test not done), she developed a sudden onset of pain and swelling in the right lower abdomen with hypotension (BP 80/50 mmHg). A drop in haemoglobin by 4 g/dL (from 8 g/dL to 4.2 g/dL) was noted. The patient was managed with discontinuation of apixaban, four units of packed red cell transfusion, and required inotropic support (noradrenaline). CT scan showed a large intramuscular haematoma [Table/Fig-4] involving the right lateral chest wall, lateral abdominal wall, extending upto the level of the right iliac bone (600 cc) with no active contrast extravasation. In view of haemodynamic instability, she was planned for intervention. However, the procedure was deferred as the patient showed improvement with blood transfusion and was off inotropes within the next 48 hours. Daily abdominal girth monitoring was done along with haemoglobin and PCV (haemoglobin stabilised at 8 gm/dL with no further drop). The patient received analgesia for pain (tab paracetamol 650 mg thrice daily for five days and inj. tramadol 50 mg i.v. when required) and underwent saline haemodialysis. The patient improved with conservative management and had an uneventful recovery. She had stable haemoglobin levels during her subsequent follow-up after 10 days.



[Table/Fig-4]: CT axial section showing abdominal wall haematoma (case 3).

DISCUSSION

Iliopsoas haematoma is a rare complication associated with trauma, anticoagulation therapy, and haemorrhagic diathesis [1]. The incidence of spontaneous retroperitoneal haematoma in patients on anticoagulation is 0.1-0.6%, irrespective of kidney disease. Risk factors include elderly individuals, anticoagulation therapy, CKD, and those with bleeding abnormalities [2]. The present case series reported three cases of symptomatic spontaneous iliopsoas haematoma that warranted intervention in the form of blood transfusion, and one patient had haemodynamic instability requiring vasopressors.

Acute onset abdominal or thigh pain was the presenting symptom in index patients. In a case report by AbdAlgayoum RTA et al., the patient had abdominal and thigh pain with circulatory collapse [3]. Other signs described include Grey Turner sign and Cullen sign (ecchymotic lesions in the flank and periumbilical area, respectively) [3]. Due to compression anywhere along the course of the femoral nerve, patients can present with weakness of the iliopsoas, paralysis of the quadriceps, paresthesia in the anteromedial aspect of the lower extremity, and loss of the knee jerk [4]. Patients with CKD present a dilemma in that they are simultaneously prothrombotic and also have an increased bleeding tendency, especially in patients undergoing haemodialysis due to the use of anticoagulation. Pathophysiological factors attributable to this bleeding risk are uremic platelet dysfunction, anaemia, use of antiplatelet agents, as well as anticoagulation. Iliopsoas haematoma should be suspected when they present with an acute onset of abdominal or thigh pain with difficulty in flexing the hip, as these might be the only presenting complaints along with a drop in haemoglobin [2,3].

All three patients presented with a cough due to infection/pulmonary oedema. Vigorous cough reflex could have caused minor trauma to the microcirculation and possibly lead to spontaneous bleeding in anticoagulated patients [1,2]. Literature review suggests that the pathophysiology of spontaneous retroperitoneal bleeding is due to diffuse occult vasculopathy and arteriosclerosis of the small vessels, which may be friable. Once bleeding starts and the haematoma expands at the microvascular level, the large vessels are stretched and ruptured [5,6]. In a study done by Berna JD et al., they attributed the causes to anticoagulation-induced immune microangiopathy and minor trauma to the microcirculation by vomiting/coughing in the presence of anticoagulation [7].

Two of the patients were on heparin, and one was on apixaban before the onset of spontaneous bleeding. In a review article by Hughes S et al., in patients with advanced renal failure receiving a therapeutic dose of unfractionated heparin, there is impaired clearance due to saturation of reticuloendothelial cells. Due to interpatient variability of accumulation, there can be an unpredictable anticoagulant response [8]. Apixaban is highly protein-bound, and several studies suggest it can accumulate in CKD and is poorly dialyzable [9,10]. The use of anticoagulation in advanced CKD and patients on haemodialysis is a challenge, as evidence from large randomised controlled trials is limited. Guidelines for the use of anticoagulation in the general population cannot be extrapolated for advanced CKD as the risk of bleeding is very high [11]. The choice of anticoagulation must be individualised, and it is preferable to use unfractionated heparin as it has a short duration of action, and its effects are easily reversed with protamine.

All three patients were managed conservatively with blood products and supportive care, and none required endovascular intervention. Vital signs should be closely monitored along with frequent haemoglobin and PCV monitoring. Haemodynamic instability with a persistent drop in haemoglobin would warrant an endovascular procedure. In a study done by Llitjos JF et al., and Artzner T et al., 31% and 53% of patients required endovascular or surgical intervention, respectively [2,12]. Early detection of haematoma with timely cessation of anticoagulation might have helped us to avoid endovascular interventions in two patients. If conservative measures fail or the patient is unstable, endovascular selective intra-arterial embolisation or deployment of stent-grafts over the punctured vessel is the treatment of choice. Open surgical repair should be reserved for cases when there is a failure of both conservative and endovascular procedures [13].

CONCLUSION(S)

The present case series of iliopsoas bleed in CKD highlights the increased risk of spontaneous iliopsoas haematoma associated with the use of anticoagulation in patients on haemodialysis. The choice and dose of anticoagulation should be individualised. It is preferable to use short-acting and easily reversible anticoagulation. A high index of suspicion is required for patients presenting with pain and a drop in haemoglobin for early detection of iliopsoas haematoma and to avoid potentially fatal sequelae. There is a need for larger randomised controlled trials to evaluate the use of anticoagulation in advanced CKD.

REFERENCES

[1] Tavone AM, Giuga G, Attanasio A, Petroni G, Mauriello S, Cordova F, et al. A rapid fatal outcome of iliopsoas hematoma: Clinical and autopsy findings. J Investig Med High Impact Case Rep. 2022;10:23247096221111760.
[2] Llitjos JF, Daviaud F, Grimaldi D, Legriel S, Georges JL, Guerot E, et al. Ilio-psoas hematoma in the intensive care unit: A multicentric study. Ann Intensive Care. 2016;6(1):01-06.
[3] AbdAlgayoum RTA, Hassan Z, Singapori M, Khanchandrani N. Spontaneous psoas hematoma secondary to anticoagulation. Consultant. 2022;62(1):e17-18.
[4] DeBolt WL, Jordan JC. Femoral neuropathy from heparin hematoma. Report of two cases. Bull Los Angeles Neurol Soc. 1966;31(2):45-50.
[5] Torres GM, Cernigliaro JG, Abbitt PL, Mergo PJ, Hellein VF, Fernandez S, et al. Iliopsoas compartment: Normal anatomy and pathologic processes. Radiographics. 1995;15(6):1285-97.
[6] Qanadli SD, El Hajjam M, Mignon F, Bruckert F, Chagnon S, Lacombe P. Life-threatening spontaneous psoas haematoma treated by transcatheter arterial embolization. Eur Radiology. 1999;9:1231-34.
[7] Berna JD, Zuazu I, Madrigal M, Garcia-Medina V, Fernandez C, Guirado F. Conservative treatment of large rectus sheath hematoma in patients undergoing anticoagulant therapy. Abdominal Imaging. 2000;25:230-34.
[8] Hughes S, Szeki I, Nash MJ, Thachil J. Anticoagulation in chronic kidney disease patients-the practical aspects. Clin Kidney J. 2014;7(5):442-49.
[9] Steuber TD, Shiltz DL, Cairns AC, Ding Q, Binger KJ, Courtney JR. A multicenter analysis of factors associated with apixaban-related bleeding in hospitalized patients with end-stage renal disease on hemodialysis. Ann Pharmacother. 2017;51(11):954-60.
[10] Jha VK, Jairam A, Mahapatra D. Newer oral anticoagulant in chronic kidney disease: What we should know. J Assoc Physicians India. 2019;67(11):60-65.
[11] Aursulesei V, Costache II. Anticoagulation in chronic kidney disease: From guidelines to clinical practice. Clin Cardiol. 2019;42(8):774-82.
[12] Artzner T, Clere-Jehl R, Schenck M, Greget M, Merdji H, De Marini P, et al. Spontaneous ilio-psoas hematomas complicating intensive care unit hospitalizations. PLoS One. 2019;14(2):e0211680.
[13] Chan YC, Morales JP, Reidy JF, Taylor PR. Management of spontaneous and iatrogenic retroperitoneal haemorrhage: Conservative management, endovascular intervention or open surgery? Int J Clin Pract. 2008;62(10):1604-13.

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