

A Case Report about an Unprovoked Life-Threatening Massive Pulmonary Thromboembolism with Successful Thrombolysis with Low Dose Thrombolytic Agent, A Case Report and Literature Review

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Abstract

Case Report

Pulmonary embolism is life-threatening emergency condition associated with high mortality, that can lead to a hemodynamic collapse and sudden cardiac arrest if prompt diagnosis is delayed. It usually needs an urgent intervention either pharmacologically or surgically such as systemic thrombolysis or surgical embolectomy, especially in patients who present in an obstructive shock. we report a case of 82 years old female patient with no medical past history of hypertension [HTN], neither diabetes mellitus [DM], she is a known case of asthma on medical treatment who was admitted with sudden shortness of breath, on examination she was tachypneic, tachycardic and hypotensive in the examination with elevated D-dimer and troponin I level. With indirect signs of pulmonary embolism in the echocardiography such as decreased acceleration time. A computed tomography pulmonary angiography [CTPA] confirmed the diagnosis of the pulmonary embolism. The patient was treated low molecular weight heparin initially then she was thrombolised with half dose of intravenous alteplase and later on she was discharged in a good medical condition on oral anticoagulant tablets twice daily. Her symptoms had resolved at the time of discharge and remain stable.

Keywords: Idiopathic, pulmonary embolism, thrombolytic drugs, miraculous response, lifelong treatment.

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INTRODUCTION

Acute Pulmonary Thromboembolism [PTE] is a common cause of cardiovascular arrest worldwide, and is consider the third most frequent thrombotic syndrome after stroke and myocardial infarction.

Usually, the embolization by a thrombus originates and migrates from outside the lungs especially the deep veins of the lower extremities or can also occur de-novo or idiopathic without any evidence of deep vein thrombosis [DVT]. It requires rapid and efficient diagnosis and management to prevent life-threatening outcomes.

CASE PRESENTATION

we report the case of an 82 years old female patient with a known past medical history of a HTN nor DM, a known case of asthma on medical treatment, who

was admitted complaining of an orthopnea, paroxysmal nocturnal dyspnea for three days, then she developed shortness breath of sudden onset. On examination she was tachypneic [respiratory rate:30 cycles per minute] with oxygen saturation at 78% in the room air, hypotension [BP=87/45mmHg], with heard[S1], second[S2] and forth[S4] heart sounds. Abdominal and neurological examinations were normal. D-dimer was markedly elevated- 21.32ug/ml [Normal <0.5ug/ ml]. Troponin I was also elevated=876 ng/l [Normal<17ng/l]. Complete blood count [CBC], clotting factors, renal function, liver function, electrolyte and thyroid function tests were in the normal range [table 1]. The tumor markers were also in the reference range [table 2]. A thoraco-abdomino-pelvic computed tomography [CT] was realized and was also normal. Electrocardiography [ECG] showed sinus rhythm ,with diffuse T wave inversion in anteroseptal and inferior leads [Figure-1] , Chest X-ray showed a cardiomegaly while the

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echocardiography showed a diminished acceleration time, with notching ,dilated right ventricle with right ventricular dysfunction associated grade II tricuspid regurgitation with presence of 60/60 sign[Figure-2]. Doppler Ultrasound of both lower limbs showed no obvious intra luminal thrombus, no calcifications in the vessel walls and no loss of compressibility of the deep veins of both lower limbs. A Computed tomography pulmonary angiography CTPA] was immediately realized and it confirmed the diagnosis of a massive bilateral, pulmonary embolism [Figure-3]. Then she received a curative dose of low molecular weight heparin, then thrombolised with a small dose of alteplase intravenously, dramatically within one hour the patient's oxygen saturation and blood pressure ware stabilized,

O2=94% and BP 110/50 mmHg respectively, later she was discharged in a good medical condition on Apixaban twice daily tablets. Her symptoms had resolved at the time of discharge and she remains stable till to date.

In the literatures, Unprovoked venous thromboembolism [VTE] is very rare and has the potential to lead to pulmonary embolism which could be disastrous, especially in young adults. We present such a case where unprovoked VTE was diagnosed and treated. This case suggests that high clinical suspicion is the key for the diagnosis of acute pulmonary embolism, especially in the absence of history suggestive of deep vein thrombosis.

Table 1

Test	Value	Normal range
HB	13.5	13-17g/dL
WBC	5500	4000-7000
Platelet	150000	150 000-400 000
Creatinine	6.7	5.7-12.5mg/L
Urea	0.25	0.15-0.55g/L
CRP	63	<5mg/L
Troponin	876	<17ng/mL
TSH	0.22	0.35-4.94uUI: mL
FT4	1.03	0.7-1.48pg/mL
FT3	2.37	1.71-3.71pg/mL
AST	54	5-34IU/L
ALT	60	0-55IU/L
INR	1	

Table 2

Test	Value	Normal range
CA19-9	14	<37 IU/ml
CA125	10.4	<35IU/ml
CA 153	14.4	<31.3 IU/ml
AFP	<2	0.89-8.87ng/ml
ACE	4.91	<5ng/ml

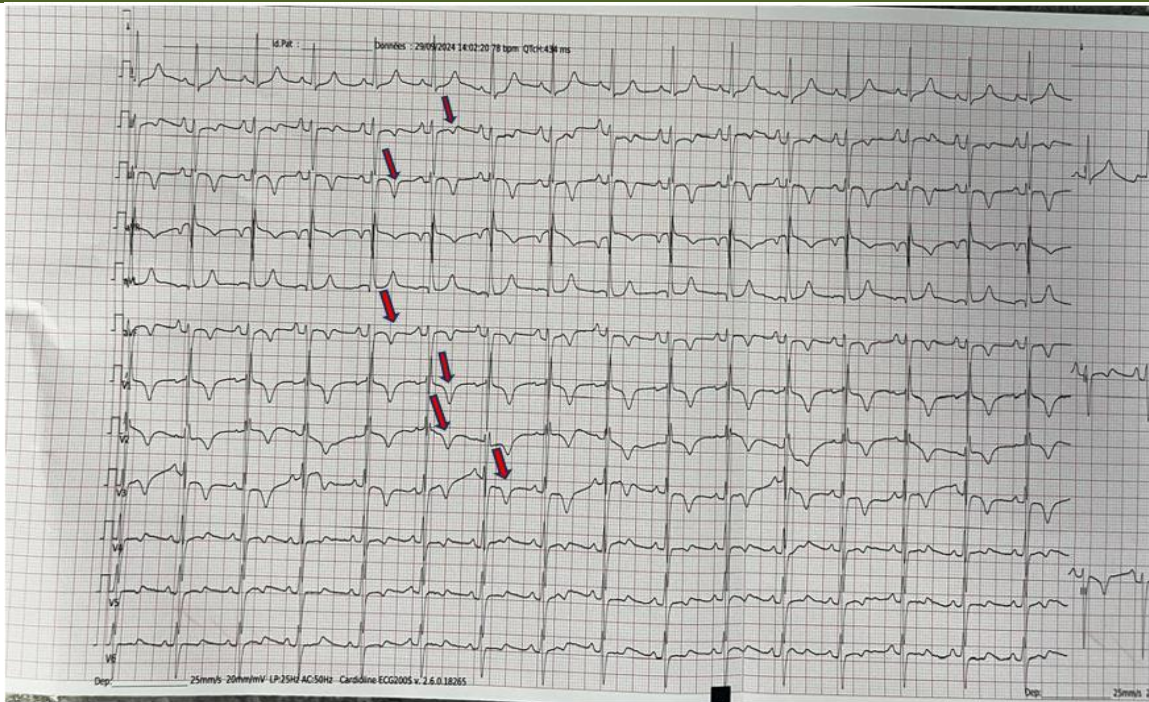


Figure-1

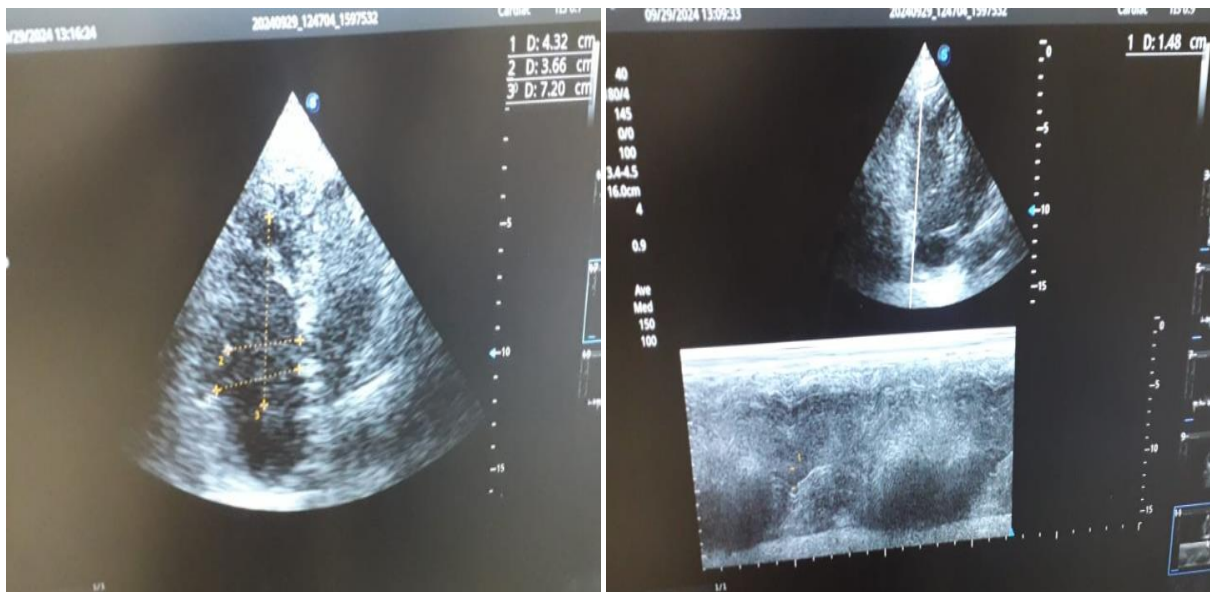




Figure-2



Figure-3

DISCUSSION

Acute Pulmonary Thromboembolism [PTE] is a common cause of cardiovascular arrest in the worldwide, and is consider the third most frequent thrombotic syndrome after stroke and myocardial infarction.

The acute pulmonary arteries obstruction is responsible of attributing right ventricular pressure overload which cause a cardiogenic shock and death. Usually, the embolization by a thrombus originates and migrates from outside the lungs especially the deep veins of the lower extremities or can also occur de-novo or idiopathic without any evidence of deep vein thrombosis [DVT]. Virchow was who described firstly the process of the processes of thrombosis as a triad of stasis, hypercoagulability, and endothelial injury [1].

Acute pulmonary embolism [PE] requires rapid and efficient diagnosis and management to prevent life-threatening outcomes.

The literatures show that the annual incidence of PE ranges from 39 to 115 per 100,000 populations [2].

According to the European Society of Cardiology [ESC] and the American Heart Association

[AHA], the PE is categorized into three prognostic groups which are as follow: a high-risk group [massive] that represents 20% of cases, an intermediate-risk group [sub-massive] represents 32 % of cases and a low-risk group [non-massive] represents 48% of patients who are diagnosed with PE. The overall death rates of massive, sub-massive and non- massive PE are 71.4%, 44.5% and 28.1% respectively .50% of patients with massive PE die in the first 30 minutes, within 1 hour 70% of them die, and more than 85% die within 6 hours of clinical symptoms onset [3].

The causes or triggers of pulmonary embolism [PE] can be an inherited thrombophilia, a recent major operation, a recent hip fracture, a pregnancy, the hormonal replacement therapies and central lines. The patients having the above-mentioned causes or triggers make a group of what is commonly called a provoked PE [PPE], on the other hand there is a second group which is called unprovoked PE or idiopathic PE and is defined as a PE without a trigger or an obvious cause [4].

The used diagnostic tools when a PE is suspected are the ECG, transthoracic echocardiography and pulmonary CT -angiography. Peripheral veins

doppler ultrasound [US] is also one of the tools which are used to identify the cause of a PE. Blood labs as the D-dimer and troponin I also are used. Tumor markers can guide to the cancerogenic PE. CBC, bleeding factors, renal and liver functions also are tested before initiation of the treatment.

In the literature, the unprovoked massive bilateral pulmonary embolism with a spectacular recovery by thrombolytic drugs is unusual and it represents a rare clinical combination of an idiopathic event and an exceptional therapeutic outcome and a high clinical suspicion is the key for the diagnosis of acute PE, especially in the absence of history suggestive of provoked causes [5].

In patients with high-risk PE, thrombolytic therapy should be given in case of hemodynamic instability as in the case of our patient [6].

All patients should receive at least 3 months of anticoagulation after a PE but as the risk of recurrence after stopping treatment in unprovoked PE, exceeds 5–10% per year a long-term [indefinite] anticoagulation is advised in most cases, if no contraindication or any bleeding risk. A dose reduction is considered for long-term secondary prevention [7].

CONCLUSION

The discussed case above shows the importance of high sense of suspension from the first presentation of the patient in the emergency department even in the absence of any risk factor, as the idiopathic cases can occur. Thrombolytic drugs administration as soon as the diagnosis is reached can lead to miraculous recovery. As the cause is not reached, life long oral anticoagulants are advised.

Conflict of Interests:

The authors declare that there is no conflict of interests regarding the publication of this paper.

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