

## Case Report

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## Unveiling the Hidden Perils: Spontaneous Iliacus Muscle Hematoma Induced by Rivaroxaban Therapy

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### ABSTRACT

A 56-year-old hypertensive male presented to the Emergency Department with acute-onset dyspnea at rest, persisting for three days. Examination revealed hypoxemia (SpO<sub>2</sub> 88% on room air) with normal chest auscultation. Laboratory investigations showed no significant abnormalities except elevated D-dimer levels. Urgent CT pulmonary angiography confirmed acute thrombosis of segmental branches of the right pulmonary artery with severe pulmonary artery hypertension on 2D echocardiography. Treatment with rivaroxaban was initiated. However, after four days, the patient developed left thigh pain and difficulty with hip flexion. Imaging revealed a left iliacus muscle hematoma, prompting the discontinuation of Rivaroxaban and CT-guided drainage by the interventional radiology team. Symptoms gradually resolved post-drainage. This case highlights the potential complication of anticoagulant therapy-induced hematoma formation, necessitating prompt intervention.

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and prompt intervention to mitigate the risk of adverse outcomes associated with rivaroxaban-induced muscular hematomas.

### Introduction

Rivaroxaban is a direct oral anticoagulant increasingly utilized for the prevention and management of thromboembolic disorders due to its convenience and efficacy. Unlike traditional anticoagulants such as warfarin, rivaroxaban exerts its anticoagulant effects by directly inhibiting factor Xa, thereby interrupting the coagulation cascade [1].

While rivaroxaban is generally considered safer, with fewer dietary and drug interactions compared to warfarin, it is not without its own set of risks. Muscular hematomas, though infrequent, have been reported as a potential adverse effect of rivaroxaban therapy [2-4]. These hematomas can occur spontaneously, particularly in the thigh region, and may lead to significant morbidity if not promptly recognized and managed. Given the increasing use of rivaroxaban in clinical practice, healthcare providers must be aware of this rare but potentially severe complication.

In this case report, we present a unique instance of a spontaneous iliacus muscle hematoma secondary to rivaroxaban therapy, highlighting the importance of considering this diagnosis in patients presenting with thigh pain while on anticoagulant treatment. This case underscores the need for heightened vigilance

### Case Presentation

A 56-year-old hypertensive male presented to the Emergency Department with complaints of acute-onset shortness of breath at rest for three days. He had no history of fever, orthopnea, paroxysmal nocturnal dyspnea, chest pain, or pedal edema.

On admission, vital signs included a temperature of 98.2°F, blood pressure of 140/80 mm Hg, and SpO<sub>2</sub> of 88% on room air. Chest auscultation revealed normal vesicular breath sounds without any additional sounds.

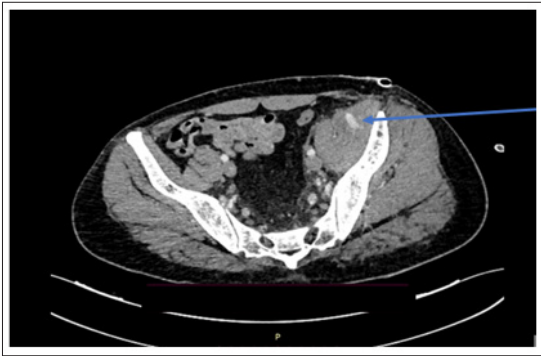
Laboratory investigations (Table 1) showed white blood cells at  $8.8 \times 10^3/\mu\text{L}$ , haemoglobin at 14.3 g/dL, platelet counts at  $168 \times 10^3/\mu\text{L}$ , prothrombin time at 16.2 seconds, the international normalized ratio at 1.4, activated partial thromboplastin time at 38.9 seconds, and D-dimer at 1.85 ng/mL. Urgent CT pulmonary angiography was suggestive of a filling defect seen in the lateral basal and posterodorsal segmental branch of the right pulmonary artery. However, the main pulmonary trunk, right and left main pulmonary arteries, and the rest of their segmental/ sub-segmental branches were normal. 2D echocardiography suggested a global LVEF of 60% with Grade 1 LVDD. RA and RV were dilated. There was severe TR. RVSP was 80 mm Hg, suggestive of severe PAH.

Table 1: Basic Investigations								
Investigations	Normal Range	8/2/24	9/2/24	10/2/24	11/2/24	13/2/24	16/02/24	18/02/24
Haemoglobin	12 - 15 g/dL	14.3	14.3	13.1	13.1	13.9	15.1	13.7
TLC	4 - 11 10 <sup>3</sup> /uL	8830	8830	8510	8510	7820	7860	11500
DLC (N/L/M/E/B)	(40-70/20-40/2-8/1-6)	71/17/10	71/17/10	76/12/08	76/12/08	63/21/12	76/12/9	76/15/10
Platelet Count	150 - 400 10 <sup>3</sup> /uL	168000	168000	170000	170000	190000	247000	262000
Bilirubin (T)	0.3 - 1.2 mg/dL	2.14	1.68	1.68	1.68	1.68	2.34	1.8
Bilirubin (D)	0 - 0.2 mg/dL	0.52	0.43	0.43	0.43	0.43	0.45	0.3
SGPT	0 - 35 U/L	15	28	28	28	28	46	58
SGOT	0 - 35 U/L	31	32	32	32	32	47	42
ALP	30 - 120 U/L	126	97	97	97	97	122	116
GGT	0 - 38 U/L	34	25	25	25	25	42	52
S. total protein	6.6 - 8.3 g/dL	6.9	6.4	6.4	6.4	6.4	6.7	6.3
S. Albumin	3.5 - 5.2 g/d	4	3.7	3.7	3.7	3.7	4.0	3.7
S. Globulin	2.5 - 3.2 g/dL	2.9	2.7	2.7	2.7	2.7	2.7	2.6
B. Urea	17 - 43 mg/dL	32	45	45	46	30	33	31
S. Creatinine	0.55 - 1.02 mg/dL	1.48	1.92	1.92	2.1	1.5	1.4	1.36
Na+	136 - 146 mmol/L	135	138	138	143	139	136	137
K+	3.5 - 5.1 mmo/L	3.7	3.8	3.8	3.3	3.6	4.2	
Cl-	101 - 109 mmo/L	102	103	103	103	103	102	109
Calcium	8.8 - 10.6 mg/dL	9.4	9.1	9.1	8.9	8.7	9.8	9.5
Uric Acid	2.6 - 6 mg/dL	7.3	7.9	7.9	6.3	4.3	4.1	3.2
Phosphorus	2.5 - 4.5 mg/dL	3.7	1.0	1.0	4.4	2.8	1.3	2.2
PT/ INR		16.2/1.4	16.2/1.4	16.2/1.4	15.6/1.38	15.6/1.38	17.4/1.5	23.6/2.14
aPTT		28.6	28.6	28.6	29.5	30.5	38.9	32.5
DRVVT Screen(LA1):	22.1 - 28.1 seconds					129.1 seconds		
DRVVT Confirm(LA2):	31 - 44 seconds					65.3 seconds		
DRVVT Screen(LA1) mixing:	31 - 44 seconds					85.7 seconds		
DRVVT Confirm(LA2) mixing:	30 - 38 seconds					48.7 seconds		
ANTI THROMBIN III	80-120% of control activity					77.2%		
PROTEIN S	60-130% of normal activity					94.7%		
PROTEIN C	70-140% of control activity					70.7%		

D dimer	< 500 ng/mL					1.85 ng/l		
Fibrinogen	200-400 mg/dL.					360.4 mg/dl		
Total cholesterol	< 200 mg/dL					88mg/dl		
Triglycerides	< 150 mg/dL					51mg/dl		
HDL-C	> 40 mg/dL					36mg/dl		
HIV						NR		
HBV						NR		
HCV						NR		
TLC- TOTAL LEUKOCYTE COUNT; DLC-DIFFERENTIAL LEUKOCYTE COUNT; SGPT- SERUM GLUTAMIC PYRUVATE DEHYDROGENASE; SGOT- SERUM GLUTAMIC OXALOACETIC DEHYDROGENASE; PT- PROTHROMBIN TIME; APTT- ACTIVATED PARTIAL THROMBOPLASTIN TIME; DRVVT- DILUTE RUSSEL VIPER VENOM TEST; HDL- HIGH DENSITY LIPOPROTEIN; HIV- HUMAN IMMUNODEFICIENCY VIRUS; HBV- HEPATITIS B VIRUS; HCV- HEPATITIS C VIRUS								

The patient was started on the novel oral anticoagulant rivaroxaban for acute pulmonary thrombosis. Four days later, the patient complained of left thigh pain, cramping, and difficulty with hip flexion. Ultrasound of the left thigh and hip revealed a bulky iliopsoas muscle with heterogeneous hypoechoic echotexture, adjacent stranding, and ill-defined hypoechoic areas. A contrast-enhanced CT scan of the left thigh confirmed a left iliacus muscle hematoma (Figure 1). Consequently, rivaroxaban was discontinued, and the hematoma was drained under CT guidance by the interventional radiology team. The patient’s thigh pain gradually improved over time.

This case emphasizes the need for vigilance in patients on anticoagulant therapy, particularly with novel agents such as rivaroxaban, as spontaneous muscle hematomas, although rare, can lead to significant morbidity if not promptly addressed.



**Figure 1:** Hyperdense Lesion in the Iliacus Muscle- Indicating Haemorrhage (Blue Arrow)

**Discussion**

Direct oral anticoagulants (DOACs), such as rivaroxaban, have significantly improved the management of thromboembolic disorders. They offer a more effective and convenient alternative to traditional vitamin K antagonists like warfarin. Rivaroxaban, a selective factor Xa inhibitor, has shown effectiveness in preventing and treating conditions such as deep vein thrombosis, pulmonary embolism, and non-valvular atrial fibrillation [5]. However, like all anticoagulants, rivaroxaban carries a risk of bleeding complications, which, although generally lower compared to warfarin, remain clinically significant [6].

This case report of a 56-year-old male developing a spontaneous iliacus muscle hematoma while on rivaroxaban underscores the importance of recognizing and managing such adverse events promptly. Rivaroxaban exerts its anticoagulant effect by inhibiting factor Xa, an essential component of the coagulation cascade that converts prothrombin to thrombin. This inhibition disrupts clot formation, providing therapeutic benefits in thromboembolic conditions [7]. However, the anticoagulant effect also predisposes patients to bleeding, which can range from minor to severe. Muscular hematomas, although relatively rare, can occur spontaneously or be precipitated by trauma or invasive procedures. In the iliacus muscle, such hematomas can cause significant morbidity due to their anatomical location and potential to compress neurovascular structures. Several risk factors can predispose patients to bleeding complications while on rivaroxaban. These include advanced age, renal impairment, concomitant use of other anticoagulants or antiplatelet agents, and pre-existing medical conditions such as hypertension. In this case, the patient had hypertension, a well-known risk factor for bleeding. The absence of trauma or invasive procedures prior to the hematoma suggests a spontaneous etiology, likely exacerbated by the anticoagulant therapy. The patient, in this case, presented with acute onset dyspnea, leading to the diagnosis of acute pulmonary embolism. Rivaroxaban was appropriately initiated, given its established role in managing this condition. The subsequent development of left thigh pain and difficulty with hip flexion raised concerns for a possible bleeding complication.

The clinical presentation of iliacus muscle hematoma typically includes acute pain in the groin, hip, or thigh, often accompanied by femoral neuropathy due to compression of the femoral nerve. Diagnosis of iliacus muscle hematoma is primarily based on imaging studies. Ultrasound may initially suggest muscle enlargement and heterogenous echotexture, but computed tomography (CT) or magnetic resonance imaging (MRI) provides more definitive characterization. In this case, the contrast-enhanced CT scan confirmed the diagnosis of a left iliacus muscle hematoma. Prompt imaging is crucial for early diagnosis and management, preventing complications such as nerve compression or compartment syndrome. Management of rivaroxaban-induced bleeding complications involves discontinuation of the anticoagulant and supportive measures. In this patient, rivaroxaban was held immediately upon diagnosis of the hematoma. In a case series by Cinar et al., 4 cases of intramuscular hematoma were reported secondary to warfarin use [8]. Similarly, Ardebol et

al. reported an intramuscular hematoma in the sartorius muscle following the use of rivaroxaban. The role of specific reversal agents for DOACs, such as Andexanet alfa, is limited to life-threatening or uncontrolled bleeding and was not indicated in this case [9]. Interventional radiology played a pivotal role in the management of this patient. CT-guided drainage of the hematoma provided symptomatic relief and prevented further complications. This minimally invasive approach is preferred over surgical intervention due to its lower risk profile and effectiveness in evacuating the hematoma. Post-procedure monitoring is essential to ensure the resolution of symptoms and to manage any potential recurrence of bleeding.

This case highlights several important considerations for clinicians managing patients on rivaroxaban. Firstly, while DOACs are generally associated with a lower risk of major bleeding compared to warfarin, vigilance for bleeding complications remains essential. Healthcare providers should maintain a high index of suspicion for spontaneous hematomas in patients presenting with unexplained pain or neurological deficits, particularly in those with risk factors such as hypertension or renal impairment. Secondly, the choice of anticoagulant therapy should be individualized based on the patient's overall risk profile. While rivaroxaban offers several advantages, including fixed dosing and fewer dietary interactions, its bleeding risk cannot be overlooked. Regular monitoring of renal function and patient education on recognizing signs of bleeding are critical components of safe anticoagulant therapy. Lastly, the management of rivaroxaban-induced bleeding complications requires a multidisciplinary approach. Collaboration between emergency physicians, haematologists, radiologists, and, when necessary, interventional radiologists ensures timely diagnosis and effective treatment. This case underscores the value of such teamwork in achieving favourable outcomes.

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