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CASE REPORT

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Mediastinal hematoma following thrombolysis for acute pulmonary thromboembolism

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Abstract

Massive pulmonary thromboembolism (PE) is a cardiorespiratory emergency and can be fatal if left untreated. The recommended treatment for PE in the presence of right ventricular dysfunction and hemodynamic instability is thrombolysis. However, the latter is a double-edged sword as life-threatening bleeding manifestations can occur post-thrombolysis. Timely identification and management of these complications can prevent a catastrophic outcome. We report a case of mediastinal hematoma with new onset hemodynamic compromise following thrombolysis for acute massive pulmonary embolism. Clinico-radiological features and Point of Care Ultrasound (POCUS) findings helped in the identification of the bleeding site in our case. Despite early diagnosis and timely intervention, the patient succumbed to secondary complications.

KEYWORDS
mediastinal hematoma, pulmonary embolism, thrombolysis

INTRODUCTION

Massive pulmonary thromboembolism is a cardiovascular disorder that warrants immediate diagnosis and management. Risk stratification is a key factor in the management of PE. In high-risk PE systemic thrombolytic therapy is the mainstay of treatment and is shown to reduce PE-related mortality.[1] In patients with intermediate-risk and low-risk PE, anticoagulation is the treatment of choice with careful monitoring for features of hemodynamic compromise.[1,2] A decision for systemic thrombolytic therapy must be made with utmost caution after weighing the risks and benefits. Mediastinal hematoma secondary to thrombolysis is very rare and is associated with very high mortality.[3,4]

CASE REPORT

A 70-year-old gentleman with diabetes mellitus and systemic hypertension, presented to the outpatient department with a history of cough for 10 days and acute onset dyspnoea for 3 days. He had noticed swelling in the left lower limb for the last 3 months. There was no history of fever, chest pain or haemoptysis.

On examination, he had tachycardia (heart rate—112/min), tachypnoea (respiratory rate—30/min) with normal blood pressure (132/70 mm Hg). He was hypoxic with a saturation of 84% on room air. Examination of other systems was unremarkable. In view of left lower limb swelling and acute onset dyspnoea, a clinical diagnosis of deep vein thrombosis with pulmonary thromboembolism was considered.

Laboratory investigations revealed a normal hemogram and renal function parameters, and elevated levels of d-dimer (5273 ng/mL), troponin I (0.071 mcg/L) and nT-pro BNP (3420 ng/L). Arterial blood gas analysis showed respiratory alkalosis (pH—7.48, PCO_2—20 mm Hg, PaO_2—50 mm Hg, HCO_3—25 mmol/L).

Electrocardiogram showed a right bundle branch block with features of right ventricular (RV) strain. Chest radiograph showed an enlarged right descending pulmonary artery (Figure 1A). A computed tomography pulmonary angiogram (CTPA) was done which showed a saddle embolus extending into the distal right main pulmonary artery and its lobar and segmental branches (Figure 2A, B). Simplified Pulmonary Embolism Severity Index (sPESI) was

3, transthoracic echocardiography (TTE) showed a dilated, 'D' shaped RV and during the course of evaluation, his blood pressure dropped to 90/60 mm Hg.

In view of the hemodynamic instability, 'high-risk' stratification, features of RV dysfunction and raised cardiac biomarkers, a decision to administer systemic thrombolysis was made. A detailed pre-thrombolysis checklist was administered and after excluding contraindications, thrombolysis was done with Reteplase (recombinant tissue plasminogen activator, tPA) given in two intravenous bolus doses of 10 units each, 30 mins apart.

Post-thrombolysis, blood pressure improved and he remained hemodynamically stable on oxygen supplementation at 2 L/min for the next 48 h. Two days after thrombolysis, he developed central chest pain, worsening dyspnoea and hypoxia requiring high-flow oxygen supplementation (12 L/min through non-rebreathing mask). He also developed hypotension requiring vasopressor support. A repeat chest radiograph

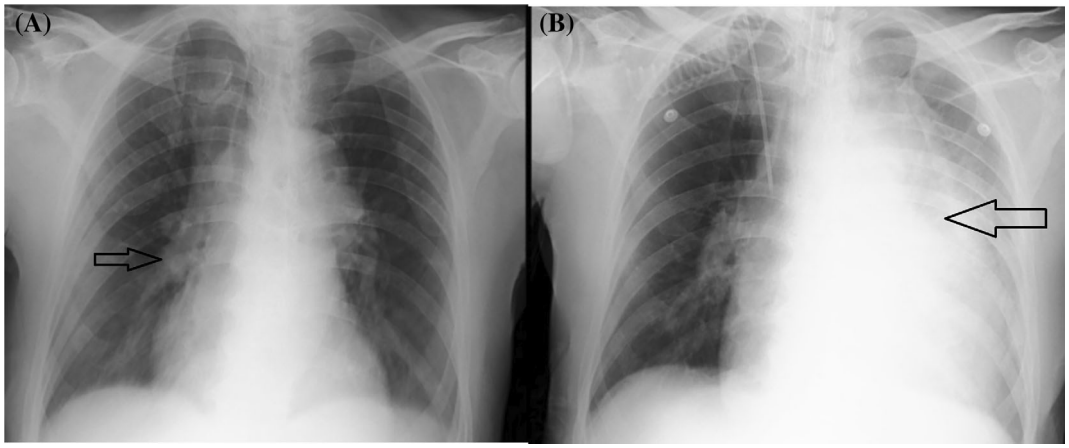

FIGURE 1 (A) Chest radiograph at admission showing enlarged right descending pulmonary artery; (B) Chest radiograph 48 h after thrombolysis showing in homogenous opacity in the left perihilar region with mediastinal widening.

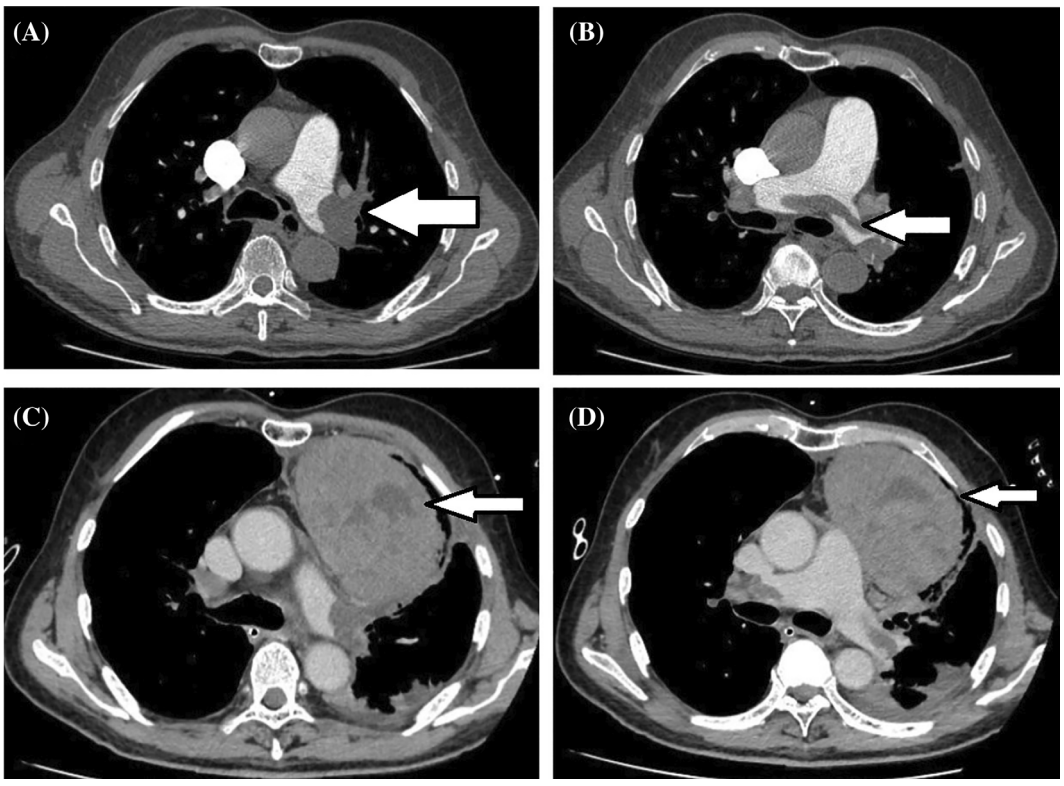

FIGURE 2 (A, B) CT Pulmonary Angiogram showing a saddle embolus extending into the left main pulmonary artery; (C, D) CT angiogram post-thrombolysis showing encapsulated anterior mediastinal hematoma compressing the mediastinal structures.

showed an inhomogeneous opacity in the left perihilar region with mediastinal widening (Figure 1B). Blood investigations revealed a fall in haemoglobin from 15.4 to 11.9 g/dL. Point of care ultrasound (POCUS) showed no evidence of pleural or pericardial effusion. However, an ill-defined soft tissue was seen in the second and third intercostal space causing right ventricular outflow tract obstruction (RVOTO) and right ventricular free wall compression. In order to delineate the lesion better, a CT angiogram was done which showed an anterior mediastinal hematoma (9.1 × 8.7 × 13.9 cm) abutting the pericardium, causing compression and passive collapse of the adjacent lung parenchyma (Figure 2C, D). The patient was intubated due to worsening hypoxemia and initiated on mechanical ventilation.

In view of RVOTO and worsening hypotension with increasing vasopressor support, an emergency thoracotomy was performed. A large encapsulated hematoma was noted in the anterior mediastinum extending into the left pleural cavity measuring approximately 500 mL. After the evacuation of the hematoma, the patient improved hemodynamically and requirement of vasopressors decreased. Repeat Transthoracic echocardiography showed no signs of RVOTO or RV dysfunction.

Mechanical ventilatory support was continued and intercostal drains placed following surgery showed no evidence of active bleeding and were removed subsequently over the next 2 days. However, the patient developed sepsis secondary to ventilator-associated pneumonia (VAP), refractory shock, renal failure requiring renal replacement therapy and succumbed to his illness.

DISCUSSION

Acute bleeding manifestations following thrombolysis are not uncommon. Post-thrombolysis, major bleeding has been reported in 9.9% and fatal or intracranial haemorrhage in 1.7%.[5] Any fatal bleeding, symptomatic bleed in a critical area (intracranial, intraspinal, intraocular, retroperitoneal or pericardial) or a bleed resulting in a fall in haemoglobin of ≥2 g/dL requiring transfusion of 2 or more units of blood is considered as a major bleed.[6]

Mediastinal hematoma are usually reported secondary to trauma, aortic dissection, misplaced central venous catheters, and some occur spontaneously.[7–10] The common sources of bleeding identified in these cases include the azygos and hemiazygos veins, vena cava and small venous tributaries of the internal mammary, brachiocephalic and inferior thyroidal veins.[7] The source of bleed may remain unrecognized despite extensive evaluation, imaging and thoracotomy in many cases.[10] On review of the literature, there is only one case report till date of a mediastinal hematoma following thrombolysis for pulmonary thromboembolism.[4]

In our patient, no active bleeder or source of bleed could be identified at thoracotomy. Thus, we presume that this self-contained bleed could have been venous in origin.

Being a potential space owing to the presence of hollow viscus organs, a mediastinal collection may go unnoticed in the absence of significant compressive symptoms.[7] Chest pain and shortness of breath are the most common symptoms reported due to mediastinal hematoma. Tachycardia and hypotension are the frequently observed signs in these patients.[7,10,11] These signs and symptoms being non-specific, presence of mediastinal compression symptoms and mediastinal widening on chest radiograph in the right clinical context should raise the suspicion of a mediastinal hematoma and prompt further evaluation. The clinical utility of POCUS in identifying the cause of hemodynamic compromise in such cases needs to be reemphasized.[4] Bedside echocardiography helped us in identifying RVOTO by the hematoma as the cause of hypotension in our case. CT angiogram plays a pivotal role in diagnosing and identifying the source of bleed in cases with a suspected mediastinal hematoma. In some cases, further evaluation may be needed including trans-oesophageal echocardiography to identify the source of bleed like innominate artery, left common carotid artery, intercostal arteries and left subclavian artery.[7]

Mediastinal hematomas can be managed conservatively in the absence of compression causing hemodynamic compromise.[10] Surgical intervention may be essential in the presence of arterial bleed and hemodynamic compromise.[7] Our patient had significant hemodynamic compromise due to compression on the right ventricular outflow tract and hence an emergency surgery was performed. Angiography along with embolization is another therapeutic option in cases where the bleeding vessel is identified and amenable to cannulation.[10] Despite maximal efforts, mediastinal hematomas are associated with a high mortality specially in those occurring post-thrombolysis.[3,4]

AUTHOR CONTRIBUTION STATEMENT

Planning: Kavitha Venkatnarayan and Chitra Veluthat. *Conduct*: Uma Maheswari Krishnaswamy, Chitra Veluthat and Kavitha Venkatnarayan. *Reporting*: Chitra Veluthat, Kavitha Venkatnarayan and Uma Maheswari Krishnaswamy. *Conception and design*: Chitra Veluthat and Kavitha Venkatnarayan. *Acquisition of data*: Chitra Veluthat. *Editing and revision*: Kavitha Venkatnarayan, Uma Maheswari Krishnaswamy, Uma Devaraj and Priya Ramachandran.

CONFLICT OF INTEREST STATEMENT

Uma Maheswari Krishnaswamy is an Editorial Board member of Respirology Case Reports and a co-author of this article. They were excluded from all editorial decision-making related to the acceptance of this article for publication.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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