(Acta Anaesth. Belg., 2021, 72, 187-190)

Unusual cause of obstructive shock following esophagectomy: a case report

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Abstract: Obstructive shock usually has an intrathoracic origin, such as pneumothorax, pericardial tamponade or pulmonary embolism. We report a case of hemodynamic shock in a 74-year-old patient four days after esophagectomy, just after the start of mechanical ventilation for bilateral pneumonia. The sudden onset of severe abdominal distension and the presence of air in the intra-abdominal drain suggested tension pneumoperitoneum, confirmed by radiography. No pneumothorax was associated. Urgent decompression was required to improve hemodynamics. Perforation of the gastrointestinal tract was ruled out. The cause was a bronchopleural fistula opened by mechanical ventilation. Rarely, cardiorespiratory failure may occur after tension pneumoperitoneum by reducing lung volume and cardiac preload, similar to obstructive shock from the usual intrathoracic causes or acting as an abdominal compartment syndrome (ACS). Its recognition and abdominal decompression are key steps in the patient's recovery. Tension pneumoperitoneum related to mechanical ventilation and airway injury without associated pneumothorax is exceptional and, to our knowledge, has never been reported as a postoperative complication of esophagectomy.

Keywords: obstructive shock, tension pneumoperitoneum, mechanical ventilation, esophagectomy

Introduction

Obstructive shock is primarily due to intrathoracic and extra-cardiac causes of cardiac pump failure and is often associated with poor right ventricular output. Its main causes are pulmonary embolism, tension pneumothorax, and pericardial tamponade. We report a case of obstructive shock of unusual etiology following esophagectomy.

Case Report

A 74-year-old patient with a history of large bilateral epiphrenic diverticula associated with dyskinesia developed squamous cell carcinoma in a left diverticulum (cT3N2). He was treated preoperatively by radiochemotherapy followed by Lewis-Santy esophagectomy (laparoscopy and right thoracotomy). The only difficulties encountered during the procedure were significant inflammatory adhesions between the left esophageal diverticulum and the left pleura and lung. A suture was performed on the left lung after visualization of a micro air leak after dissection. Insertion of a left chest tube failed due to multiple pleural adhesions. The ventilatory mode during the procedure was volume-controlled ventilation. A unipulmonary ventilation was applied in the left lung via a double-lumen endotracheal tube during the right thoracotomy to allow access to the mediastinal cavity and esophageal dissection. Extubation was uncomplicated. The patient was admitted to the intensive care unit for the postoperative course. He required tracheal intubation on the fourth postoperative day, due to respiratory failure from bilateral pneumonia. Within 1 hour, inspiratory pressures increased to 45 cm H_2O and tidal volume decreased to 300 ml, saturation decreased to 79% despite an inspired fraction of 100% oxygen, heart rate increased to 165/min, and systolic hypotension to 65 mmHg and livedo reticularis of the lower extremeties were observed despite extensive vasopressor support. At the same time, abdominal distension was observed and intra-abdominal pressure was measured at 23 mmHg via a urinary catheter. The bag connected to the abdominal drain was filled with air. Bronchoscopy and chest radiography were quickly performed and

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Paper submitted on Jan 23, 2021 and accepted on Oct 01, 2021.
Conflict of interest: None.

showed a correct position of the endotracheal tube with no obvious proximal tracheobronchial lesion and the presence of significant pneumoperitoneum with no evidence of pneumothorax (Fig. 1). Rapid abdominal decompression by opening the abdominal drain and - reopening one of the laparoscopic ports resulted in rapid improvement in hemodynamic and ventilation parameters: systolic blood pressure rose above 100 mmHg, heart rate fell to 120/min, livedo reticularis of the lower extremities disappeared, ventilation pressures fell below 35 cmH_2O and oxygen saturation reached 95%. Thoracoabdominal CT scan, performed before urgent surgery, revealed a large and persistent pneumoperitoneum, with a small pneumomediastinum but no pneumothorax (Fig. 2) Esogastric perforation was suspected. The bag connected to the abdominal drain had to be opened regularly during transfer to the operating room to allow for an air leakage. Laparotomy revealed no digestive perforation. Intraoperative gastroscopy and bronchoscopy revealed no anastomotic leakage or proximal tracheobronchial lesion. A large mediastinal air leakage through the hiatal orifice was noted. The leak was estimated at two liters per minute, suggesting a direct link with mechanical ventilation and a distal bronchopleural fistula. Due to diffuse pleural adhesions, pulmonary examination could not be performed, an epiploplasty to cover the pulmonary defect was performed, and a 32Fr trans-hiatal drain was positioned in the mediastinal cavity. The postoperative course was complicated by a severe acute respiratory distress syndrome (ARDS) that required mechanical ventilation for several weeks and high-dose corticosteroid therapy contributing to the persistent air leak . Finally, the patient died on day 33 due to respiratory and infectious complications

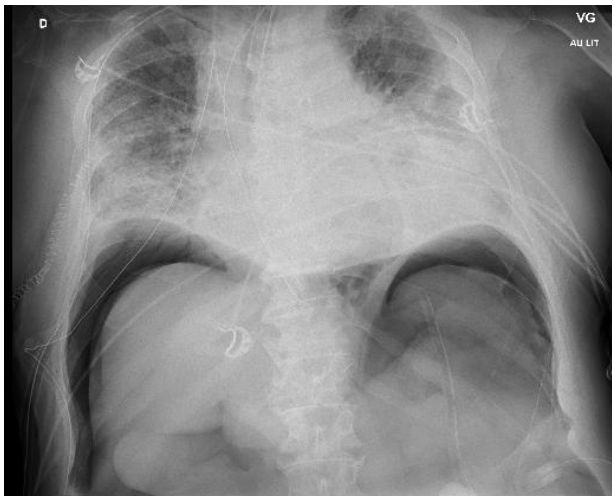

Fig. 1 — Pre-decompression X-ray of the chest and abdomen, showing a large pneumoperitoneum, with no evidence of pneumothorax or pneumomediastinum. Elevation of the diaphragmatic cupolas and significant reduction in lung volumes suggest intra-abdominal hypertension.

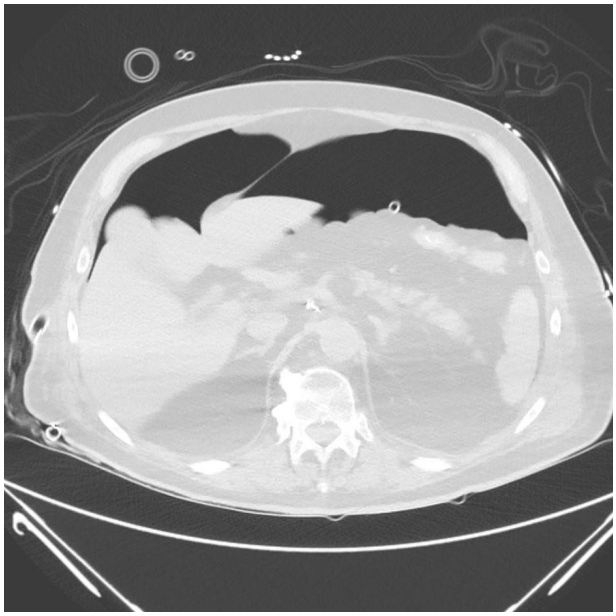

Fig. 2 — Abdominal CT scan performed after abdominal decompression, but before surgery, showing persistence of a large pneumoperitoneum.

Discussion

We report an unusual case of obstructive shock secondary to tension pneumoperitoneum following the initiation of positive pressure ventilation by mechanical ventilation occurring after esophageal surgery. The causes of obstructive shock for the right heart can be divided into two categories: pulmonary vascular and mechanical. In the former, the right ventricle is unable to generate sufficient pressure to overcome the high pulmonary vascular resistance associated with pulmonary embolism or pulmonary hypertension. In the second, shock is related to decreased preload, rather than pump failure (e.g., reduced venous return to the right atrium or insufficient filling of the right ventricle) and thus mimics severe hypovolemia. Mechanical causes of obstructive shock include tension pneumothorax and pericardial tamponade (1, 2). In this case, a massive accumulation of air in the abdominal cavity caused sudden intra-abdominal hypertension (IAH) measured at 23 mmHg with consequent elevation of the diaphragmatic cupolas with a reduced lung volumes and increased in intrathoracic pressures and compression of the inferior vena cava (3). These phenomena reduce preload and thus right cardiac output, as in the mechanical causes of obstructive shock mentioned above. According to

the World Society of the Abdominal Compartment Syndrome (WSACS) definition criteria (4), the combination of intra-abdominal hypertension greater than 20 mmHg and new organ failure, due to tension pneumoperitoneum, is a form of abdominal compartment syndrome (ACS). It is urgent to remove the mechanical obstacle to venous return in order to reverse the shock, as we have done by decompression of the abdominal cavity with air evacuation. It should be noted that after stabilization of the hemodynamic and respiratory parameters, diagnostic procedures will be performed to identify the cause.

Surgical exploration of the abdomen is recommended in case of suspected digestive perforation. This remains the most frequent cause of pneumoperitoneum (5). Several cases of tension pneumoperitoneum have been reported, with mainly digestive etiologies and various modes of presentation (6-8). Acute massive pneumoperitoneum, as described in this case, is a very unusual manifestation. Although our patient developed symptoms four days after esophagectomy, suggesting digestive perforation, a link to mechanical ventilation was evident. Esophageal intubation should be suspected and was quickly ruled out by bronchoscopy. Surgical exploration ruled out digestive perforation or anastomotic leak, but revealed an airway injury.

The absence of associated pneumothorax is an additional feature of our case. To our knowledge, in the last 25 years, only six cases of tension pneumoperitoneum involving mechanical ventilation with airway injury and without pneumothorax have been reported in the literature (9-14). Ventilation difficulties were reported in all patients, but hemodynamic instability was reported in 4 of them. Three were transferred to the operating room without prior decompression, suggesting that the hemodynamic instability was not as precarious as in our patient. Of these 6 cases, 3 were attributed to barotrauma, 1 to a bronchoperitoneal fistula due to a pleuroperitoneal shunt, 1 to posterior tracheal wall tear following percutaneous tracheostomy and 1 to rupture of a main bronchus during a bi-pulmonary transplantation. In the latter two cases, the mechanism of air passage to the abdominal cavity appears to be similar to that described in our patient, through the esophageal hiatus. Dissection during esophageal surgery favors the passage of air to the abdomen, a place of less resistance compared to the mediastinal cavity, being a more rigid anatomical space. The hypothesis of a very peripheral left bronchopleural fistula, occurring during the difficult dissection and located on the mediastinal side of the left lung, was therefore retained. The absence of pneumothorax is explained by the presence of numerous adhesions between the pleural layers, as described during the initial surgery. The literature also reports a few cases of tension pneumoperitoneum occurring after cardiopulmonary resuscitation. Gastric rupture is found in most of them, but rare cases have been described without evidence of digestive perforation (15).

Esophagectomy performed according to the Lewis Santy technique and characterized by a double approach is one of the most widely used surgical techniques in the treatment of lower third esophageal carcinoma (16). Minimally invasive approaches are increasingly preferred to reduce the morbidity and mortality of esophageal surgery. Nevertheless, it remains a high-risk procedure and complications still occur in 50-75% of cases (17). These complications include, in order of frequency, respiratory complications, anastomotic leaks or suture loosening, hemorrhagic or cardiac complications, chylothorax and recurrent laryngeal nerve damage (17, 18). Tension pneumoperitoneum with obstructive shock has, to our knowledge, never been reported as an immediate postoperative complication of esophageal surgery.

Conclusion

The sudden onset of abdominal distension associated with increased chest pressure and shock after the introduction of mechanical ventilation should suggest the presence of tension pneumoperitoneum. Urgent abdominal decompression is necessary to avoid a fatal outcome. In the post-esophagectomy period, in addition to digestive etiologies, a pulmonary origin may be present and should therefore be investigated.

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