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Case Report
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To Give or Not to Give: Thrombolysis for Massive Pulmonary Embolism in a Patient with Profound Anaemia

Corinne Yee Lyn Lau[1], Young Koh[1], Sameera Ganti[2], Derek Jiun Yi Heng[3], Eunizar Omar[2]

[1]Sengkang General Hospital, Clinic of Emergency Medicine, Singapore
[2]University of Malaya Medical Centre, Clinic of Emergency Medicine, Kuala Lumpur, Malaysia
[3]Woodland Health Campus, Clinic of Emergency Medicine, Singapore

Abstract

Massive pulmonary embolism (PE) is uncommon but potentially fatal, requiring immediate thrombolytic therapy. Thrombolysis, however, carries a significant bleeding risk and is generally contraindicated in cases of active bleeding, which is an important differential to consider in patients with severe anaemia. Physicians must balance possible bleeding against the risk of rapid clinical deterioration. We report a case of autoimmune haemolytic anaemia presenting with massive PE and profound anaemia successfully treated with thrombolysis.

Keywords: Emergency, critical care, respiratory, pulmonary embolism, autoimmune haemolytic anaemia, thrombolysis

Introduction

Severe dyspnea is a common presenting complaint to the emergency department (ED), with the most common causes being lower respiratory tract infection, heart failure and chronic airway disease (1). Massive pulmonary embolism (PE) and anaemia are less common causes of dyspnea, and their co-occurrence complicates diagnosis and management.

Autoimmune haemolytic anaemia (AIHA) is a rare disease where antibodies rapidly destroy red blood cells. It is usually acquired and affects 1 to 3 cases per 100,000 persons per year. AIHA can occur at any age, but its incidence increases with age (2). It has been described as an under-recognized cause of venous thromboembolism (VTE). Various mechanical and chemical processes have been proposed to explain how AIHA causes a pro-thrombotic state (3). Acute PE has been recognized as early as 1967 to be the leading cause of death in AIHA patients (4).

Emergency thrombolytic treatment in massive PE can be life-saving. However, one of the absolute contraindications for thrombolytic therapy is active bleeding, which is a differential to consider in a hypotensive patient with severe anaemia. That said, anaemia alone is neither an absolute nor relative contraindication to thrombolysis (5).

We describe a case of massive PE and profound anaemia secondary to AIHA and the considerations for thrombolytic therapy in such patients.

Case Report

A 78-year-old woman presented with one week of worsening dyspnea, pallor, and lethargy, resulting in functional decline. She denied chest pain, haemoptysis, melena, or limb swelling and had no known comorbidities or risk factors for VTE.

On arrival, her temperature was 36.5 °C, blood pressure 90/50 mmHg, heart rate 108 bpm, respiratory rate 17 breaths/min, and oxygen saturation 83% (room air). She was pale and icteric but without signs of active bleeding. Cardiopulmonary and abdominal examinations were unremarkable, and rectal examination revealed no blood.

Corresponding Author: Young Koh MD, Sengkang General Hospital, Clinic of Emergency Medicine, Singapore
E-mail: kohyoung6@gmail.com **ORCID ID:** orcid.org/0009-0000-6989-4135
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Electrocardiography showed sinus tachycardia. Venous blood gas revealed pH 7.38, bicarbonate 14 mmol/L, potassium 4.1 mmol/L, and lactate 10.7 mmol/L. Haemoglobin was too low to register on the point-of-care analyzer. Point-of-care ultrasound (POCUS) demonstrated a dilated right ventricle and right atrium with a D-shaped left ventricle, suggestive of right heart strain. There was no abdominal free fluid or aortic aneurysm.

Laboratory tests confirmed severe haemolytic anaemia: haemoglobin 4.5 g/dL, haematocrit 14.8%, mean corpuscular volume 131 fL, reticulocytes 13%. Bilirubin was raised (total 64 µmol/L, direct 25 µmol/L), while liver enzymes and electrolytes were normal. Troponin-T (40 ng/L) and NT-proBNP (662 pg/mL) were elevated. A chest radiograph was normal.

Computed tomography pulmonary angiography (CTPA), performed within two hours of arrival, demonstrated extensive bilateral PE with right ventricular strain. Soon after, the patient developed worsening hypotension despite fluid resuscitation. A central venous line was inserted, and intravenous noradrenaline was commenced. Packed red blood cell transfusion was started while multidisciplinary discussion ensued between the ED, intensive care, and haematology teams.

Despite resuscitative efforts, she remained in obstructive shock. Given the haemodynamic instability, the decision was made to administer systemic thrombolysis with 100 mg recombinant tPA infused over two hours. During thrombolysis, the patient received close monitoring for haemodynamics and bleeding. Blood pressure improved within an hour, allowing vasopressor reduction. Intravenous hydrocortisone (100 mg) was administered empirically.

The direct antiglobulin test later confirmed warm AIHA (IgG and C3d positive). She was treated with high-dose corticosteroids and received several transfusions. Her haemoglobin gradually stabilized, and she was discharged after seven days on tapering oral prednisolone and anticoagulation. Follow-up CTPA at six weeks showed complete resolution of emboli and normalization of right heart size.

The patient mentioned in this report died of a myocardial infarction a few months after the index visit and had no next-of-kin available for contact. As per our institutional guidelines, which are in line with COPE guidelines for case reports on deceased and anonymized patients, consent is waived in such instances.

Discussion

This case highlights critical concepts when managing an acutely breathless and hypotensive patient in the ED. While anaemia is a known cause of breathlessness, it rarely presents with hypotension in the absence of clinically evident ongoing bleeding. Such a presentation should direct the clinician to look for other potential sources of blood loss and causes of shock. The assessment of undifferentiated shock and dyspnea is supplemented by the POCUS RUSH protocol, which is now the international standard of care (6). Since thrombolysis was indicated in the context of profound anaemia, a multi-disciplinary team approach was instituted early.

AIHA increases the risk of PE by 2.6 times as compared to those without the condition (7). Retrospective data suggest that 10-25% of AIHA patients develop PE, though many cases remain unrecognized. Risk appears heightened in the context of intravascular haemolysis and transfusion requirements. Notably, mixed-type AIHA (warm + cold autoantibodies) has been associated with deep vein thrombosis/PE in at least one reported case. Similarly, cold agglutinin disease, particularly when triggered by infections or lymphoproliferative disorders, may predispose to thrombotic complications. There are also emerging reports of AIHA complicated by PE following coronavirus disease 2019, including cases involving cold agglutinin disease (8-10). Despite various hypotheses, the exact mechanism relating AIHA and a pro-thrombotic state remains unclear. The most widely accepted hypothesis suggests that the destruction of red blood cell membrane organization by autoantibodies leads to loss of membrane phospholipid asymmetry and increased expression of anionic phospholipids, especially phosphatidylserine, which in turn results in a pro-thrombotic state (11). The management of AIHA is challenging as extensive workup is needed to diagnose the exact type, with varying treatment strategies for each. There are four known types of AIHA: warm antibody AIHA, cold antibody AIHA, paroxysmal cold hemoglobinuria and Coombs negative AIHA (12). Depending on the type, steroids, rituximab, immunosuppressive drugs (e.g., cyclophosphamide) and splenectomy may be considered. Treatment of the cause is the mainstay in cases of secondary and drug-induced AIHA (13).

Workup for AIHA takes time, and the management priorities for AIHA-PE need to be focused on rapid diagnosis, stabilization, and treatment of PE. As described by the ESC guidelines, massive PE is defined by the presence of hemodynamic instability. The ESC guidelines recommend use of rtPA in cases of massive PE, given as an infusion of 100 mg over 2 hours. The guidelines also suggest cautious volume loading and early use of norepinephrine in cases of acute right heart strain or failure (14).

This patient fulfilled the obstructive shock criteria and was deemed to have a massive PE. She was given a cautious fluid bolus of 500 mL of Lactated Ringer's and norepinephrine was started early. The dilemma remained with regard to rtPA administration

in view of the severe anaemia. Massive PE is a time-critical diagnosis, but the diagnosis of AIHA was not apparent at the ED. The patient remained undifferentiated, and ongoing bleeding was a concern. To the authors' knowledge, there are no guidelines available regarding use of rtPA for PE in the context of severe anaemia, and only case reports exist (Table 1). The only related guideline, as of 2020, is by the American Society of Haematology, which recommends use of rtPA in ischemic stroke secondary to sickle cell disease (15).

In summary, i) anyone presenting with undifferentiated shock and dyspnea should have emergency assessment with POCUS as a supplement to the usual history taking and physical examination, ii) clinicians should consider the cause of anaemia after excluding ongoing massive bleeding, iii) anaemia itself is not a contraindication to the use of systemic thrombolysis in the context of a massive PE, and iv) a multidisciplinary approach is essential for patient care and safety.

Conclusion

AIHA is an uncommon but important cause of profound anaemia and a risk factor for thromboembolic disease. Its coexistence with massive PE presents a diagnostic and therapeutic challenge. This case demonstrates that in the absence of active bleeding, thrombolysis can be safely administered despite profound anaemia when PE is immediately life-threatening.

For emergency physicians, key takeaways include the role of POCUS in rapid diagnosis, the importance of early multidisciplinary consultation, and the need to view anaemia as a relative not absolute barrier to thrombolytic therapy in massive PE.

This is one of few documented cases of successful thrombolysis for massive PE in an unrecognized AIHA patient with haemoglobin as low as 4.5 g/dL. It illustrates that timely, individualized decisions

Table 1. Reported cases of autoimmune hemolytic anaemia complicated by pulmonary embolism

Author/year	Age/sex	AIHA type	HGB at presentation	Suspected trigger	PE severity	Thrombolytic agent	Outcome
Xu et al. (2), 2020	59F	Warm AIHA	9.2 g/dL	Not mentioned	Massive PE, cardiac arrest	IV alteplase (50 mg)	Survived; recovery on 3-month follow-up
Randhawa et al. (8), 2019	66F	Warm AIHA	7.2 g/dL	Triple therapy for *H pylori*	Submassive PE	rtPA (100 mg)	Died
Imataki et al. (9), 2020	66M	Mixed-type AIHA	4.1 g/dL	Not mentioned	Fulminant/massive PE	Not mentioned	Died
Solari et al. (16), 2021	75M	Warm AIHA	8.3 g/dL	Idiopathic	Segmental (right laterobasal) and bilateral subsegmental PE	Anticoagulation with rivaroxaban	Survived
Lee et al. (17), 2016	56F	Mixed-type AIHA	2.8 g/dL	Not mentioned	Bilateral subsegmental PE	Heparin & warfarin	Survived
Shiroshita et al. (18), 2023	60F	Warm AIHA (post-steroids)	5.8 g/dL	After starting methylprednisolone	Left subsegmental PE	Apixaban	Survived
Patil et al. (10), 2022	51F	Cold agglutinin disease AIHA	5.1 g/dL	COVID-19 infection	Bilateral lower lobe segmental PE	Heparin	Survived
Present case (2024)	78F	Warm AIHA (IgG + C3d)	4.5 g/dL	Idiopathic	Massive PE with obstructive shock	rtPA (100 mg over 2 h) + steroids + transfusion	Survived; complete PE resolution

Table 1 these cases demonstrate a spectrum of severity in presentation for PE patients with AIHA, with the present case being one of two reported cases of massive PE anaemia treated successfully with full-dose thrombolysis
AIHA: Autoimmune hemolytic anemia, HGB: Hemoglobin, PE: Pulmonary embolism, COVID-19: Coronavirus disease-2019

grounded in pathophysiologic reasoning and team collaboration can be lifesaving even in the most complex scenarios.

Ethics

Informed Consent: The patient mentioned in this report died of a myocardial infarction a few months after the index visit and had no contactable next-of-kin. As per our institutional guidelines, consent is waived in such instances.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.J.Y.H., Design: S.G., Analysis or Interpretation: Y.K., Literature Search: C.L.Y.L., S.G., E.O., Writing: C.L.Y.L., Y.K., S.G., D.J.Y.H.

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