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Case Report

Therapeutic Challenge in the Face of a Thoracic Catastrophe: Association of Pulmonary Embolism and Stanford Type A Aortic Dissection Extended to the Coronary Artery - A Case Report

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Abstract

Introduction: The combination of pulmonary embolism (PE) and Stanford type A aortic dissection (AAD) is a rare and complex cardiovascular emergency. The therapeutic management of this combination represents a major challenge.

Case report: A 49-year-old hypertensive male smoker presented with sudden migrating chest pain, dyspnoea and haemoptysis. Examination revealed asymmetric brachial pulses. The ECG showed extensive anterior subepicardial ischaemia. Echocardiography revealed septo-apical hypokinesia and pericardial effusion. Angioscan confirmed an AAD with coronary extension and distal left PE. Biological tests showed elevated troponin and D-dimer levels. Conservative medical treatment was instituted with beta-blockers and antihypertensives, with an immediate favourable outcome. Although surgery was indicated, it could not be performed due to a lack of technical facilities. Follow-up at one month was favourable.

Conclusion: This case illustrates the possibility of a rare association between PE and AAD extending to the coronary arteries. Although the optimal treatment is surgery, medical management may allow initial stabilisation in the absence of immediate surgical possibility. However, the prognosis remains guarded, requiring prolonged close monitoring.

INTRODUCTION

Thoracic emergencies represent a major challenge in medicine, due to the diversity and severity of the pathologies that can cause them [1]. Among these pathologies, pulmonary embolism (PE), acute coronary syndrome (ACS) and Stanford type A aortic dissection (AAD) are dramatic events that require rapid and coordinated management [1,2]. When these three conditions occur together, the pathophysiological mechanisms involved exacerbate circulatory disorders, making the prognosis particularly poor and treatment all the more difficult [3]. Although these unusual situations represent a real therapeutic challenge, there are as yet no precise recommendations [4]. The most commonly used management protocols are still based on expert consensus [5]. The present case illustrates this rare association, detailing the diagnostic and therapeutic approach used to try to save a patient facing these serious and life-threatening pathologies.

CLINICAL OBSERVATION

History of the disease

The patient was 49 years old, with cardiovascular risk factors including arterial hypertension which had been irregularly monitored for about seven years, a sedentary lifestyle and 7 pack-years of smoking. He presented with sudden onset mid-thoracic pain, migrating and descending

to the lumbar region, intensity 8/10 on the visual analgesic scale. This pain was associated with exertional dyspnoea and haemoptysis.

Physical examination

The physical examination revealed a blood pressure of 160/100 in the left arm, compared with 135/82 mmHg in the right arm; a heart rate of 84 beats per minute; and asymmetric pulses in both thoracic limbs (110 on the left, 98 on the right).

Further investigations

The emergency electrocardiogram (ECG) showed regular sinus rhythm at 83 cycles per minute, extensive anterior subepicardial ischaemia with an antero-septo-apical subepicardial lesion (Figure 1). Doppler echocardiography showed hypertrophy of the left ventricular walls, septo-apical hypokinesia, good systolic function of both ventricles and a circumferential pericardial effusion of small and large volume opposite the right atrium with no sign of collapse (Figure 2). The angioscanner showed an intimal flap in the ascending aorta separating two channels (true & false) and an endoluminal defect in the basal posterior branch, suggesting a Stanford type A aortic dissection and a distal left pulmonary embolism (Figure 3). Troponin was elevated to five times normal and D-dimer to 10 times normal. The rest of the laboratory work-up was normal.

Diagnosis

We accepted the diagnosis of distal pulmonary embolism associated with Stanford stage A aortic dissection extending to the coronary arteries.

Treatment and evolution

Treatment was mainly medicinal, with Atenolol 50mg (1/2 tablet morning and evening); lactulose sachet (10 spoonfuls 3 times a day); sodium picosulphate drop (10 drops/day); Nicardipine 50mg (1 tablet morning and evening). Compression stockings were also worn. This treatment resulted in an immediate favourable outcome,

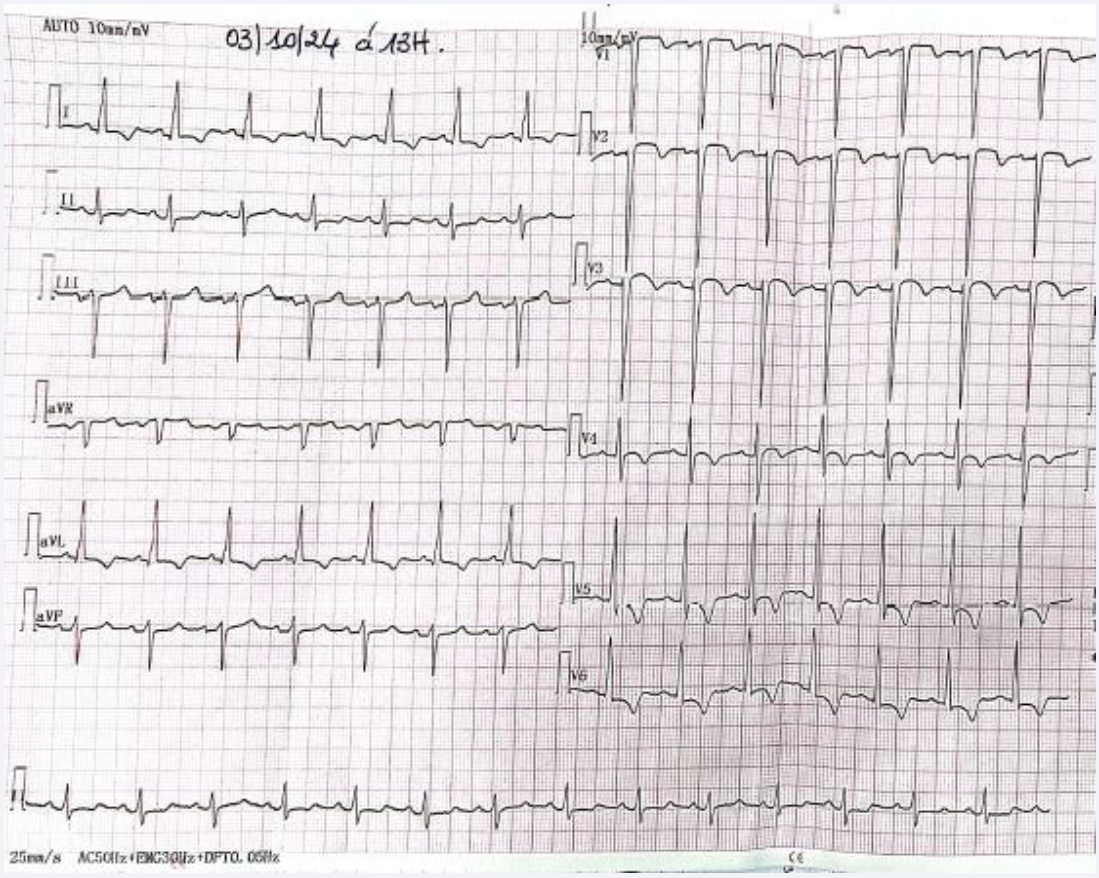

Figure 1 12-lead surface electrocardiogram showing extensive anterior subepicardial ischaemia with an anterosepto-apical subepicardial lesion.

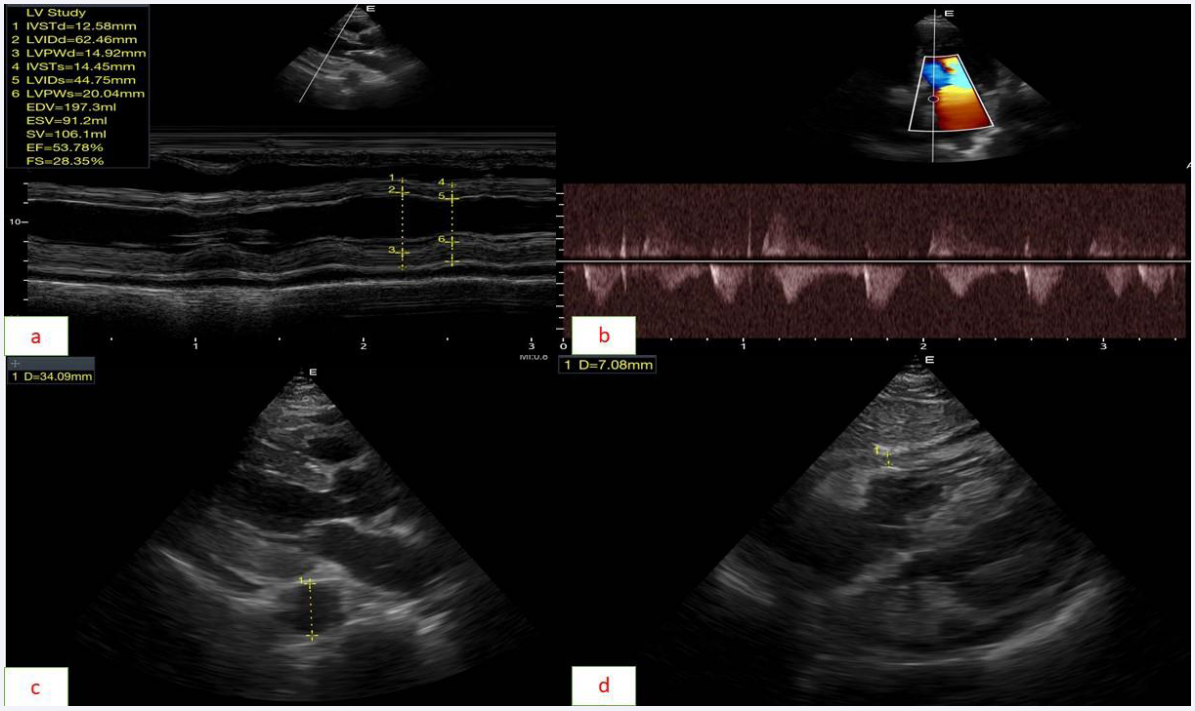

Figure 2 Transthoracic Doppler echocardiogram

a: left paraspinal long axis section showing hypertrophy of the cardiac walls with good left ventricular function

b: Apical 5-chamber section coupled to continuous and colour Doppler showing aortic insufficiency flow

c & d: Left para-sternal long axis and sub-costal 4 cavity sections showing circumferential pericardial effusion

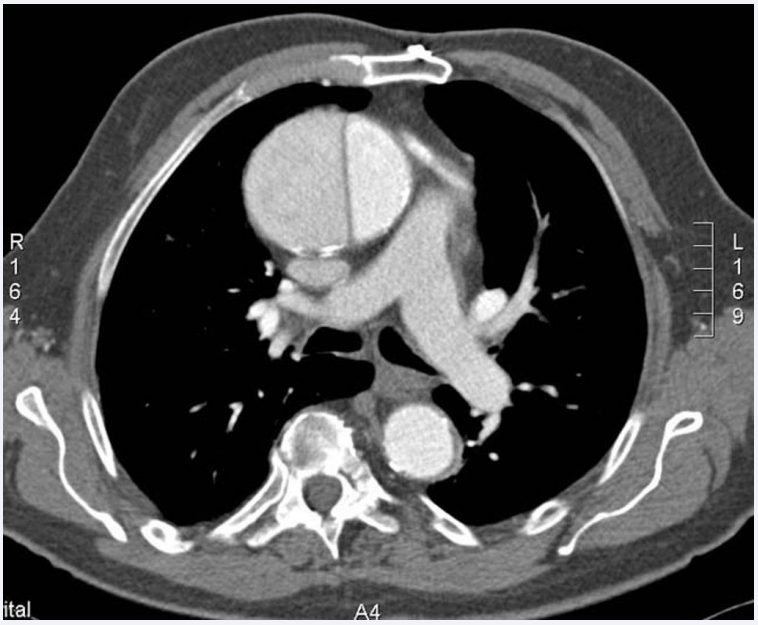

Figure 3 Thoracic angioscan showing an intimal flap in the ascending aorta separating two channels (true & false) suggestive of a Stanford type A aortic dissection and distal left embolism.

with blood pressure of 115/75 mmHg at the twelfth hour of admission. An indication for surgery was given, although it was not performed due to a lack of technical facilities in our setting. The patient was discharged home after 3 weeks and a clinico-biological check-up at one month was unremarkable. The patient was seen again one month after hospitalisation and the clinical examination revealed a 2/6 aortic insufficiency murmur with no other abnormalities.

DISCUSSION

The combination of PE and coronary artery disease is a very rare and serious condition, requiring urgent treatment [4]. This combination represents a major challenge because of the fundamental differences in the pathophysiology and therapeutic management of these two pathologies. PE, which results from obstruction of the pulmonary artery by a clot originating in the deep veins of the lower limbs, affects approximately 1 to 2 people per 1,000 inhabitants per year [6]. AAD, on the other hand, is much rarer, with an estimated incidence of 2 to 3 cases per 100,000 population per year [7]. This type of dissection mainly affects individuals aged between 50 and 70, often in association with cardiovascular risk factors such as arterial hypertension, degenerative connective tissue disease or smoking-induced atherosclerosis [7].

Clinically, both conditions can present with acute chest pain, making the differential diagnosis complex. Chest pain in AAD is usually tearing, occurring abruptly and radiating to the back or flank, whereas that in PE is often associated with acute dyspnoea and other signs of respiratory distress [8]. The coexistence of these two conditions can therefore lead to a difficult clinical picture, with symptoms of circulatory failure (hypotension, shock), respiratory distress (hypoxaemia, tachypnoea) and severe chest pain [3].

The ECG can provide important information, although it cannot specifically distinguish between AAD and PE [9]. In PE, the most common ECG abnormalities include sinus tachycardia, signs of right ventricular overload and changes in repolarisation (T wave inversion in V1-V3) [4]. In AAD, ECG abnormalities are generally less specific and include signs of myocardial damage or abnormal intraventricular conduction due to coronary involvement, which could lead to a false diagnosis of myocardial infarction. Rapid imaging is therefore essential to confirm the diagnosis.

Transthoracic echocardiography coupled with Doppler is an excellent tool for assessing these two pathologies in the emergency room. The 'EASY screening' protocol enables urgent and appropriate assessment of acute chest pain syndrome [10]. Aortic dissection is suggested by positive signs of effusion and/or abnormal aortic signs (insufficiency or dilatation) [10]. Pulmonary embolism is suspected in the presence of a dilated right ventricle and a D-shaped left ventricle in the short-axis view. Acute coronary syndrome is suspected by dyskinesia of movement of the left ventricular wall [10].

Treatment of this dual therapeutic challenge must be extremely rapid and personalised, with coordination between cardiologists, pulmonologists, vascular surgeons and intensive care units. Treatment strategies for PE include anticoagulation, which remains the first-line treatment in non-massive forms, and thrombolysis or surgery in severe cases [6]. However, the presence of an AAD means that these treatments must be used with great caution. Anticoagulation can worsen aortic dissection by promoting expansion of the aortic haematoma or even rupture [4]. Consequently, in a case of PE associated with an AAD, the therapeutic option depends on the patient's haemodynamic stability. Emergency surgery for aortic dissection is often the priority, followed by management of PE once the patient is stable. Treatments specific to AAD, including surgery or aortic stenting, should be considered without delay [8]. Management of PE may include the use of a mechanical decompression device, clot extraction by thrombectomy or anticoagulation, but the bleeding risks associated with AAD require careful risk-benefit assessment.

The prognosis of this combination is severely compromised, given the high mortality associated with each condition. In the case of massive PE, mortality can be as high as 30%, especially in the absence of early treatment [11]. Similarly, AAD has a guarded prognosis, with a mortality of 50% in the first 48 hours if not treated surgically [7]. The combination of the two conditions increases the risk of major complications, such as cardiogenic shock, acute respiratory failure and multiple organ failure [11]. However, rapid and appropriate management, depending on the patient's clinical course, improves the chances of survival.

CONCLUSION

This case report describes the therapeutic challenge presented by the combination of pulmonary embolism and Stanford type A aortic dissection extending to the coronary arteries. Rapid recognition of the clinical signs and appropriate diagnostic tests, followed by a well-coordinated therapeutic strategy, improved this patient's short-term prognosis. Although mortality remains high in this type of case, modern approaches such as endovascular surgery and combined management of the

three pathologies may offer hope, even in such dramatic situations.

AUTHOR CONTRIBUTIONS

Wendlassida Martin NACANABO: Conceptualization; Data curation; Formal analysis; Investigation; Visualization; Writing – original draft.

Taryètba André Arthur SEGHDA: Visualization; Writing – review & editing

Ella Hatoula LENGANI: Writing – original draft

Léa Francoise Wendlassida SAWADOGO: Writing – original draft

Anna TALL/THIAM: Supervision

Nobila Valentin YAMEOGO: Supervision

André Koudnoaga SAMADOULOUGOU: Supervision, validation

Patrice ZABSONRE: Supervision, validation

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DATA AVAILABILITY STATEMENT

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

CONSENT

We have obtained the patient's consent for publication. Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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