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Lung: Case Report

# Cardiac Tamponade After Video-Assisted Thoracoscopy Surgical Diaphragmatic Plication

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Video-assisted thoracoscopy surgical diaphragmatic plication is the standard of care for diaphragmatic eventration. However, it is associated with complications like injuries to the bowel, liver, spleen, and lung parenchyma. We report life-threatening cardiac tamponade after Video-assisted thoracoscopy surgical diaphragmatic plication. The mechanisms contributing to the injury are described as well.

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Video-assisted thoracoscopic surgery (VATS) is a preferred surgical modality for treating thoracic surgical lesions due to its small incision, limited blood loss, and quick recovery.[1] The reported complications related to VATS diaphragm plication are injuries to the structures lying underneath the diaphragm.[2] We describe the mechanism of injury to the left ventricle causing cardiac tamponade during VATS diaphragmatic plication. Written informed consent was obtained from the patient for publication of this case report, and the article adheres to the applicable EQUATOR (Enhancing the QUAlity and Transparency Of health Research) guidelines.[3]

A 56-year-old patient weighing 93 kg presented with shortness of breath and dyspepsia. The chest radiograph of the patient showed eventration of the left hemidiaphragm (Figure 1). The patient was scheduled for VATS diaphragmatic plication.

In the operating room, anesthesia induction and placement of a left-sided 39F double-lumen tube (Mallinckrodt Medical) were uneventful. Bupivacaine 0.25% was administered for pain relief through a catheter placed in the epidural space. Subsequently, the patient was positioned in the left-lateral position, and one-lung ventilation (OLV) was initiated. Using a standard 3-port system, diaphragm plication was performed with multiple interrupted mattress sutures with polypropylene and absorbable pledgets. The procedure was uneventful, and the patient was extubated and transferred to the intensive care unit for observation. The intercostal drains were removed on postoperative day 2. A repeat chest radiograph did not show any signs of pleural collection or cardiomegaly and the patient was transferred to the ward facility. On postoperative day 3, the patient returned to the intensive care unit with restlessness, tachypnea, desaturation, and cold peripheries; the heart rate and blood pressure were 120/min and 75/43 mm Hg, arterial blood gas showed pH 7.22, $PCO_2$ 23 mm Hg, $PO_2$ 45 mm Hg, and lactates 5.6 mmol/L. The trachea was intubated, and resuscitation started with 500 mL of fluid challenge, 0.04-0.06 μg/kg/min noradrenaline, and 0.04 μg/kg/min adrenaline. The patient's Wells score was 6, indicating a moderate probability of acute pulmonary embolism; therefore, an emergency computed tomography pulmonary angiogram was planned, and a decision was taken to start the patient on heparin therapy. Simultaneously, transthoracic echocardiography (TTE) was performed to assess the right ventricular function and to visualize any mobile thrombi in the right atrium, but no thrombus was found. However, a circumferential pericardial effusion was noted, but the image was poor. Therefore, a transesophageal echocardiography probe (X7-2t; Philips Healthcare) was inserted to quantify pericardial effusion and assist in bedside drainage. A circumferential pericardial effusion with a maximal thickness of 18 mm with a diastolic right atrium and right ventricular collapse was noted. Approximately, 500 mL of blood with clots was drained at the bedside using a subxiphoid incision, and the patient's hemodynamics improved. After 15 minutes, the hemodynamic parameters started to deteriorate, and a repeat transesophageal echocardiography examination showed notable pericardial collection. The patient was

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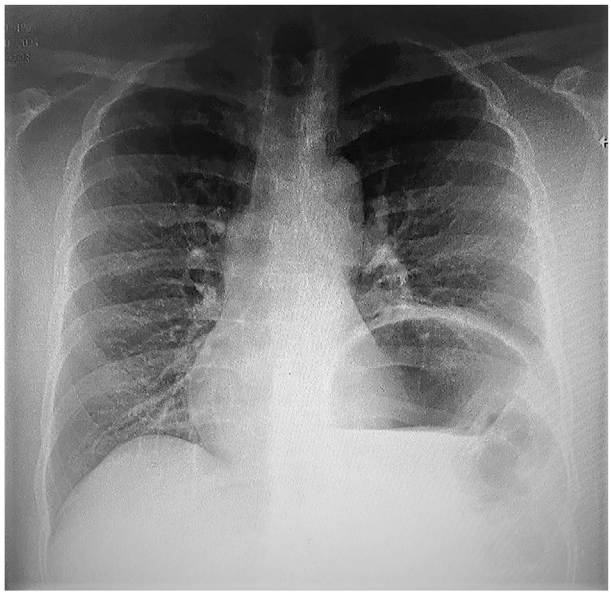

**FIGURE 1** Chest radiograph demonstrates eventrated left hemidiaphragm.

transferred to the operating room to explore the site of bleeding. On sternotomy, a small myocardial tear was noted on the inferolateral surface of the left ventricle near the obtuse marginal artery (Figure 2), and a needle mark was seen in the adjacent pericardium. The damaged myocardium was repaired with a 6-0 Prolene suture (J&J MedTech); the subsequent perioperative period remained uneventful.

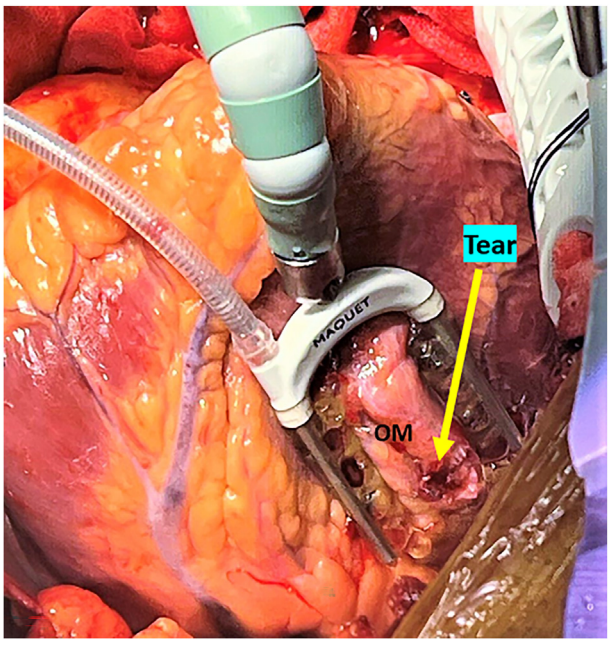


**FIGURE 2** Heart in vertical position shows myocardial tear (yellow arrow) near obtuse marginal artery (OM).

## COMMENT

The goal of the VATS plication surgery is to flatten the diaphragm and to promote expansion of the ipsilateral lung.[4] Any diaphragmatic surgery carries the risk of injury to the bowel and surrounding organs like the liver, stomach, and spleen due to their close proximity. Pulmonary thrombosis is a well-known complication after thoracic surgery.[5] The present patient was obese with a Wells score of 6, suggesting a moderate possibility of acute pulmonary embolism.[6] The Wells scoring evaluates the possibility of acute pulmonary embolism and is based on: clinical features and past history including symptoms of DVT (3 points), no alternative diagnosis that better explains the illness (3 points), tachycardia with pulse >100 (1.5 points), immobilization (≥3 days) or surgery in the previous 4 weeks (1.5 points), prior history of DVT or pulmonary embolism (1.5 points), presence of hemoptysis (1 point), and presence of malignancy (1 point). A score of <2 indicates low risk, a score of 2-6 indicates moderate risk, and a score of >6 indicates high risk of acute pulmonary embolism. In view of moderate risk of pulmonary embolism, heparin therapy was considered. Simultaneously, a TTE examination was performed to assess the right ventricular function, and to visualize any mobile thrombi in the right atrium, but no thrombus was found.

Cardiac tamponade after VATS diaphragm plication is not described. Therefore, it is usually missed and is considered only after excluding other causes. The thickness of the eventrated diaphragm decreases after administration of the muscle relaxants, thereby increasing the risk of injury to the surrounding structures. The role of OLV is interesting; the surgical lung collapses around the bronchus during OLV and the diaphragm ascends higher in the thorax because of its redundancy and paralysis (Figure 3). We believe the collapse of the lung, and the ascent of the diaphragm brought the inferolateral wall of the LV and the diaphragm in close apposition rendering myocardium vulnerable to injury during diaphragm plication. Additionally, the absence of any pericardial fat on the inferolateral wall of the left ventricle can further increase the possibility of myocardial injury. The diagnosis and immediate treatment of the cardiac tamponade are paramount in preventing unfavorable outcomes. Köckerling and colleagues[7] reported a mortality rate of 48% in patients who developed cardiac tamponade after laparoscopic hernia surgery. In the present case, the cause for initial presentation to the intensive care unit was thought to

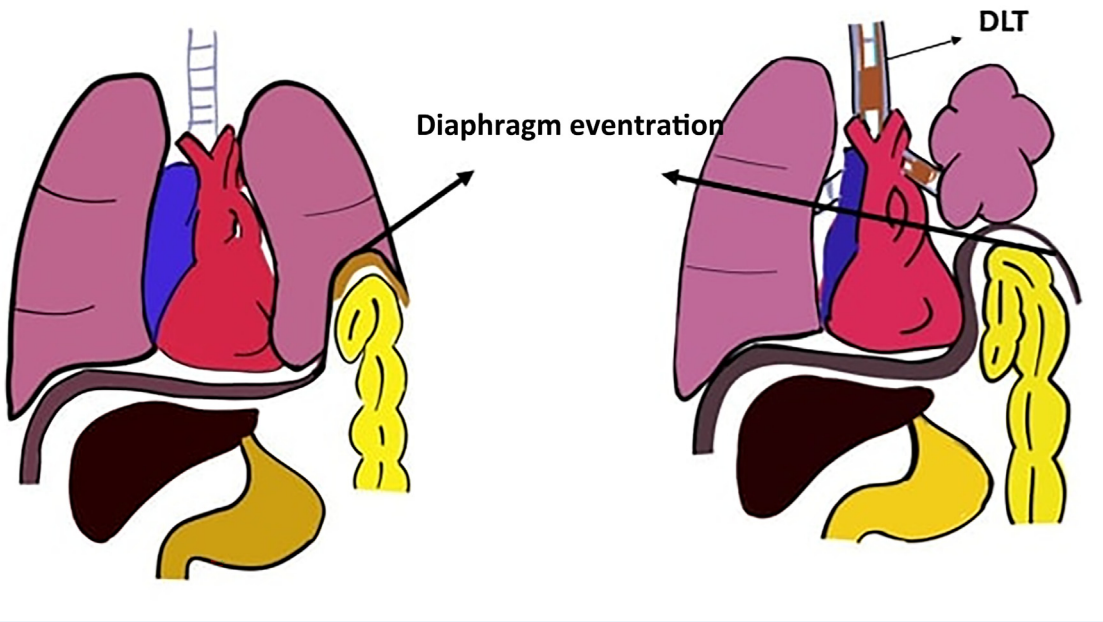


**FIGURE 3** Schematic diagram shows the relationship between the diaphragm, left lung, and heart in the preoperative period (left panel), and after one-lung ventilation (right panel). (DLT, double-lumen tube.)

be acute pulmonary embolism; however, it was only after the point of care ultrasound examination that the exact diagnosis was made.

To conclude, we describe cardiac tamponade after VATS diaphragmatic plication and the importance of point of care ultrasound in diagnosing the underlying pathology in the presence of acute respiratory failure with acute hemodynamic collapse. The effect of OLV on the expected change in the relationship of the diaphragm with the left ventricle needs to be considered while plicating the diaphragm.

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## DISCLOSURES

The authors have no conflicts of interest to disclose.

## PATIENT CONSENT

Obtained.

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