

Iliopsoas hematoma associated with low-molecular-weight heparin use: A case report

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Abstract

Iliopsoas hematoma is an uncommon clinical entity that may develop in association with anticoagulation states, coagulopathies and hemodialysis, or anticoagulant therapy. Here, we report a case of unilateral iliopsoas hematoma in a 60-year-old man who received low-molecular-weight heparin for anticoagulation due to continuous veno-venous hemofiltration. The patient presented with fever and productive cough for 2 days. He received continuous veno-venous hemofiltration due to rising blood urea nitrogen (22.7 mmol/L; normal references: 3.2–7.1 mmol) and creatinine (1345 μ mol/L; normal references: 53–106 μ mol/L). Low-molecular-weight heparin (enoxaparin, 3500–5500 Da, 5–10 IU/kg/h) was delivered continuously by pumps for anticoagulation therapy. At day 12 post heparin treatment, the patient complained left back pain. Platelet count ($243 \times 10^9/L$) was normal, but both activated partial thromboplastin time (67.5 s) and prothrombin time (17.3 s) were prolonged. Abdominal computed tomography scan revealed left iliopsoas swelling with an indistinct border. Low-molecular-weight heparin was discontinued. Anti-Xa was not monitored throughout the treatment. No improvement was seen, and 3 days later, the patient died after his family decided to terminate therapy. This case highlights the needs for careful anticoagulation as well as close monitoring, and particularly the use of anti-Xa to guide the treatment.

Keywords

Iliopsoas hematoma, low-molecular-weight heparin, anticoagulation, continuous veno-venous hemofiltration

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Introduction

Iliopsoas hematoma is a distinct clinical entity that can present as a rare life-threatening event^{1,2} and may develop in association with anticoagulation states, coagulopathies and hemodialysis, or anticoagulant/antithrombotic therapy.^{3–5} Iliopsoas hematoma may occur unilaterally or bilaterally.⁶ Here, we report a case of unilateral iliopsoas hematoma in a 60-year-old man who received low-molecular-weight heparin for anticoagulation due to continuous veno-venous hemofiltration (CVVH), without anti-Xa monitoring. This case highlights the need for careful anticoagulation as well as close monitoring, and particularly the use of anti-Xa to guide the treatment.

Case report

A 60-year-old man presented with fever and productive cough for 2 days. He had a history of type 2 diabetes for 12 years and received peritoneal dialysis for diabetic nephropathy for

5 years. He also received antihypertensive drugs for hypertension for 10 years. There was no personal or family history of coagulopathy or stroke, trauma, chest pain, or illicit intravenous drug use.

Admission physical examination revealed the patient to be febrile (39.1°C), tachycardic with a pulse of 112/min, a respiratory rate of 22/min, and hypertensive with a blood pressure of 170/80 mm Hg. Bibasilar crackles were heard on auscultation. The peritoneal dialysis catheter was correctly positioned in the right lower abdomen, with no local erythema. There was no exudate buildup in catheter dressings.

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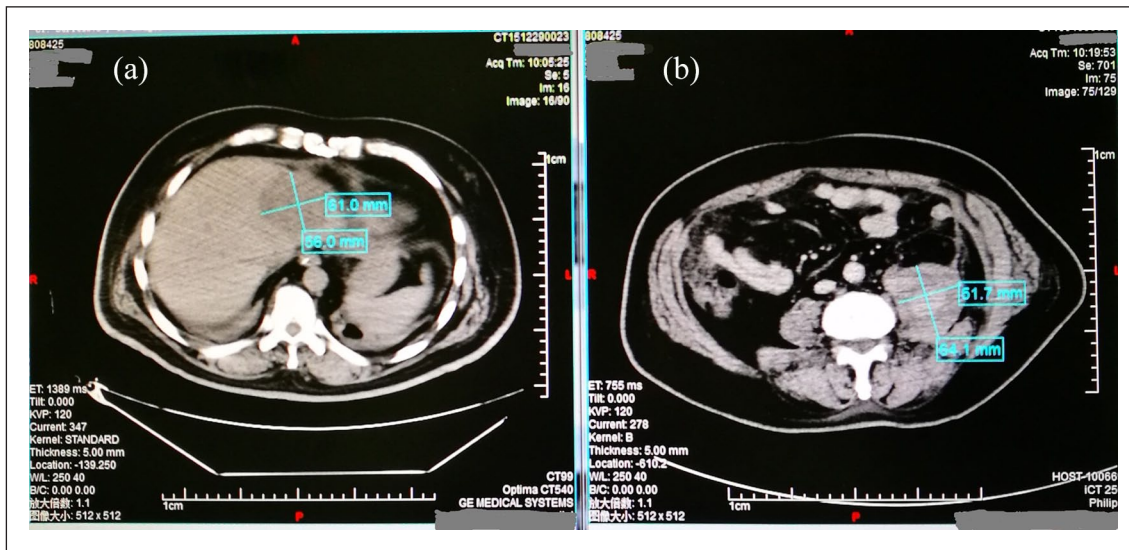


Figure 1. Abdominal CT scan of a 60-year-old man who received low-molecular-weight heparin for anticoagulation in continuous veno-venous hemofiltration: (a) CT scan at day 5 shows that a hypointense shadow in the left hepatic lobe with an indistinct border, and (b) abdomen CT scan at day 16 reveals left iliopsoas swelling, with inhomogeneous densities, and occasional hyperintense opacities and an indistinct border.

His systemic examination was otherwise unremarkable, with no tenderness, ecchymosis, or other external signs of trauma.

Initial blood workup showed neutrophilic leukocytosis with a white blood cell count of $12.8 \times 10^9/L$ (normal reference: $4.0\text{--}10 \times 10^9/L$) and 82.6% neutrophils (normal reference: 50%–70%). The patient was anemic (hemoglobin 95 g/L; normal reference: 120–160 g/L), with a normal platelet count ($181 \times 10^9/L$; normal reference: $100\text{--}300 \times 10^9/L$). Both his prothrombin time (PT; 12.1 s, normal reference: 10–14 s) and activated partial thromboplastin time (aPTT; 34.1 s, normal reference: 20–40 s) were normal. Blood chemistry showed elevated blood urea nitrogen (20.5 mmol/L; normal reference: 3.2–7.1 mmol) and creatinine (1125 $\mu\text{mol/L}$; normal reference: 53–106 $\mu\text{mol/L}$). Blood glucose was 6.4 mmol/L (normal reference: 3.9–6.1 mmol/L), K^+ 4.14 mmol/L (normal reference: 3.5–5.5 mmol/L), Na^+ 145 mmol/L (normal reference: 135–145 mmol/L), Cl^- 108 mmol/L (normal reference: 95–105 mmol/L), and Ca^{++} 2.1 mmol/L (normal reference: 2.25–2.58 mmol/L). Liver function test was normal. Arterial blood gas analysis showed pH 7.40 (normal reference: 7.35–7.45), PaO_2 60.1 mmHg (normal reference: 95–100 mmHg), $PaCO_2$ 44.8 mmHg (normal reference: 35–45 mmHg), and HCO_3^- 29.6 mmol/L (normal reference: 22–27 mmol/L). Chest X ray revealed patchy opacities in the right lower lung. The right diaphragmatic surface and costophrenic angle were also blurred. Furthermore, the cardiac silhouette was enlarged.

Treatment for pulmonary infection with cefoperazone-sulbactam (1.5 g every 12 h) was initiated. The patient also received subcutaneous insulin (30 units/day) and oral nifedipine sustained release tablet (30 mg daily). Peritoneal dialysis continued with 2.5% low calcium peritoneal dialysate.

At day 3 post admission, the patient became disoriented. He was still febrile (40.3°C). SPO_2 declined to 88% despite

of 5 L/min oxygen via a nasal cannula. Blood gas analysis showed pH 7.30, PaO_2 53.10 mmHg, $PaCO_2$ 58.50 mmHg, and HCO_3^- 29.3 mol/L. Blood chemistry revealed that blood urea nitrogen (22.7 mmol/L) and serum creatinine (1345 $\mu\text{mol/L}$) continued to rise, and his plasma glucose also increased (8.4 mmol/L). K^+ , Na^+ , Cl^- , and Ca^{++} were within normal range. His C-Reactive Protein (CRP) was 183 mg/L (normal reference: <8 mg/L), and procalcitonin 66.06 ng/mL (normal reference: <0.5 ng/mL). The liver function, platelet count, aPTT, and PT were normal. Thromboelastogram showed normal coagulation factor reaction time (9.9 min; normal reference: 5–10), fibrinogen reaction time (1.9 min; normal reference: 1–3), α (alpha) angle (64.9°; normal reference: 53°–72°), shear stress coefficient strength (19,233 d/s; normal reference: 4500–11,000 d/s), and hyperfibrinolysis (0.0%; normal reference: <8%) with increased platelet function (79.4 mm; normal reference: 50–70).

The patient was diagnosed with respiratory failure (type II) and pulmoencephalopathy, and transferred to the intensive care unit (ICU) and placed on mechanical ventilation. Imipenem (0.5 g every 6 h) was added, and low-molecular-weight heparin (enoxaparin, 3500–5500 Da, 5–10 IU/kg/h) and insulin were delivered continuously by pumps. Peritoneal dialysis was discontinued and CVVH was undertaken at 32 mL/kg/h for renal insufficiency. The patient became oriented. Arterial blood gases showed normal pH (7.43), improved PaO_2 (88.6 mmHg), normal $PaCO_2$ (36.5 mmHg), and HCO_3^- (23.5 mmol/L). However, fever persisted (39.5°C), and the patient had difficulty weaning off mechanical ventilation.

Abdominal computed tomography (CT) at day 5 showed a hypointense shadow in the left hepatic lobe (61 mm \times 56 mm) with an indistinct border (Figure 1(a)). Liver abscess was confirmed upon ultrasound-guided drainage. The culture

revealed *Klebsiella pneumonia* that was sensitive to third-generation cephalosporin and imipenem. Imipenem (1.0 g every 8 h) and metronidazole (0.5 g every 8 h) were given, and mechanical ventilation and CVVH continued.

At day 12, the patient complained left back pain, which improved the next day without treatment. Platelet count ($243 \times 10^9/L$) was normal, but both aPTT (67.5 s) and PT (17.3 s) were prolonged; his thromboelastogram (heparin cup) was normal. No cutaneous and mucosal bleeding was observed. The patient had no hemoptysis and his fecal occult blood test was negative. Abdominal CT scan at day 16 revealed left iliopsoas swelling, about 61.7 mm \times 64.1 mm, with inhomogeneous densities, and occasional hyperintense opacities and an indistinct border (Figure 1(b)). A diagnosis of iliopsoas hematoma was made. Anticoagulation therapy with low-molecular-weight heparin was discontinued and switched to ex vivo anticoagulation with citrate. Mechanical ventilation and anti-infection therapy (imipenem 1.0 g every 8 h, metronidazole 0.5 g every 8 h) continued. No improvement was seen, and 3 days later, the patient died after his family decided to terminate therapy.

Discussion

Anticoagulation therapy is a risk for bleeding, and patients who receive heparin are recommended to be carefully monitored.⁷ The index patient in this report received low-molecular-weight heparin for anticoagulation therapy because of CVVH. He developed iliopsoas hematoma despite previously normal aPTT and PT, and normal platelet counts. Iliopsoas hematoma is rare during blood purification therapy. Only one case of iliopsoas hematoma was reported in an elderly patient with acute heart failure and anuria who received continuous hemodiafiltration.⁸ Although our patient developed iliopsoas hematoma during hemofiltration, he received anticoagulation therapy with low-molecular-weight heparin, which has been previously associated with retroperitoneal hematoma.^{9–11} Our patient had elevated aPTT and PT following anticoagulation therapy, suggesting that iliopsoas hematoma in our case was due to the use of low-molecular-weight heparin.

CT examination is required for diagnosis of iliopsoas hematoma. Angiographic study can directly identify the culprit vessel and institute arterial embolization therapy.¹² However, angiography may also miss the bleeding vessel.¹³ Because iliopsoas hematoma is not common and atypical in clinical manifestations, missed diagnosis or misdiagnosis often occurs.¹⁴ Left back pain appeared at day 12 of our patient, but CT scan was not promptly conducted, leading to delayed diagnosis.

Treatment of iliopsoas hematoma mainly includes conservative therapy, ultrasound or CT guided percutaneous drainage and decompression, embolization, surgical incision and drainage, and retroperitoneal laparoscopy. Therapeutic modality is chosen according to clinical manifestations and severity of iliopsoas hematoma. Conservative therapy is recommended

for patients with mild bleeding and no apparent nerve compression symptoms, which includes discontinuation of anticoagulation drugs as done in the current case, bed rest, fluid infusion, and blood transfusion. For patients with clinically significant pain and suboptimal conservative therapy, ultrasound or CT guided percutaneous drainage and decompression.^{8,15} Angiography can be considered for those with life-threatening acute massive bleeding; if the bleeding point of a pulsating artery can be identified, arterial embolization is the treatment of choice.¹² For patients with complete paralysis of the femoral nerve due to compression by iliopsoas hematoma following trauma, decompression by surgical incision and drainage is indicated.¹⁶ Retroperitoneal laparoscopic treatment may also be considered.¹⁷

Based on the European and US Guidelines, anti-Xa should be closely monitored in patients receiving low-molecular-weight heparin. However, the current version of the Chinese Guidelines does not include such a recommendation. Also, the test is not available at the authors' institute. We suspect that this is the case in many other developing countries.

In conclusion, anticoagulation with low-molecular-weight was used in the anticoagulation therapy of CVVH. This case highlights the need for careful anticoagulation as well as close monitoring, and particularly the use of anti-Xa to guide the treatment.

Declaration of conflicting interests

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Ethical approval

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Informed consent

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

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