



The Role of Emergency Pleural Drainage in the Obstructive Shock in a Left Massive Hemothorax: A Case Report

Krisdianto Putra Lainy*¹, Charles Saputra², Januario Bria³, Oka Wijaya⁴, Afina Chaerunnisa⁵, Radietya Alvarabie⁶

¹Atambua Hospital, Atambua

²Department of Cardiology and Vascular Medicine, Atambua Hospital, Atambua

³Department of Surgery, Atambua Hospital, Atambua

⁴Department of Pulmonology and Respiratory Medicine, Atambua Hospital, Atambua

⁵Faculty of Medicine, Nursing and Health Science, Monash University, Melbourne

⁶Faculty of Medicine, Universitas Pertahanan Republik Indonesia, Bogor

Corresponding Author:

Kridianto Putra Lainy | Atambua Hospital, Atambua | lainy_andre@yahoo.com

Submitted: November 29th, 2023

Accepted: March 20th, 2023

Published: June 28th, 2024

Respir Sci. 2024; 4(3): 172-9

<https://doi.org/10.36497/respirsci.v4i3.139>

Abstract

Background: Spontaneous hemothorax is much less common, and the causes include malignancies, anti-coagulant medications, vascular ruptures, endometriosis, pulmonary infarctions, adhesions with pneumothorax, and hematologic abnormalities such as hemophilia. This report presented a confirmed case of obstructive shock in a patient with massive left hemothorax and elaborated on the role of emergency pleural drainage in this particular clinical situation.

Case: Reported a case of a 56-year-old man with a 2-month history of dyspnea, leg swelling, abdominal bloating, hemoptysis, and fatigue. Physical examination revealed an asymmetrical chest wall expansion with a predominance of abdominal breathing. His neck and face were markedly discolored and swollen, with distended veins. The left hemithorax was dull on percussion and, on auscultation, significantly reduced air entry at the left lung base.

Discussion: The patient was given an O₂ non-rebreathing mask (NRBM) on arrival. The patient was administered two vasopressors (dopamine, 2.5 mcg/kg per body weight/minute, and norepinephrine, 0.1 mcg/kg per body weight/minute). Given the patient's hemodynamic instability and high probability of imminent death. The patient required urgent intervention to relieve the obstructive shock. The surgical department was inserted to treat the massive hemothorax, which drained approximately 1.5 liters of blood. The patient received supplementary oxygen, antibiotics, and furosemide.

Conclusion: The pleural fluid drainage alleviated the dyspnea. As supportive therapy, the patient received oxygen, antibiotics, and furosemide. On the follow-up, the mediastinal shift had resolved simultaneously.

Keywords: emergency pleural drainage, hemothorax, obstructive shock



[Creative Commons Attribution-NonCommercial 4.0 International License](https://creativecommons.org/licenses/by-nc/4.0/)

INTRODUCTION

Severe blunt force to the chest frequently leads to extensive thoracic bleeding, or hemothorax, primarily resulting from numerous rib fractures. Additionally, it can cause injuries to the pulmonary, cardiovascular, and diaphragmatic systems.¹

Hemothorax is characterized by pleural fluid containing a hematocrit equal to or exceeding 50% of the hematocrit in the peripheral blood.² A pleural effusion is characterized by fluid accumulation between the visceral and parietal layers of the pleura.³ A pleural effusion is classified as hemorrhagic when the red blood cell count in the pleural fluid exceeds 100,000 cells/ μ l.²

Spontaneous hemothorax is much less common, and the causes include malignancies, anti-coagulant medications, vascular ruptures, endometriosis, pulmonary infarctions, adhesions with pneumothorax, and hematologic abnormalities such as hemophilia.⁴

The prevailing cause of trauma typically involves either blunt or penetrating injuries to structures within or outside the thoracic region, leading to hemorrhagic entry into the thoracic cavity. Hemorrhage can arise from various sources, including the chest wall, intercostal or internal mammary arteries, great vessels, mediastinum, myocardium, lung tissue, diaphragm, or abdominal region.⁵

A substantial hemothorax has the potential to elevate hydrostatic pressure,

applying force to the vena cava and pulmonary parenchyma. Elevated hydrostatic pressure can disrupt preload, elevate pulmonary vascular resistance, and ultimately lead to tension hemothorax.⁵ These processes contribute to hemodynamic instability, cardiovascular collapse, and fatality.

There are few reports of spontaneous hemothorax causing obstructive shock.⁴ Obstructive shock is linked to the mechanical obstruction of blood flow to the heart, particularly affecting the left ventricle. The typical pathophysiology of obstructive shock involves a decrease in the preload of the left ventricle (LV).⁶

Elevated intrathoracic pressure interferes with venous return in tension pneumothorax, while heightened right ventricular (RV) afterload obstructs blood flow from the right to the left heart in pulmonary embolism. Diminished cardiac compliance disrupts the heart's diastolic filling in cardiac tamponade.⁶

The decrease in LV preload results in a relative elevation of both LV contractility and heart rate, but ultimately, stroke volume and cardiac output (CO) decrease.⁶ Due to the blockage of blood flow, swollen jugular veins and an enlarged inferior vena cava (IVC) are frequently noticeable during physical examinations or bedside sonography.⁴

Obstructive shock is characterized by the critical need to identify the obstructive cause for effective management, and the response to treatment is rapid. Moreover, patients with impaired consciousness or severe shock require airway management.

The initial stage of circulatory support involves volume resuscitation. In patients experiencing obstructive shock, the venous system is similar to patients with volume overload.⁷

The jugular vein and inferior vena cava are swollen, and the pressure of both central venous and pulmonary artery occlusions increases.⁴ Nevertheless, their cardiac output can respond to volume resuscitation due to the heightened cardiac filling pressure in obstructive shock. Rapid recognition and treatment of the obstructive shock are crucial for the outcome, and if blood pressure does not improve promptly, vasopressors can be empirically administered.⁶

This report aimed to present a case of obstructive shock in a patient with a significant accumulation of blood in the left pleural cavity (massive left hemothorax) who underwent urgent pleural drainage. It detailed the role of promptly performing emergency pleural drainage in addressing this type of shock, and the substantial accumulation of blood on the left side will be described.

CASE

The data were collected using the patient's medical records and a direct examination. Reported a case of a 56-year-old male who arrived at a local hospital after experiencing symptoms such as shortness of breath, swollen legs, a bloated abdomen, coughing up blood, and overall fatigue for two months. The patient denied any weight loss, chest pain, or night

sweats. The patient had no history of previous disease or trauma.

Upon the patient's admission, the results of the routine blood tests were as follows: Hemoglobin level was 11.9 g/dL (within the normal range of 13.0-18.0), white blood cell count was 13,600/mm³ (normal range 4000-11,000), platelet count was 365,000/ μ L (normal range 150,000-450,000), neutrophils accounted for 74.8% (normal range 50-70%), lymphocytes were at 8.92% (normal range 20-40%), creatinine level was 0.9 mg/dL (normal range 0.5-1.3), and urea level was 89 mg/dL (normal range 10-60). No serological indications of hepatitis B or hepatitis C were found.

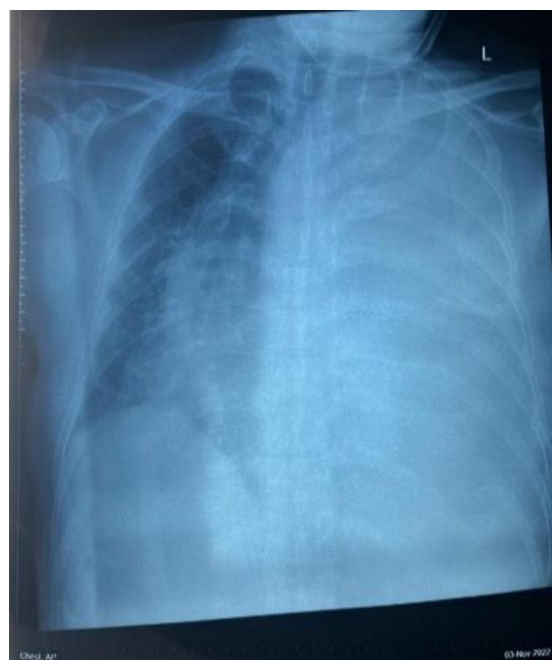


Figure 1. The anterior chest radiograph demonstrates a large left-sided pleural effusion extending into the left upper zone, causing contralateral tracheal deviation and mediastinal shift.

The chest X-ray (CXR) revealed a significant accumulation of fluid in the left pleural area that extended into the upper

portion of the left lung (Figure 1). Fluid accumulation caused a right-sided tracheal and mediastinal shift.

The results of the patient's physical examination were as follows: blood pressure (BP): 90/60 mmHg, heart rate (HR): 98x/min, respiratory rate (RR): 36x/min, temperature (T): 36.5°C, and SpO₂: 92%. Further physical examination showed chest wall expansion, abdominal breathing, face, and neck swelling, discoloration, and distended veins. Percussion on the left side of the chest indicated a dull sound, and auscultation revealed significantly reduced airflow at the lower part of the left lung. The patient's heart sounds were within the normal range, and he also exhibited leg swelling.

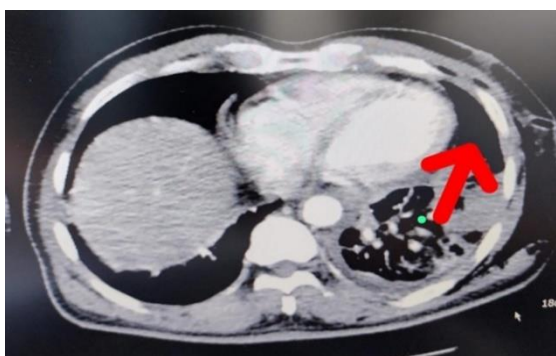


Figure 2. CT pulmonary angiogram in axial soft tissue windowing. Arrows indicate the presence of fluid. There was a left hydropneumothorax and atelectasis of the left lung, accompanied by a multifocal picture of diffuse mixed ground glass opacities (GGO) consolidation of the left lung, suspected pneumonia, and pulmonary tuberculosis (TB).

After being transferred to a more comprehensive medical facility, a multislice spiral computed tomography (MSCT) pulmonary scan was conducted. The MSCT scan showed multiple areas of diffuse, hazy lung opacity on the left side, indicating suspicion of tuberculosis and pneumonia.

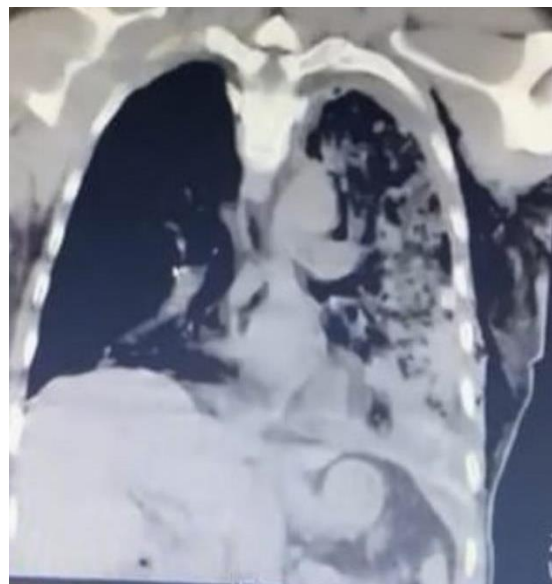


Figure 3. CT pulmonary angiogram in the coronal plane. A chest tube was installed in the left anterolateral 5-6th intracostal with a tip in the left pleural cavity.

An urgent MSCT pulmonary scan was requested to assess the characteristics and extent of the pleural effusion before proceeding with any interventions. The aim was also to rule out the possibility of a pulmonary embolism, as shown in Figures 2, 3, and 4.

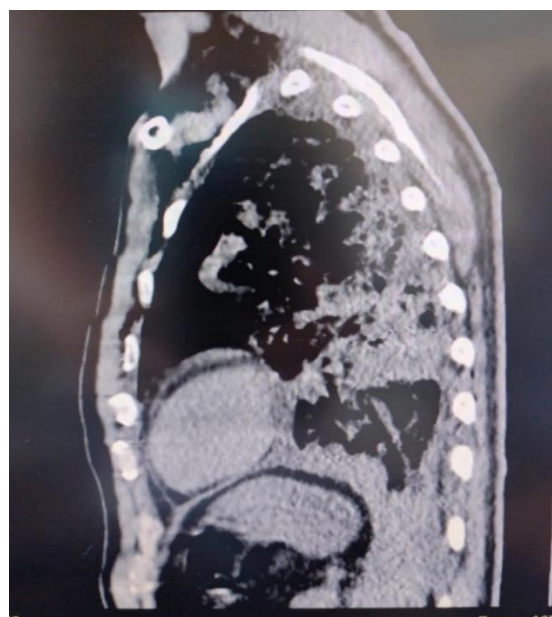


Figure 4. CT pulmonary angiogram in the sagittal plane. Subcutaneous emphysema was seen in the subcutaneous left anterolateral hemithorax.

Additionally, an echocardiogram revealed a considerable left-sided pleural effusion that led to the displacement of the left atrium and left ventricle, ultimately resulting in obstructive shock. The pleural fluid analysis was not performed on this patient.

Upon arrival, the patient displayed significant hemodynamic instability. Immediate measures involved administering oxygen through a non-rebreathing mask (NRBM) to achieve a target oxygen saturation level above 96%.

Additionally, the patient required two vasopressors (dopamine at a rate of 2.5 mcg/kg per minute of body weight and norepinephrine at a rate of 0.1 mcg/kg per minute of body weight) to sustain a blood pressure reading of 90/60 mmHg. A Foley catheter was inserted to monitor urine output. Given the patient's unstable hemodynamic condition and imminent death threat, urgent intervention was deemed necessary to alleviate the obstructive shock. A surgical consultation was conducted over the phone.

Thoracentesis (pleural drainage) was considered a potential therapeutic approach. This procedure involves removing more significant quantities of pleural fluid to relieve breathing difficulties caused by substantial pleural effusion. For individuals with new pleural effusions, a diagnostic thoracentesis is usually recommended.⁸

In the surgical theatre, a chest tube was placed on the left side to address the extensive accumulation of blood within the chest cavity. This procedure resulted in the

drainage of approximately 1.5 liters of blood. The patient was given additional oxygen support, antibiotics, and furosemide. As a general recommendation, antibiotics from the quinolone generation are often preferred.



Figure 5. AP erect chest radiograph following left-sided thoracentesis. This demonstrates almost complete resolution of the left-sided pleural effusion, with minimal residual fluid within the left costophrenic recess.

A subsequent chest X-ray (depicted in Figure 5) conducted the next day indicated significant improvement in the left pleural effusion, almost reaching full resolution after removing the chest tube. The initial mediastinal shift had also been resolved. With the patient's condition stabilizing, the patient was transferred to a more comprehensive medical facility for a CT pulmonary angiogram.

DISCUSSION

The significance of urgent intervention in cases of obstructive shock occurring in patients with a substantial left-

sided pleural effusion. A pleural effusion, marked by an atypical fluid buildup in the pleural space caused by excessive production of fluid or compromised absorption, is commonly seen in pleural disorders.

Following tuberculosis (TB) treatment, the patient's condition improved notably. The initial rapid breathing rate of 30 breaths per minute has now returned to the normal range of 20-24 breaths per minute, and the initial body weight of 45 kg has increased to 52 kg.

A growing amount of fluid accumulating within the pleural cavity can decrease lung capacity, contributing to rapid breathing and lowered oxygen levels. If left unaddressed, the rising pressure within the chest could lead to diminished venous blood flow and compression of the right ventricle, presenting as low blood pressure.^{3,9} In regions where TB is prevalent, the pleural variant emerges as the primary origin of pleural effusion. This statement underscores the importance of considering this diagnosis for all individuals experiencing pleuritic symptoms of unknown origin.¹⁰

Hemorrhaging into the pleural cavity can occur due to various disturbances affecting the chest wall tissues, pleura, or structures within the chest. The physiological reaction to the occurrence of a hemothorax becomes evident in two significant domains: hemodynamic and respiration. The extent of the hemodynamic reaction is contingent upon the volume and speed of blood loss.¹¹

Hemodynamic alterations differ based on the bleeding volume and the speed at which blood loss occurs. A blood loss of up to 750 mL in an individual weighing 70 kg typically does not lead to noteworthy hemodynamic shifts. In the same person, a loss of 750-1500 mL results in the initial signs of shock, including rapid heart rate, accelerated breathing, and a decrease in pulse pressure.¹²

Signs of shock accompanied by inadequate tissue perfusion arise with a blood volume loss of 30% or more (approximately 1500-2000 mL). Given that the pleural cavity in a 70-kg individual can accommodate 4 L or more of blood, a severe hemorrhage can transpire without visible external evidence of blood loss.¹³

A substantial buildup of blood in the pleural cavity can impede the regular movement involved in breathing. In trauma, this can lead to disruptions in ventilation and the intake of oxygen, particularly when coupled with injuries to the chest wall.^{8,14}

When a considerable amount of blood accumulates, individuals often encounter difficulty breathing, leading to the observation of rapid breathing, known as tachypnea. The quantity of blood needed to evoke these symptoms varies according to multiple factors, such as the specific organs affected, the extent of the injury, and the individual's existing pulmonary and cardiac capacity.¹⁴

Distinguishing a hemothorax from other effusions containing blood can be achieved by conducting a pleural fluid hematocrit test. A pleural fluid hematocrit

value exceeding 50% of the patient's peripheral blood hematocrit indicates a hemothorax.¹¹

In both cases of cardiogenic and obstructive shock, patients usually exhibit indications of reduced cardiac output, such as cool extremities and weak pulses. They also display symptoms indicating elevated filling pressures in the LV or the RV, such as pulmonary edema, peripheral edema, and distended jugular veins.^{15,16}

Within the unaffected and unscarred pleural space, a hemothorax is observed as a fluid meniscus obscuring the costophrenic angle or the surface of the diaphragm. This fluid also follows the pleural margins along the chest wall when visualized on an upright CXR. This radiographic presentation is analogous to what is seen with any other type of pleural effusion.^{14,17}

Once a pleural effusion is confirmed, the underlying cause should be determined. Approaches for diagnosis encompass percutaneous pleural biopsy, bronchoscopy, thoracoscopy, and open pleural biopsy. Nevertheless, in the Emergency Department (ED), thoracentesis and chest tube insertion are usually adequate for assessment and treatment.⁸

CONCLUSION

The subsequent pleural drainage effectively removed larger quantities of pleural fluid, alleviating the breathing difficulties associated with a sizable pleural effusion.

REFERENCES

1. Ota K, Fumimoto S, Iida R, Kataoka T, Ota K, Taniguchi K, et al. Massive hemothorax due to two bleeding sources with minor injury mechanism: a case report. *J Med Case Rep.* 2018;12:291.
2. Huggins J. Hemorrhagic Pleural Effusions and Hemothorax [Internet]. *Cancer Therapy Advisor.* 2019 [cited 2023 Nov 2]. Available from: <https://www.cancertherapyadvisor.com/home/decision-support-in-medicine/pulmonary-medicine/hemorrhagic-pleural-effusions-and-hemothorax/>
3. Jahir T, Hossain S, Dolkar T, Patel MJ, Risal R, Khan A, et al. A Case of Bilateral Hemorrhagic Pleural Effusion Due to Dual Antiplatelet Therapy in a Dialysis Patient. *Cureus.* 2022;14(4):e24450.
4. Aramaki H, Fukaguchi K, Yamagami H, Maeda Y, Tsunoda S, Akazawa K, et al. Obstructive shock due to a spontaneous haemothorax caused by primary lung cancer. *Respirol Case Rep.* 2023;11(2):e01082.
5. Gomez LP, Tran VH. *Hemothorax.* Treasure Island (FL): StatPearls Publishing; 2024.
6. Kim KS. Obstructive Shock. In: Suh GJ, editor. *Essentials of Shock Management.* Singapore: Springer; 2018.
7. Koya HH, Paul M. *Shock.* Treasure Island (FL): StatPearls Publishing; 2023.

8. Watkins CL. Emergent Management of Pleural Effusion [Internet]. Medscape. Medscape; 2023 [cited 2023 Nov 2]. Available from: <https://emedicine.medscape.com/article/807375-overview>
9. Malek R, Soufi S. Pulmonary Edema. Treasure Island (FL): StatPearls Publishing; 2023.
10. Butt MF, Symonds M, Khurram R. Tension hydrothorax in a patient with SARS-CoV-2 pneumonitis and pleural Mycobacterium tuberculosis. *BMJ Case Rep.* 2021;14(7):e243760.
11. Antonangelo L, Faria CS, Sales RK. Tuberculous pleural effusion: diagnosis & management. *Expert Rev Respir Med.* 2019;13(8):747–59.
12. Hooper N, Armstrong TJ. Hemorrhagic Shock. Treasure Island (FL): StatPearls Publishing; 2022.
13. Poole DC, Pittman RN, Musch TI, Østergaard L. August Krogh's theory of muscle microvascular control and oxygen delivery: a paradigm shift based on new data. *J Physiol.* 2020;598(20):4473–507.
14. Mancini MC. Hemothorax [Internet]. Medscape. Medscape; 2020 [cited 2023 Nov 2]. Available from: <https://emedicine.medscape.com/article/2047916-treatment?form=fpf>
15. Sugaya A, Funakoshi H, Mizobe M, Hara R, Kotani S, Kubota T. Left atrial appendage rupture: A rare complication of right-sided haemothorax: A case report. *Trauma Case Rep.* 2022;40:100667.
16. Parlow S, Cheung M, Verreault-Julien L, Wu KY, Berardi P, Nair V, et al. An Unusual Case of Obstructive Shock. *JACC Case Rep.* 2021;3(18):1913–7.
17. Garg SK, Malik A. Spontaneous hemothorax and implication of diagnostic evaluation: A case report. *Respir Med Case Rep.* 2022;36:101604.