

 Veli Burak Tackin¹,  Burak Ustun²,  Rauf Onder³

¹Antalya Training and Research Hospital, Department of Cardiovascular Surgery, Antalya, Türkiye

²Ardahan State Hospital, Department of Emergency, Ardahan, Türkiye

³Akdeniz University Hospital, Department of Cardiovascular Surgery, Antalya, Türkiye

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Corresponding Author: Veli Burak Tackin

Antalya Training and Research Hospital, Department of Cardiovascular Surgery, Antalya, Türkiye

Email: brk.veli@gmail.com

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Challenges in pulmonary embolism-related pleural effusion: Tube troubles with methemoglobinemia

Abstract

Pulmonary embolism (PE), often arising from deep vein thrombosis, can present with pleural effusion. Inserting a chest tube for massive effusions carries inherent risks, including chest tube malposition and methemoglobinemia. We report a case of a 32-year-old female with moderate-to-high risk PE and massive pleural effusion. Complications included chest tube malposition, secondary pneumothorax, and methemoglobinemia. Initial chest tube placement drained pleural fluid but resulted in pneumothorax and methemoglobinemia due to prilocaine use. Careful management and monitoring were crucial. Pleural effusion can complicate PE, warranting interventions, but complications like chest tube malposition and methemoglobinemia should be anticipated and managed promptly.

Keywords: Pulmonary embolism, pleural effusion, pneumothorax, methemoglobinemia

INTRODUCTION

Pulmonary embolism (PE) is a medical condition characterized by the occlusion of pulmonary arteries, resulting in high morbidity and the potential for a fatal course (1). PE develops in 20% of untreated proximal deep vein thrombosis, and 10-20% of these cases result in fatality. Effective anticoagulation can reduce the mortality rate by 5-10 times (2). The most common causes of pleural effusion are; congestive heart failure, cancer and pneumonia, and PE comes in 4th place (3). In the presence of massive effusion causing respiratory failure, thoracentesis is required.

Intercostal placement of a chest tube is generally considered an easy and fairly straightforward bedside procedure. However, this procedure carries the risk of several complications (4). Local anesthetic agents are commonly used during chest tube placement, as in other minor surgical procedures. Occasionally, the use of local anesthetic agents can rarely lead to methemoglobinemia, which can result in respiratory failure (5).

In this case, we presented many complications encountered in a medium-high risk PE patient who presented to the emergency department with a complaint of respiratory distress accompanied by massive pleural effusion.

CASE

A 32-year-old, 45 kg female patient with mental disability and limited mobility applied to the emergency department with a complaint of shortness of breath that started 1 hour ago. When she arrived at the emergency room, her general condition was moderately poor, she was breathing spontaneously, she was conscious and slightly confused, and vital signs were; heart rate of 135/min, blood pressure of 80/40 mmHg, temperature of 36.6°C, respiratory rate of 30/min, oxygen saturation 76% without oxygen support. Upon physical examination, breath sounds could not be auscultated in the right hemithorax. The electrocardiogram did not reveal any specific pathology. In the chest X-ray, no lung parenchyma was observed in the right hemithorax and mediastinal shift was accompanied (Figure 1).

In a hypotensive, tachycardic, hypoxic patient with the absence of breath sounds in the right hemithorax and mediastinal shift on the chest X-ray, the possibility of tension pleural effusion was considered. PE could not be ruled out, but the decision was made to place a chest tube firstly to correct the mediastinal shift. Under local anesthesia, a chest tube was inserted at the 5th intercostal space along the right midaxillary line. Approximately 2000 cc of serous fluid was drained, and the patient's blood pressure improved to 110/70 mmHg, heart rate to 110/min, and oxygen saturation to 95%.

Given the patient's normotensive status after chest tube insertion, high-risk PE was not considered. However, a chest X-ray revealed malpositioning of the chest tube, which settled in the basal part of the right hemithorax (Figure 2). During tube repositioning, a secondary pneumothorax occurred, and the tube resettled in the same basal area (Figure 3). The chest tube was removed, and a new tube was placed toward the apex. Tube oscillation was normal, air movement was present, and no pleural effusion or pneumothorax was detected on the follow-up chest X-ray (Figure 4). Echocardiography performed after chest tube placement: Ejection fraction: 60%, no contraction defect, right ventricle (RV) / left ventricle (LV) diameter >1 , no aortic and mitral valve regurgitation, no tricuspid regurgitation, McConnell negative. There is no D septum.

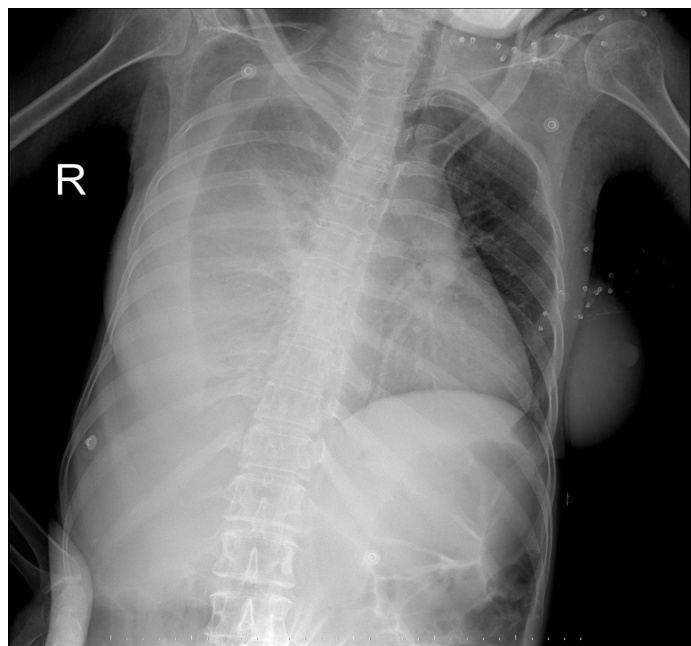


Figure 1. Chest X-ray taken at the time of admission to the emergency department

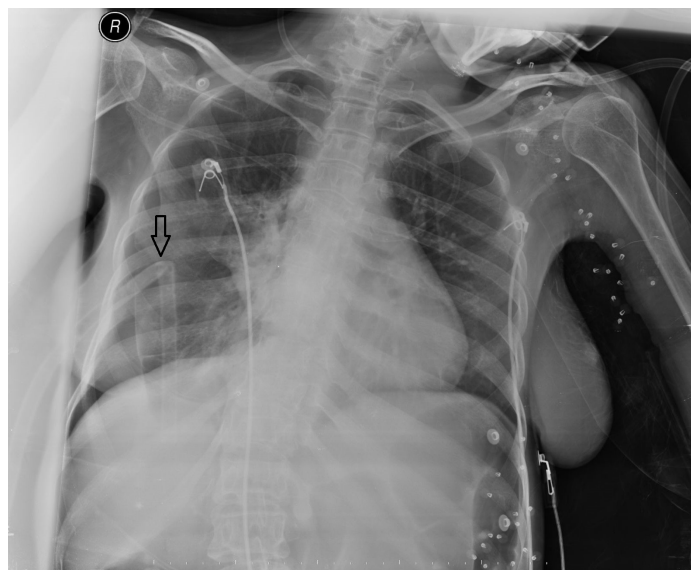


Figure 2. First chest X-ray after chest tube placement and malposition of the tube from king to basal

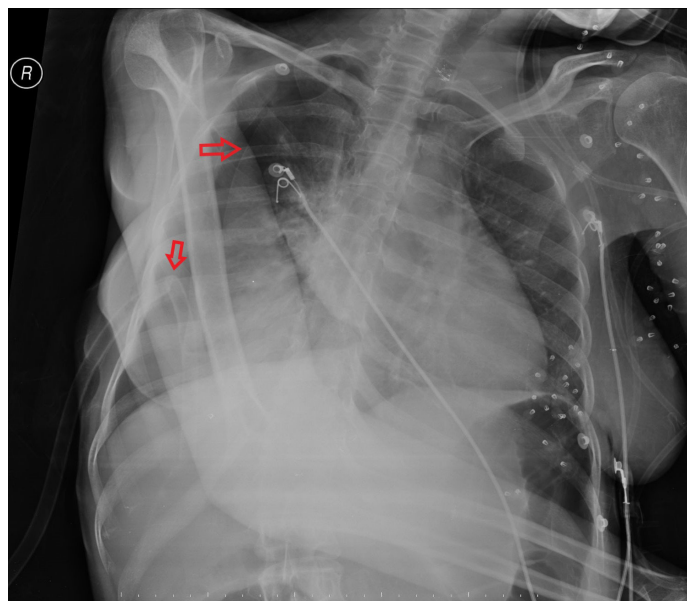


Figure 3. Chest X-ray showing the secondary pneumothorax line resulting from the second placement of the chest tube and the basal malposition of the tube



Figure 4. Chest X-ray showing the final state of the chest tube placed for the 3rd time

In the initial presentation, the venous blood gas analysis showed a pH:7.45, pCO₂ :26 mmHg, pO₂:46 mmHg, cHCO₃:20.8 mEq/L, cBase (ecf):-5.4, and methemoglobin : 1.1%. Troponin and C-reactive protein levels were 10 times higher than the upper limit. Other laboratory parameters were within normal ranges. During the follow-up of the patient, whose clinical stabilization was thought to have been achieved, sPO₂ decreased from 10 lt/min to 85% with the support of a mask with a reservoir. The patient was reevaluated for complications. Given the absence of crackles and wheezes in lung sounds, re- expansion injury was not considered prominent.

A computed tomography (CT) angiography revealed a 1.5

cm pleural effusion in the right hemithorax, along with thromboembolic findings in the right main pulmonary artery, right lower lobe artery, left main pulmonary artery, and left lower lobe artery. Enlargement of the right ventricle with an RV/LV ratio of 1.5 was observed. In computerized tomography (CT) angiography, the patient had a 1.5 cm pleural effusion in the right hemithorax, an appearance compatible with thromboembolism in the right main pulmonary artery, the artery leading to the right lower lobe, the left main pulmonary artery, the artery leading to the left lower lobe, and enlargement of the right ventricle. RV/LV: 1.5 was detected. Because the patient was normotensive, had high troponin levels, and had widening of the right cavities, it was considered as medium-high risk PE. Therapeutic doses of low-molecular-weight heparin were initiated.

Upon observing an sPO₂ of 85% despite receiving maximum oxygen support, methemoglobinemia was considered. In control arterial blood gas, pH: 7.39, pCO₂: 28 mmHg, pO₂: 54 mmHg, cHCO₃: 18.8 mEq/L, Base (Ecf): -8, methemoglobin: 7.0% were detected and methemoglobinemia was diagnosed. In the new blood gas taken 1 hour later, pH: 7.36, pCO₂: 32 mmHg, pO₂: 61 mmHg, cHCO₃: 18.8 mEq/L, Base (Ecf): -7.1, methemoglobin: 16.7% were detected.

The patient was admitted to the chest diseases service for further monitoring and treatment. A blood gas analysis performed four hours later showed a methemoglobin level of 7%, decreasing to 2% in the analysis conducted twelve hours later. With 2 L/min nasal oxygen, the sPO₂ improved to 95%. On the third day, the chest tube was removed from the patient who showed no air leak and had no pleural effusion on the chest X-ray. Discharged with a prescription for oral anticoagulants on the seventh day, the patient achieved recovery.

DISCUSSION

PE is the top cause of death from cardiovascular diseases and is responsible for approximately 300000 deaths annually. Major trauma, surgery, lower limb fractures, joint replacements, and spinal cord injury are strong provoking factors for PE. While paralytic stroke is considered a moderate risk factor, immobility for more than 3 days is categorized as a low risk factor (6). In our case, the patient had been immobilized due to mental retardation for an extended period, and no prophylactic measures such as anticoagulation or antiembolism stockings were in place. According to the ESC 2019 acute PE guideline, there is no indication for urgent thrombolysis in patients with medium-high risk PE, but there is a relative indication (6). We treated with therapeutic doses of low molecular weight heparin.

Small uncomplicated pleural effusions can be managed under observation unless it causes respiratory dysfunction. However, effusions that do not respond to medical treatment, causing respiratory failure, malignant effusions, and complicated parapneumonic effusions will likely require drainage. The most common indications for placing a chest tube include exudative

effusions, empyema, hemothorax, pneumothorax, and post-thoracic surgery (7). In this case, an chest tube was placed due to the presence of massive pleural effusion in the right hemithorax, resulting in respiratory failure and mediastinal shift.

Chest tube placement is generally considered a simple bedside procedure. Although tube thoracostomy is one of the commonly used procedures today, complication rates of up to 37% can be observed in tube placement due to this procedure (8). It carries various risks and potential complications, including diaphragmatic, mediastinal, cardiac, major vascular, esophageal injury, and extrathoracic organ injuries, such as hepatic, gastric, or splenic injuries, as well as the potential to cause secondary pneumothorax (9). Malposition occurred as a complication in 34.8% of patients with chest tube insertion, and 13.3% of the patients required tube repositioning and 3.8% required tube replacement (10). In a large case series study, iatrogenic pneumothorax developed in 36.9% of patients with chest tube insertion (11). What makes this case interesting is the occurrence of multiple complications in the same patient following chest tube placement. The chest tube malposition led to its initial placement at the basal region of the hemithorax. Subsequently, during an attempt to reposition the chest tube towards the apex, it was inadvertently placed back into the basal region, resulting in secondary pneumothorax. Finally, the existing chest tube was removed, and a new tube was correctly placed in the hemithorax towards the apex.

Prilocaine is a commonly used local anesthetic agent that can lead to methemoglobinemia. It has a half-life of approximately 55 minutes, and methemoglobinemia can start to develop within 20 to 60 minutes after its administration (12). Methemoglobinemia was detected in 1% of patients after the use of local anesthetic agents (13). The treatment for methemoglobinemia usually involves administering methylene blue. Prilocaine dosing should be carefully considered. The recommended dosage for treatment is 1 mg/kg, and the maximum safe prilocaine dose for adults is 8 mg/kg or 600 mg per administration (14).

Our patient's case, the administered prilocaine dose was 500 mg. However, methemoglobinemia risk can be influenced by various factors beyond just the dose. The patient's cachectic condition and thin subcutaneous fat tissue, in combination with the site of injection, likely contributed to the increased risk of methemoglobinemia. Prilocaine oxidizes hemoglobin and can lead to methemoglobinemia. Normally, the body's regulatory systems maintain methemoglobin levels below 1-2%. When levels exceed this range, it can lead to respiratory failure (5). We supplied 100% oxygen therapy, saline infusion. Regular monitoring was crucial. The patient's methemoglobin levels decreased after 4 hours and returned to normal after 12 hours, indicating successful management of this complication.

CONCLUSION

In conclusion, we have seen most of the complications that may

be encountered in the management of a patient with massive pleural effusion in a single case. Although pleural effusion is not a diagnosis, it occurs due to the underlying disease. In immobilized patients presenting to the emergency department with symptoms like shortness of breath, PE should be considered. While pleural effusion can be associated with various causes, it can also be related to PE, and in cases where massive pleural effusion leads to respiratory distress, procedures such as thoracentesis may be required. However, simple surgical procedures like the placement of an chest tube can carry a set of risks that can lead to serious complications. If the patient remained hypotensive after chest tube placement, urgent thrombolytic therapy would have to be given, which would lead to other complications in the patient who had just undergone the surgical procedure. Methemoglobinemia may develop due to the use of local anesthetic agents and rapid diagnosis and treatment is required. It is necessary to know the complications of the surgical procedure and patient follow-up. The focus should not only be on the surgical procedure performed, but other additional pathologies should not be overlooked.

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