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Perioperative Considerations for Chylothorax

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Case Presentation

A 73-year-old male with a past medical history of hypertension, type-2 diabetes mellitus, obesity, and gout was transferred to our facility with a two-month history of increasing dyspnea, recurrent right pleural effusions, and a mediastinal mass of indeterminate etiology after a computer tomography-guided biopsy and thoracentesis. Though previously active, he had experienced decreased exercise tolerance, manifested by an inability to walk five steps without stopping to catch his breath. He also reported orthopnea, cough, peripheral edema, weight loss, and fatigue. A few days before his admission he underwent thoracentesis with two liters of fluid removed from his right pleural space at an outside hospital. At our facility a chest X-ray revealed a large right pleural effusion. Computed tomography demonstrated profound lymphadenopathy involving the left thoracic inlet, mediastinum and bilateral hila, a large right pleural effusion, bilateral lung nodules, and a small pericardial effusion (Figure 1). An electrocardiogram showed sinus rhythm. He was taken to the operating room for video-assisted thoracoscopic surgical mediastinal biopsy and pleural biopsy to investigate the etiology of his condition.

Pre-operative examination revealed decreased breath sounds over the right lung fields and bilateral wheezing. His breathing was non-labored, oxygen saturation was 94% on 5 liters

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per minute of nasal cannula oxygen. His heart rate was 80-90 beats per minute and regular and no jugular venous distention was detected. In the pre-operative holding area the anesthesia team performed a transthoracic echocardiogram confirming a small pericardial effusion without evidence of impaired right ventricular diastolic filling. The left ventricle did not appear underfilled. A pre-induction radial arterial catheter was placed in addition to standard monitors. Pulsus paradoxus was not appreciated on palpation or upon analysis of the arterial pressure waveform. While attempting to move from the stretcher to the operating table, the patient manifested increasing dyspnea with decreasing oxygen saturation. Five hundred ml of isotonic crystalloid solution were administered prior to induction of anesthesia with 90mg ketamine, 100mcg fentanyl, and 100mg succinylcholine in the sitting position due to the patient's inability to tolerate the supine position. Following direct laryngoscopy, a 39 French left-sided double lumen endobronchial tube was placed to permit lung isolation. Mechanical ventilation was instituted using a pressure-controlled volume guaranteed mode (tidal volume 400ml, PEEP 4 cmH₂O). Hypotension necessitated treatment with intravenous epinephrine and additional intravenous crystalloid fluid to stabilize blood pressure. No additional interventions were required to maintain the patient's hemodynamic status, and he was then positioned for surgery.

Video-assisted thoracoscopic surgery demonstrated creamy opaque fluid in the right pleural space (Figure 2), of which four liters were evacuated. After being placed in a specimen container this fluid rapidly solidified once it reached room temperature (Figure 3). To confirm our suspicion of chyle, the sample was tested for triglycerides, which was 1522 mg/dL (normal less than 110mg/dL). The anterior mediastinal mass was biopsied for frozen pathologic evaluation, demonstrating malignant etiology. A right-sided 28 French pleural chest tube was placed for continued chylothorax drainage. The trachea was extubated without complication and he had an uneventful recovery in the post-anesthesia care unit.

Post-operatively, flow cytometry and permanent pathologic evaluation were consistent with primary mediastinal large B-cell lymphoma (PMBL). With associated lymphadenopathy likely obstructing lymphatic flow, the patient was made nil per os (NPO), started on total parenteral nutrition. Care was transitioned to the

Hematology/Oncology service, and therapy was initiated with etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin, and rituximab (EPOCH-R). He subsequently underwent thoracic duct embolization by interventional radiology to seal the chyle leak with minimal disruption to his chemotherapy regimen. The patient was ultimately discharged from the hospital, continuing chemotherapeutic treatment.

Case Discussion

Pleural effusions cause dyspnea and decreased arterial oxygenation.¹ Thoracentesis can relieve dyspnea,² however has inconsistent effects on oxygen saturation.³⁻⁵ In the presence of a pleural effusion, intrapulmonary shunt induces decreased oxygen saturation.^{6, 7} Progression from hypoxemia to hemodynamic instability may be rapid. Pericardial effusions gradually accumulate fluid within the pericardium increasing intrapericardial pressure. Compensatory sympathetic activation in the form of tachycardia, pericardial stretch, and

increased myocardial contractility progressively fail as pressure equalizes within intracardiac chambers. During this progression, right ventricular dilation increases myocardial oxygen consumption,⁸ decreases compliance and cardiac output,⁹ and can cause interventricular septal bowing, reducing left ventricular stroke volume. Continued progression of a large pericardial effusion can subsequently lead to cardiac tamponade and hemodynamic compromise.¹⁰

Experimental evidence¹¹ and case reports¹²⁻¹⁵ have demonstrated a link between occurrence of pleural effusions and subsequent development of cardiac tamponade. Increased intrapleural pressure transmitted to the pericardial space may contribute to increased pericardial pressure and can induce tamponade-like physiology.¹² While our patient had a small pericardial effusion before induction, no pulsus paradoxus was detected on examination, and bedside transthoracic echocardiography failed to demonstrate right ventricular diastolic collapse. However, after induction of anesthesia, our patient now presented pulsus paradoxus and experienced rapid hemodynamic instability. Despite pre-induction administration of 500ml intravenous crystalloid solution, it was necessary to give inotropic medication to temporarily stabilize the patient's hemodynamics while further intravenous crystalloid was administered and the pleural effusion was drained. It is possible that the transition from negative to positive pressure ventilation, increasing mean airway pressure and decreasing venous return to the right ventricle altered pericardial pressure, transitioning to tamponade-like physiology. Though our patient had his pleural effusion drained a few days prior, the rapid re-accumulation of fluid likely worsened his hypoxemia and hemodynamic instability with induction of anesthesia. Case reports have shown that drainage of pleural effusions before the drainage of a pericardial effusion improves patient hemodynamics.¹²

Patients with large pleural effusions, particularly in the presence of pericardial effusions, can benefit from pre-operative pleural drainage to alleviate hypoxemia and hemodynamic instability associated with induction of anesthesia. Providers should be prepared for emergent drainage of pleural effusions before or immediately after induction of anesthesia. Arterial blood pressure monitoring should be in place before induction of anesthesia to accurately quantify pulsus paradoxus. Further evaluation of pericardial effusions before induction should be performed with echocardiography to evaluate for right ventricular diastolic collapse. Hypoxemia can be present in patients with a pleural effusion, and one should optimize oxygen delivery via adequate pre-oxygenation, patient positioning, hemoglobin concentration, and possibly thoracentesis. In this case the patient's hemodynamic status stabilized after epinephrine, additional crystalloid, and eventual drainage of the pleural effusion. Although there was no compressive effect on the pericardium or great vessels detected on pre-operative imaging, further considerations for potential adverse hemodynamic effects stemming from the mediastinal mass would have been made, had the administration of epinephrine, volume, and the eventual evacuation of the pleural effusion not resulted in hemodynamic stability.

PMBL is a unique subtype of diffuse large B-cell lymphoma (DLBCL). It accounts for 2–4% of all non-Hodgkin lymphomas and 6–12% of all DLBCLs.¹⁶⁻¹⁸ PMBL is characterized by distinct clinical, pathologic, and genetic features. It typically involves the mediastinum,¹⁹

and mostly occurs in females aged 25-35 years, with a female to male ratio of 2:1. Patients with PMBL commonly present with cough, chest pain, dyspnea on exertion, pleural and pericardial effusions, and particularly superior vena cava syndrome in 20–30% of the cases.²⁰ The tumor usually presents as a bulky mediastinal mass greater than 7 cm in diameter. Despite the aggressive features, PMBL has a more favorable prognosis than DLBCL, not otherwise specified. PMBL is clinically and biologically related to nodular sclerosing classic Hodgkin lymphoma, and both lymphomas in the mediastinum are thought to arise from the thymic B cells.^{21, 22} Microscopically, PMBL reveals characteristic delicate fibrosis, surrounding small clusters of lymphoma cells and individual tumor cells. Diagnosis of PMBL can be challenging since there are no definitively specific features and an accurate diagnosis relies on the combination of the clinical features, morphology, and immunophenotype.

Cardiothoracic Surgery Commentary (Drs. Bell and Meguid)

Chylothoraces are a challenging problem for the thoracic surgeon. The accumulation of chyle in the pleural space occurs secondary thoracic ductal disruption, transection, or increased lymphatic flow.²³ The diagnosis is obtained through routine laboratory evaluation of the pleural fluid (i.e. pH, lactate dehydrogenase, complete blood count, glucose, amylase) in addition to fluid triglyceride level. A triglyceride concentration greater than 110 mg/dl is relatively specific for this diagnosis while levels much lower than 50 mg/dl suggest an alternate etiology.²⁴ If additional information is warranted, lipid protein pheresis can be performed. The presence of chylomicrons is pathognomonic for chylothorax.²⁵

Etiologies are commonly classified as either traumatic or atraumatic. Traumatic causes account for more than 50% of chylothoraces.²⁶ The vast majority of these injuries are postoperative but nonoperative blunt and penetrating injuries have been described.^{27, 28} Atraumatic disease can be secondary to a variety of lymphatic disorders; however, malignancy is the leading cause, with lymphomatous malignancies being more frequently associated than solid organ malignancies.²⁴

The primary goals of treatment of a chylothorax, regardless of etiology, are cessation of the leak, improvement of symptoms, evacuation of the pleural space and cessation of nutritional losses.²⁹ Tube thoracostomy placement, either surgically or minimally invasively, is an essential component of initial management, and is a reasonable first intervention. This allows for control of symptoms and real-time assessment of drainage volume. In general, low volume chylothoraces respond to conservative measures while large volume outputs typically require more aggressive interventions. High volume chylothoraces (>1.5L/day) should be managed more aggressively as patients are at high-risk for malnutrition, immunosuppression, and infections.³⁰

The Initial conservative measure is typically dietary restriction combined with pleural drainage. Clinicians vary in aggressiveness with this therapy. While some recommend complete enteral rest with total peripheral nutrition, others will employ low-fat diets (<5-10gm/day) with supplementation of medium chain fatty acids. Medium chain fatty acids provide nutrition for enterocytes while having direct systemic rather than portal absorption.

Takuwa et.al demonstrated resolution of post-operative chylothoraces following dietary restriction of fat to less than 10 gm/day alone in 60% of patients experiencing chyle leak following lobectomy.³¹ If these measures fail, operative interventions including pleurodesis, thoracic duct ligation or embolization are recommended.²⁹

For malignant chylothoraces, the above principles hold true with the distinction that chemotherapy or radiation therapy may offer potential treatment. Pleurodesis without embolization or ligation of the thoracic duct, treatment of the malignancy or resolution of the chylothorax otherwise, is not commonly successful due to the high volume of the chyle leak disrupting pleural symphysis. Unfortunately, these treatments may delay procedural interventions and worsen nutritional and immunologic reserves of patients in the meantime. In a single-center review of 18 patients with malignant chylothoraces, clinical resolution of chylothoraces mirrored patients' responsiveness to chemotherapy in patients with lymphoma. For those with metastatic or solid organ disease chemotherapy was ineffective in resolution of chylothoraces.³² On the other hand, surgical intervention with thoracic duct ligation will delay nonsurgical treatment of the cancer by six weeks or more, pending any postoperative complications such as wound infections.

In the present case, the patient presented with mediastinal lymphadenopathy and an effusion refractory to serial thoracentesis. Though the patient had not been diagnosed with a chylothorax prior to consultation, suspicions were elevated due to the interval of recurrence of the effusion and the radiographic suspicion of lymphoma. The patient underwent thoroscopic exploration, evacuation of a large volume of chylous fluid and mediastinal biopsy through a standard three port thoroscopic approach, which he tolerated well. NPO was initiated with supplemental total peripheral nutrition. The patient was then placed on a chemotherapeutic regiment after pathologic confirmation of diagnosis. Interestingly, while the patient demonstrated a biochemical response to chemotherapy, he continued to have a high output from his chest tube. Hence, he eventually required ductal embolization between sequential cycles of chemotherapy for definitive management of the chylothorax. This option was chosen over surgical thoracic duct ligation so as to not delay further administration of chemotherapy. Thoracic duct embolization was performed by a skilled interventional radiologist who first performed lymphangiography to identify and map the course of the thoracic duct, via an inguinal lymph node. A lymphatic channel in the abdomen was then accessed percutaneously, and a microcatheter was advanced into the thoracic duct. This was then successfully embolized with a combination of coils and ethylene vinyl alcohol copolymer (Onyx, Medtronic, Fridley, Minnesota) under fluoroscopic guidance.

Thoracic Anesthesiology and Critical Care Commentary (Drs. Montenij and Dieleman)

Pleural effusions are frequently observed in the surgical and medical intensive care unit.^{33, 34} Usually, the accumulation of fluid in the pleural space is secondary to another disease process, such as heart failure, atelectasis, malignancy, hypoalbuminemia, pancreatitis, or empyema.³³ Extensive use of fluids during resuscitation imposes a serious risk for a

critically ill patient to develop pleural effusions. Moreover, the use of sedatives and muscle relaxants at the institution of mechanical ventilation may result in atelectasis. Especially in cardiac surgical patients, postoperative pleural effusions are a common complication and mainly consist of exudate or hemorrhagic fluid.^{34, 35} In these patients, the prevalence of clinically significant pleural effusions that require drainage may be as high as 10%. Although pleural effusions can contribute to respiratory failure and hemodynamic instability, their clinical significance can vary significantly among patients. Thoracentesis represents both a diagnostic and therapeutic procedure in this setting, but should not be automatically performed because of this heterogeneity in underlying causes, and also because of the potential clinical implications: effects of thoracentesis on pulmonary mechanics and gas exchange are not completely understood, and thoracentesis can even worsen hypoxemia.

Pleural effusions influence arterial oxygenation due to effects on lung mechanics, pulmonary gas exchange, and cardiac function.^{3, 36} Both alveoli and small blood vessels can be compressed by high intrapleural pressures and the effects on ventilation and perfusion probably depend on the underlying disease, the distribution of the disease in the lungs, the nature of the pleural effusions, and the pleural fluid volume. Thoracentesis changes regional ventilation and perfusion and therefore their match or mismatch. Moreover, the net effect on ventilation-perfusion (mis-) matching depends on the timing and technique of drainage.⁶ This differential effect on ventilation and perfusion might explain the variable response to the drainage of pleural effusions. Although thoracentesis usually leads to relief of dyspnea, the hypoxemia may worsen.² This observation can be explained by the timing of thoracentesis and the evaluation of its effects. Re-expansion of compressed lung tissue can take time and can be preceded by an increase in perfusion. This will lead to a temporary increase in intrapulmonary shunting and therefore worsening of hypoxemia. Another explanation is the development of re-expansion edema due to an abrupt increase in regional pulmonary blood flow. As a result, the effect of thoracentesis is multifactorial, time dependent, and difficult to predict.

In the present case, the accumulation of chyle in the pleural space contributed to significant respiratory and hemodynamic compromise. Preoperative thoracentesis could have led to a more stable induction of anesthesia. However, the choice not to drain the pleural fluid preoperatively is justified for a number of reasons. First, as described above, the effects of pleural effusions and its drainage on pulmonary gas exchange are poorly understood and therefore difficult to predict. Second, a thorough hemodynamic assessment including transthoracic echocardiography did not reveal major derangements in the circulatory condition of the patient. Third, induction of anesthesia would be followed by surgical drainage, which could have been performed very rapidly in case of emergency. Furthermore, not only the presence of pleural effusion may have contributed to the observed rapid hemodynamic deterioration. High-volume chyle leak and protein-losing enteropathy probably contributed to intravascular volume depletion in this patient. Finally, mediastinal masses can lead to mechanical compression of the heart or major vessels thereby impeding venous return and right ventricular ejection.³⁷ However, in this case the patient remained hemodynamically stable once the pleural effusions were drained. Hence, mediastinal mass effect seems less likely to have been the primary contributor to the encountered hemodynamic instability.

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Figure 1.
Computed tomography of the chest showing right pleural effusion and conglomerate lymphadenopathy encasing the trachea and the aortic arch.

Figure 2.
Video-assisted thoracoscopic surgery demonstrating right-sided chylothorax.

Figure 3.
Solidified sample of pleural fluid demonstrating solidification of chyle at room temperature (positioned upside down).