Deep Vein Thrombosis Complicated by Spontaneous Iliopsoas Hematoma in Patient with Septic Shock

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient:	**Male, 73**
Final Diagnosis:	**Iliopsoas hematoma**
Symptoms:	**Altered mental status • lower extremity edema**
Medication:	**—**
Clinical Procedure:	**—**
Specialty:	**Cardiology**

Objective: Rare co-existance of disease or pathology

Background: Spontaneous retroperitoneal hemorrhage (SRH) is a rare and difficult-to-diagnose entity. It is not associated with trauma, pathology, or iatrogenic manipulations. Few cases have been reported, with the only precipitating factor recognized being bleeding diatheses such as anticoagulation states, inherited coagulopathies, and hemodialysis. However, none of these have been described in combination with septic shock, which itself is associated with platelet dysfunction, coagulation dysfunction, and vasculopathy.

Case Report: Our case involves an elderly man presenting with altered mental status of unknown etiology, in addition to hemodynamic instability, presumably due to septic shock, without any overt signs of bleeding. After his initial exam revealed lower-extremity edema and decubitus ulcers, a venous Doppler was performed, which revealed extensive deep vein thrombosis. It was unknown whether the sepsis or DVT occurred first. Therapeutic anticoagulation with heparin was subsequently started. On hospital day 4, a CT abdomen with contrast identified retroperitoneal hematoma after the patient's hemoglobin lowered without any overt signs of bleeding. The diagnosis of spontaneous retroperitoneal hematoma was one of exclusion and posed a therapeutic dilemma (conservative versus invasive management).

Conclusions: Sepsis-related coagulopathy and heparin use in an elderly patient predisposed him to an iliopsoas hematoma. In this case, conservative management with reversal of anticoagulation and blood transfusion was sufficient to stabilize the patient.

MeSH Keywords: Blood Coagulation • Hematoma • Hemorrhage • Shock, Septic

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Background

Hemorrhage in the retroperitoneal space can present as a rare, life-threatening event. It is commonly seen as a complication of femoral artery catheterization or after pelvic and lumbar trauma. Other conditions that can lead to retroperitoneal hemorrhage are ruptured abdominal aortic aneurysm and bleeding from an underlying condition involving the kidneys or adrenal glands. Spontaneous cases, especially with underlying coagulopathies or platelet dysfunction, have been very rarely reported, as presented here [1].

Case Report

A 73-year-old man was brought to the Emergency Department by his son after a 3-day history of altered mental status. The patient had been doing well until a month prior when he started feeling weak and lethargic. On initial examination in the Emergency Department, the patient was agitated, confused, and not oriented to person, place, or time. He was found to be febrile (101.5°F [38.3°C]), tachycardic with a pulse of 115/min, regular rate and rhythm, hypotensive with a blood pressure of 85/60 mm Hg, a respiratory rate of 20/min, and an oxygen saturation of 99% on room air. He had multiple stage 3 to 4 draining ulcers on his left lower extremity and bilateral lower-extremity +2 pitting edema. The rest of his systemic examination was unremarkable, with no tenderness, ecchymosis, or other external signs of trauma.

Initial blood work (Table 1) revealed leukocytosis (13,000/μL) with bandemia (27%), dehydration, an elevated lactate (47 mg/dL), and a urinary tract infection. Urine output was decreased (150 ml in 5 h).

Given the patient's high likelihood of infection, including cellulitis associated with decubitus ulcers and a urinary tract infection, and the patient meeting sepsis criteria, he received a 4-liter bolus of normal saline followed by maintenance at a rate of 250 ml/h, and broad-spectrum IV antibiotics. Despite adequate fluid hydration, the patient remained hypotensive. Vasopressors (norepinephrine) were initiated to help maintain blood pressure. In addition, cardiac markers were normal, EKG revealed normal sinus rhythm with tachycardia, and echocardiography showed an ejection fraction of 55% with mild diastolic dysfunction and a central venous pressure of 8 cmH$_2$O, ruling out cardiogenic shock. He was subsequently transferred to the intensive care unit.

Surgical debridement of the infected decubitus ulcer was performed at bedside. Initial blood and wound cultures obtained at the time of admission, from 2 different sites, grew *Streptococcus agalactiae*. A urine culture grew *Citrobacter amalonaticus*. His WBC count slightly trended down and lactic acid normalized.

Table 1. Initial lab values.

	CBC		Urinalysis
White Blood Cells 10³ per μL	13	Nitrites	Positive
Hemoglobin (g/dL)	12.7	Leukocyte esterase	Positive
Hematocrit (%)	43.8	WBC/hpf	20–30
Platelets 10³ per μL	210	RBC/hpf	10–20
Bands (%)	27	Bacteria	Many
	BMP		**VBG**
Sodium (mEq/dL)	136	pH	7.34
Potassium (mEq/dL)	4.4	pCO$_2$ (mmHg)	37
Chloride (mEq/dL)	99	pO$_2$ (mmHg)	122
Bicarbonate (mEq/dL)	22	HCO$_3$ (mmol/L)	16
BUN (mg/dL)	46		
Creatinine (mg/dL)	1.76		**Other**
Glucose (mg/dL)	203	GFR (mL/min/1.73 m²)	35.46
Calcium	9.1	Lactate (mg/dL)	47

CBC – complete blood count; BMP – basic metabolic panel; BUN – blood urea nitrogen; WBC – white blood cell; RBC – red blood cell; GFR – glomerular filtration rate; VBG – venous blood gas.

Table 2. Changes in coagulation profile and CBC.

Variable	On admission	Day 4 (after hematoma)	Day 5 (after blood transfusions)
Blood cells			
Red blood cells 10^6 per µL	4.74	2.2	3.44
Hemoglobin (g/dL)	12.7	6.1	10.5
Hematocrit (%)	43.8	18.7	31.4
White blood cells 10^3 per µL	13	9	9.8
Platelets 10^3 per µL	210	109	117
Coagulation profile			
PT (sec)	17	18	18
INR	1.4	1.3	1.3
aPTT (sec)	40	140	60
Anti-Xa factor activity	0.25	0.41	0.59

PT – Prothrombin time; aPTT – activated partial thromboplastin time; INR – international normalized ratio.

Doppler studies showed an extensive right lower-extremity occluding thrombus from the popliteal vein proximally through the superficial femoral vein. The patient was started on a weight-based therapeutic dose of unfractionated heparin and adjusted daily according to his coagulation profile. The patient's cell counts, PT, INR, aPTT and anti-Factor Xa activity were measured daily (Table 2). Normal lab values for fibrinogen (220 mg/dL), fibrinogen degradation product (35 mg/dL), and D-dimer (300 ng/mL) on hospital day 1 ruled out DIC.

The patient continues to have altered consciousness, and remained hypotensive, and his lab values remained stable except for a significant drop in hemoglobin value (13.4 g/dL to 6.1 g/dL) from hospital day 3 to day 4 (Table 2). Heparin was discontinued immediately. Coagulopathy was reversed with 2 units of fresh frozen plasma, and 4 units of packed red blood cells were transfused. Thereafter, his hemoglobin remained steady at about 10 g/dL. There were no overt or occult signs of bleeding; fecal occult blood tests were all negative.

CT of the abdomen and pelvis showed a retroperitoneal hematoma with expansion of the left iliopsoas musculature very close to encased portions of the left common and external iliac arteries (Figure 1).

There was no increase in size of the iliopsoas hematoma detected with serial CT scans for the remainder of the hospital course. An inferior vena cava filter was placed to prevent embolization of the clot in his leg after the heparin was discontinued. No angiography or surgical intervention were needed because the patient's hemoglobin remained stable thereafter. The patient died on day 7 of hospitalization secondary to multi-organ failure.

Discussion

Iliopsoas hematomas usually present with severe persistent pain in the lower abdominal quadrants, as well as inguinal or lumbar regions, and radiate to the anterior, medial, or lateral aspects of the lower extremities. This occurs along with variable degrees of weakness and numbness, depending on the branches of the lumbar plexus involved. Classic symptoms of an abdominal mass or ecchymosis of the flank are rarely seen. Few patients present with generalized weakness, syncope, dyspnea, chest pain, headache, and hip or back pain [1,2].

In our case, there were no obvious classic clinical stigmata of retroperitoneal bleeding, except for a sudden drop in hemoglobin. The patient was already in shock due to sepsis, as evidenced by his hypotension, despite adequate hydration, fever, altered mental status, leukocytosis, and positive blood culture. His septic shock, along with altered mental status, likely masked typical presentation, including pain, weakness, or numbness in the absence of obvious or occult signs of bleeding.

Although the differential diagnosis includes an iliopsoas abscess, particularly since the patient initially presented in septic shock, his rapid and significant drop in hemoglobin in the absence of overt signs of bleeding made an abscess less likely. The CT scan was primarily done to look for closed-space bleeding. Furthermore, the bleeding was limited to within the iliopsoas fascia, and the spinal structures were intact. A psoas abscess usually results from tracking of a prevertebral abscess from the spine.

The use of therapeutic unfractionated heparin (with elevated aPTT but therapeutic anti-Xa activity) is a strong determinant
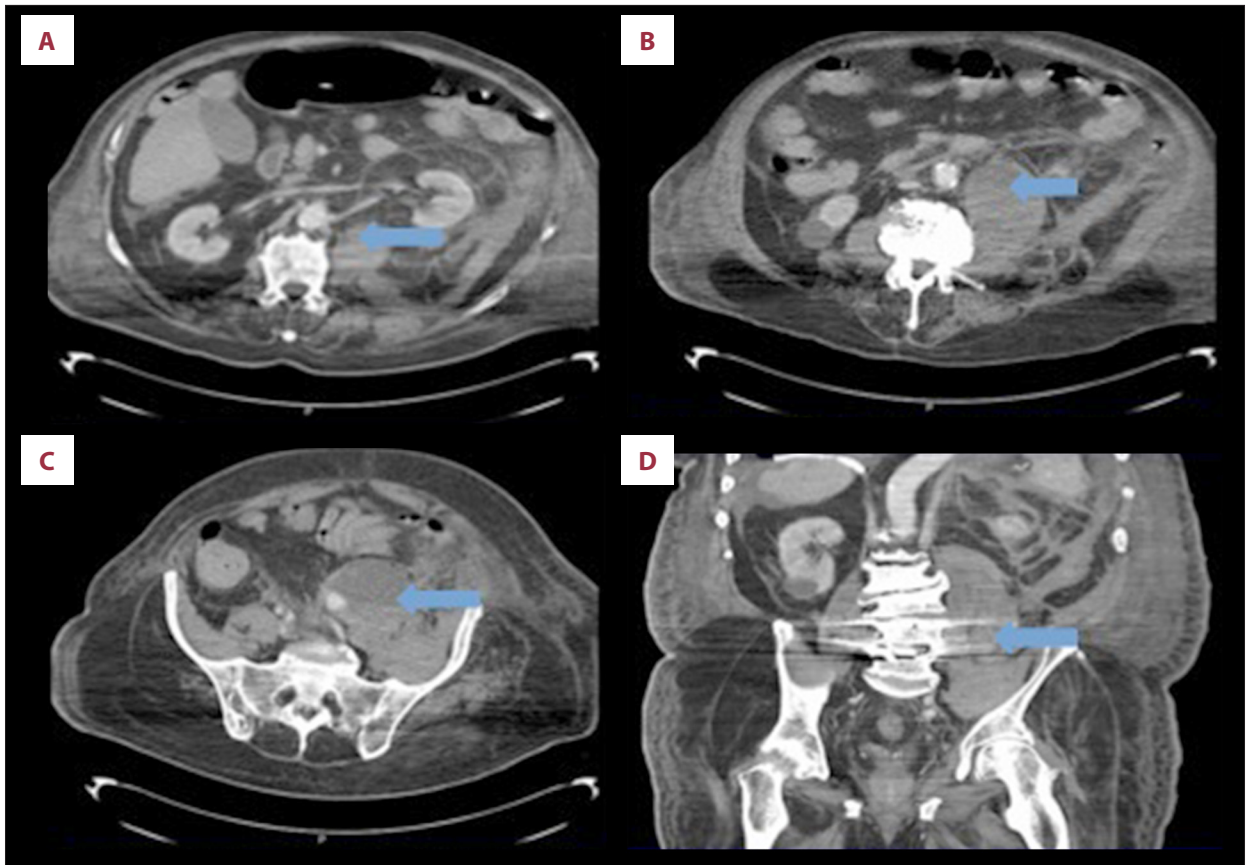
Figure 1. Axial section at levels L4 (**A**), L5 (**B**), S1 (**C**) showing the retroperitoneal hematoma (arrow) confined to the left iliopsoas muscle displacing viscera. The hematoma is very close to the encased portion of the left common and external iliac arteries. No active extravasation is visualized. Coronal section (**D**) of the iliopsoas hematoma confined to the left iliopsoas muscle.

for the occurrence of this retroperitoneal hematoma, as described in previous case reports [3,4]. In addition to other factors (e.g., advanced age), an underlying coagulopathy associated with sepsis, without overt DIC, might have also contributed to the iliopsoas hematoma.

In the absence of overt DIC, there is a well-defined sepsis-associated coagulopathy associated with the same pathological process of inflammatory cytokines, endothelial injury, and bacterial endotoxins, leading to activation of procoagulant pathways [5,6], inhibition of fibrinolysis, and thrombocytopenia [7]. Ironically, the heparin used to treat the coagulopathy can lead to retroperitoneal bleeding.

Another factor associated with an increased risk of bleeding is advanced age, which is associated with a prothrombotic state with increased fibrinolytic activity reflected as elevated D-dimer and plasmin-antiplasmin complexes. Advanced age is also associated with arteriosclerosis, which renders blood vessels friable and prone to rupture [8].

Regardless of the etiology of the retroperitoneal bleed, all high-risk patients should be managed in the ICU. Initial measures include hemodynamic monitoring, discontinuing anticoagulation, normalization of related coagulation factors, fluid resuscitation, and blood transfusions. Early recommendations suggested angiography had limited diagnostic or therapeutic use because hemorrhage was deemed to be related to diffuse small vessels, but this has been challenged by several case reports in which angiograms identified active bleeding sites. Bleeding was then controlled using a combination of agents, such as coils, gelatin, and/or polyvinyl alcohol [9,10]. Panetta et al. reported that if conservative treatment (4 or more units of packed red blood cell transfusions in 24 h, or total of 10 units transfused) fails to maintain hemodynamic stability, then the patient should have urgent endovascular treatment. Embolization is particularly helpful in iatrogenic injuries like femoral catheterization, percutaneous nephrostomy, and renal biopsy rather than spontaneous hematomas[11]. Similarly, endovascular procedures with stent grafting, instead of embolization, have been used successfully in retroperitoneal hematomas associated with inferior vena cava and aortic rupture [12,13]. Surgical procedures that include controlling bleeding and evacuations

are reserved for patients that remain unstable despite adequate fluid and blood resuscitation, if the interventional procedure is unsuccessful or unavailable, or if patient develops compartment syndrome leading to impaired cardiovascular, respiratory, or renal function [1]. In our patient, the abdominal CT scan with contrast showed a large retroperitoneal hematoma on the left, displacing viscera, but the overlying vessels (common and external iliac) were still intact, without extravasation. Thus, the hematoma was likely limited to the iliopsoas muscle, confined by its fascia. Small muscular arteries or veins are a likely source. The patient responded well to initial resuscitation with fresh frozen plasma and packed red blood cells. Angiography or surgery was not performed given the patient's age, stable vitals and hemoglobin, and the fact that the hematoma was confined. The possibility of losing the "tamponade effect" from the confined hematoma and the risks associated with an invasive procedure without a macroscopic vessel injury to repair also suggested a conservative approach.

Conclusions

Spontaneous retroperitoneal hematomas are difficult to diagnose, particularly in patients with altered mental status. Physicians should have a high degree of suspicion in any patient with a sudden drop in hemoglobin and abrupt changes in vital signs. Sepsis or septic shock, with or without overt DIC, in an elderly adult, may precipitate or aggravate unsuspected events. Other known factors, such as use of anticoagulants, antiplatelet agents, and hemodialysis, should raise suspicion.

Conflict of interest

None.

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