MHA MEDIHEALTH ACADEMY

Eurasian Journal of
Anesthesiology & Intensive Care
Case Report

doi
10.51271/EAJAIC-0009

Bedside management of massive pulmonary embolism: a case report

Seyyid Furkan Kına, Savaş Altınsoy, Yusuf Özgüner, Gizem Avcı, Jülide Ergil

Department of Anesthesiology and Reanimation, Ankara Etlik City Hospital, Ankara, Turkiye

Received: 15/03/2024 ◆ **Accepted**: 01/04/2024 ◆ **Published**: 16/05/2024

Cite this article: Kına SF, Altınsoy S, Özgüner Y, Avcı G, Ergil J. Bedside management of massive pulmonary embolism: a case report. *Eurasian J Anesthesiol Intens Care.* 2024;1(2):42-44.

Corresponding Author: Seyyid Furkan Kına, kinafurkan@gmail.com

ABSTRACT

Pulmonary thromboembolism can lead to a clinical syndrome that can be confused with acute myocardial infarction, aortic dissection, tamponade, acute valvular dysfunction, cancer and pneumonia. In this report, we present a case of acute massive pulmonary embolism with a possible chronic background who was followed and treated in various centers.

Keywords: Acute pulmonary embolism, echocardiography, thrombolytic therapy

INTRODUCTION

Massive pulmonary embolism (MPE) is a severe form of venous thromboembolism in which large blood clots occlude the pulmonary arteries and disrupt ventilation/perfusion ratios.[1] Despite advances in diagnosis and treatment, the correct approach is still a matter of debate.[2] Many diseases predispose to MPE and make the diagnosis difficult.[2] The treatment of this fatal disease with low diagnosis rates may also vary on a patient basis. Heparin is an indispensable agent for MPE to prevent the spread of blood clots and their movement to other parts of the body. However, thrombolytic agents have reduced the high mortality rates to remove the formed clot.[3] Although thrombolytic agents reduce high mortality rates, they should be used with caution because they predispose to bleeding.

In this case report, we would like to describe the diagnosis and successful management of a case of MPE characterized by atypical symptoms using only bedside methods under emergency conditions.

CASE

A 68 year old man with hypertension, insulin dependent diabetes mellitus and Pulmonary thromboembolism (PTE) (3 years ago). He presented to the hospital with intermittent hemoptysis, increasing exertional dyspnea and typical chest pain while on apixaban treatment. Echocardiographic (EKO) evaluation revealed an ejection fraction (EF) of % 50 and no overt valvular disease. Myocardial perfusion scintigraphy was planned for his complaints. The patient was re-evaluated in another center due to persistent hemoptysis. After D-dimer and deep venous doppler, hemoptysis was attributed to apixaban treatment and apixaban was discontinued and acetylsalicylic acid treatment was started. Two weeks after this treatment change, the patient was evaluated in another emergency department due to increased dyspnea. Troponin-I follow up showed a course of 19/40/50 ng/L. Diagnostic coronary angiography was planned considering myocardial infarction without ST segment elevation. Coronary angiography revealed a 70% lesion in the left anterior descending coronary artery (LAD) osteal (Figure 1).

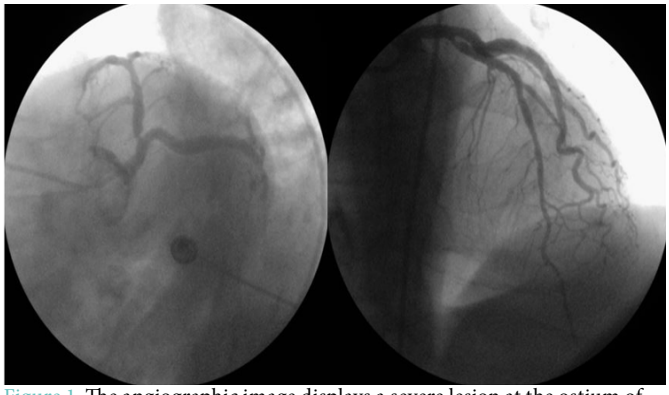

Figure 1. The angiographic image displays a severe lesion at the ostium of the left anterior descending coronary artery (LAD), despite which the flow is maintained at TIMI 3 level

One day later, LAD- left internal mammary artery (LIMA) bypass was performed in the working heart and the patient was discharged with clopidogrel treatment. The patient was admitted to different centers within a two-week period after his complaints did not improve. Furosemide and levofloxacin were started considering atypical pneumonia and heart failure. The patient was admitted to our center on the 20th day after coronary artery bypass graft (CABG) due to increased dyspnea.

Initial examination revealed overt lobar pneumonia and signs of mild overload. Laboratory values; C reactive protein (CRP) 167 mg/L, troponin-I 26 ng/L and renal function tests were normal. EKO showed EF 45%, anterolateral wall was mildly hypokinetic, grade 1 tricuspid valve insufficiency and systolic pulmonary artery pressure was 45 mmHg. These findings were considered as sequelae of old PTE. Chest radiography showed bilateral basal infiltrations. Considering atypical pneumonia and mild overload with the current findings, intravenous antibiotherapy and diuretic treatment were planned and the patient was hospitalized in the intensive care unit.

On admission, 1x0.8 mg enoxaparin was started at a prophylaxis dose. Ramipril and sprinolactone were started at low doses for left ventricular dysfunction and signs of overload. Clopidogrel and metoprolol treatment was continued. On the 2nd day, CRP decreased to 100 mg/L and the patient's fever was under control. On physical examination, wheezing and rhonchi regressed. On the 3rd day of follow up, sudden hypotension and tachycardia developed starting with presyncope. Dopamine was initially started for hypotension and noradrenaline was added in the follow up.

Type 5 myocardial infarction, acute left ventricular failure, septic shock, acute renal failure and PTE which may be LAD-LIMA related were considered as differential diagnosis. Serial electrocardiography (EKG) monitoring was performed, but no evolutive changes were observed except sinus tachycardia, troponin monitoring showed moderate elevation and the diagnosis of myocardial infarction was ruled out. D-dimer resulted as 7141 ng/ml.

Arterial blood gas samples taken at 15 minute intervals showed deepening hypocarbia. EKO follow up revealed progressive deterioration of right ventricular function, dilated right ventricle and severe tricuspid regurgitation. Pulmonary artery pressure was measured as 65 mm Hg at this stage. Upon documentation of typical findings such as "D" finding in the left ventricle and right ventricular failure with preserved apical function, the patient was diagnosed with acute massive pulmonary embolism (Figure 2). Acute renal failure was considered because the patient had severe orthopnea and anuria. Pulmonary angiography could not be planned under these conditions. The patient was clinically and echocardiographically accepted as pulmonary embolism and Alteplase treatment was planned.

Alteplase was administered as 5 mg bolus and 45 mg 1 hour infusion at a total dose of 50 mg. Serial EKG and neurologic monitoring were performed during administration.

The patient had no symptoms except mild chest burning. Alteplase treatment was completed without complications. In approximately one hour, neuradrenaline support was discontinued and dopamine dose was reduced to 3 mcg/kg/min. Alteplase treatment was not completed to 100 mg considering possible hemorrhagic complications due to the onset of significant improvement in hemodynamics. Meanwhile, right ventricular failure regressed, the "D" sign disappeared and pulmonary artery pressure started to decrease (Figure 3).

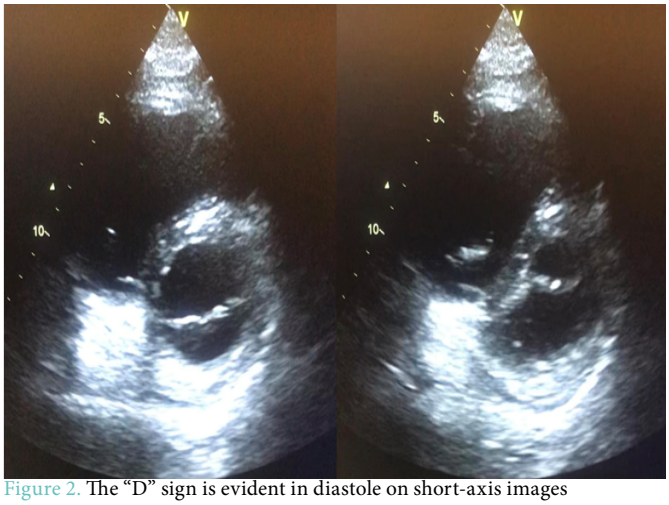

Figure 2. The "D" sign is evident in diastole on short-axis images

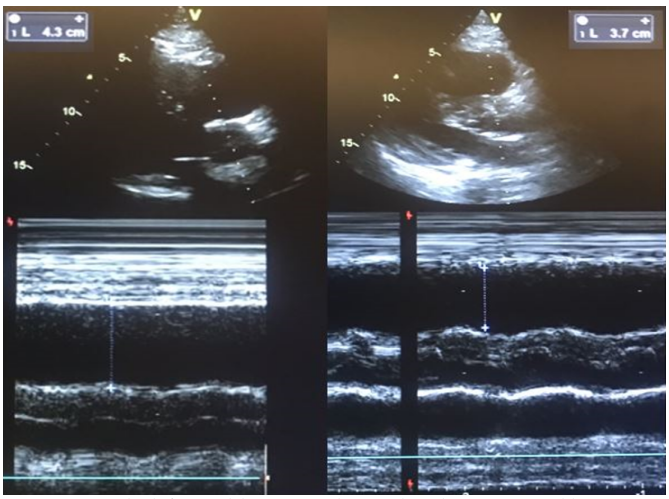

Figure 3. On the left, RV dilatation is depicted in the parasternal long axis view. On the right, a reduction in RV size is observed at 2 hours following thrombolytic treatment

Within four hours, the patient's urine output started and oxygen requirement decreased. One hour after alteplase treatment, respiratory rate and effort also decreased markedly. Hypocapnia improved on EKG follow up. Lactate level decreased from 8 mmol/L to 1.6 mmol/L.

Heparin infusion was started 12 hours after the alteplase dose and infusion was maintained for 72 hours with an activated partial thromboplastin time of 45 seconds. Treatment was continued with warfarin due to impaired renal function. The patient was successfully discharged without complications.

DISCUSSION

The European Society of Cardiology recommends >3 months of anticoagulation therapy in all PTE's.[4] If the embolism is related to an identifiable transient condition, anticoagulation

may be discontinued after 3 months of treatment with a class 1-B recommendation. However, lifelong anticoagulation therapy is recommended in PTE's associated with recurrent deep vein thrombosis or antiphospholipid syndrome, which are not related to a transient secondary condition. In lifelong treatment, after 6 months of full dose, reduced doses are recommended with class 2-A recommendation (such as apixaban 2.5 mg BID, rivaroxaban 10 mg).[5]

When we retrospectively analyzed our patient, exertional dyspnea 2 months ago could be attributed to the 70% lesion in the LAD and hemoptysis could be attributed to apixaban 5 mg BID treatment since D-dimer was normal at that time. At this stage, in line with guideline recommendations, instead of discontinuing apixaban completely, it may be preferable to taper the dose and plan diagnostic coronary angiography.

On angiography images, coronary flow can be interpreted as Thrombolysis in Myocardial Infarction (TIMI) 3 despite a severe lesion with 70% stenosis of the LAD osteal. This may account for the mild troponin elevation and explain the patient's symptoms. However, persistence of hemoptysis despite discontinuation of anticoagulation should be a warning that there may be another underlying etiology. In such cases, instead of attributing the symptoms to a single cause, investigating other possible conditions and in this case, bringing PTE to mind may provide an earlier diagnosis.

CONCLUSION

PTE, which is frequently confused with coronary artery disease and heart failure in terms of symptoms, has a poor prognosis with sudden hemodynamic collapse when left untreated. A European epidemiologic study showed that only 7% of patients who died of PTE were started on time. Treatment could not be initiated in 34% of patients and the diagnosis could only be made by autopsy in 59%.[6] These epidemiologic data show that PTE is an insidious disease.

Although the gold standard diagnostic methods of pulmonary embolism are computed tomography, pulmonary angiography, ventilation/perfusion scintigraphy or pulmonary arteriography, it is not technically possible to apply these methods in cases where hemodynamics are impaired. Concomitant renal failure limits the use of radiopaque material. In our case, profound hypotension, anuria and severe orthopnea developed within minutes and only bedside methods were used for diagnosis. In such an unstable patient population, thrombolytic therapy can be administered after confirmation of the diagnosis with serial arterial blood gas and ECO follow up.

ETHICAL DECLARATIONS

Informed Consent Form

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Aykan AÇ, Gökdeniz T, Gül İ, et al. Reduced-dose systemic fibrinolysis in massive pulmonary embolism: a pilot study. *Clin Experim Emerg Med*. 2023;10(3):280-286. doi: 10.15441/CEEM.23.015
2. Mohamad T, Kanaan E, Ogieuhi IJ, et al. Thrombolysis vs anticoagulation: unveiling the trade-offs in massive pulmonary embolism. *Cureus*. 2024;16(1):e52675. doi: 10.7759/CUREUS.52675
3. Khandait H, Harkut P, Khandait V, Bang V. Acute pulmonary embolism: diagnosis and management. *Indian Heart J*. 2023;75(5):335-342. doi: 10.1016/j.ihj.2023.05.007
4. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS):the task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). *Eur Respir J*. 2019;54(3):1901647. doi: 10.1183/13993003.01647-2019
5. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *Eur Heart J*. 2020;41(4):543-603.
6. Cohen AT, Agnelli G, Anderson FA, Arcelus JI, Bergqvist D, Brecht JG. Venous thromboembolism (VTE) in Europe. The number of VTE events and associated morbidity and mortality. *Thromb Haemost*. 2007;98(4):756-764.