Lesson 303: PreAnesthetic Assessment of the Patient With Cirrhosis-Related Pulmonary Complications

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Professional Gaps

Patients with advanced liver disease frequently present for surgical procedures. Most anesthesiologists do not know the extent to which cirrhosis and liver-related pulmonary disease may contribute to morbidity and mortality during anesthesia.

Learning Objectives

At the end of this activity, the participant should be able to:

1. Identify the most common etiologies for development of cirrhosis.
2. Outline the mechanisms for increased pulmonary complications associated with advanced liver disease.
3. Identify the 3 biochemical measurements used to determine the Model for End-Stage Liver Disease (MELD) score.
4. List the differential diagnosis for hypoxia in a patient with cirrhosis.
5. Tabulate the pathophysiology of hepatopulmonary syndrome (HPS) and portopulmonary hypertension (POPH).
6. Compare and contrast clinical features of HPS and POPH.
7. Outline diagnostic criteria for POPH.
8. List diagnostic criteria for HPS.
9. Recognize treatment options for HPS and POPH.
10. Prescribe an anesthetic plan for patients with HPS or POPH.

Case History

A 57-year-old man with alcoholic cirrhosis and ongoing gross hematuria presented for transurethral
resection of a bladder tumor. Past medical history was significant for hypertension, paroxysmal atrial flutter, chronic obstructive pulmonary disease (COPD), morbid obesity (body mass index 40 kg/m²), obstructive sleep apnea, and previous use of tobacco and alcohol. The patient had been diagnosed with alcoholic cirrhosis several years earlier and exhibited clinical features of portal hypertension. He had a prior hospitalization for hepatic encephalopathy and upper gastrointestinal bleed secondary to esophageal varices. The patient reported a 6-month history of worsening shortness of breath and dyspnea on exertion with severely impaired exercise tolerance. On physical examination, he was mildly dyspneic at rest. Preoperative oxygen saturation was 88% on room air. Blood pressure was 117/64 mm Hg with a heart rate of 66 beats per minute. Cardiac examination was unremarkable. Lung examination revealed expiratory wheezes.

Cirrhosis is the 12th leading cause of death in the United States, accounting for approximately 32,000 deaths in 2010, with a mortality rate of 10.3 per 100,000. Infection with the hepatitis C virus and alcoholic liver disease are the 2 most common causes of cirrhosis. However, with the increase in obesity, the diagnosis of nonalcoholic fatty liver disease also is increasing. Patients with cirrhosis are living longer through improved medical and surgical treatments and presenting with increased frequency for surgical interventions. Many of these patients undergo surgery during the last 2 years of life. Perioperative morbidity and mortality should be anticipated as a result of the effects of advanced liver disease on virtually all organ systems. Even patients with well-compensated cirrhosis are at greater risk for complications of surgery and exposure to anesthesia. Furthermore, underlying pulmonary disorders inherently carry an increased perioperative risk for exacerbation of lung disease, pneumonia, and respiratory failure requiring prolonged mechanical ventilation. Although liver disease may manifest itself as dysfunction in multiple organ systems, this review focuses on preanesthetic assessment of pulmonary disorders associated with chronic liver disease, with an emphasis on hepatopulmonary syndrome (HPS) and portopulmonary hypertension (POPH).

Predictive Models and Perioperative Risk

Historically, the Child-Turcotte-Pugh (CTP) scoring system was used to predict surgical risk in patients with liver disease. Although the CTP score is clinically useful and simple to calculate, a limitation is its subjective grading of ascites and encephalopathy. The Model for End-Stage Liver Disease (MELD) score was developed to predict 3-month postprocedure mortality in patients undergoing transjugular intrahepatic portosystemic shunt procedures (Figure 1). In 2002, the United Network for Organ Sharing subsequently adopted the MELD score to prioritize and fairly allocate donor organs. Using a complex logarithmic formula, the MELD score incorporates 3 objective variables: total bilirubin concentration, serum creatinine, and international normalized ratio (INR). The MELD score ranges from 6 to 40, with low scores reflecting early liver disease and higher scores representing severe liver disease. Table 1 compares the clinical components of CTP with MELD scores.
The magnitude of perioperative risk depends on several factors including severity of hepatic decompensation and type of surgery. A study of 800 cirrhotic patients undergoing major surgery demonstrated that the MELD score correlated with both short- and long-term mortality. Thirty-day mortality rates correlated directly with MELD score and ranged from 5.7% (MELD score <8) to more than 50% (MELD score >20). In addition, studies have shown that patients with cirrhosis have a greater mortality when undergoing emergency surgery compared with patients without liver disease. Morbidity and mortality risks are especially high in patients undergoing cardiac and open abdominal procedures including cholecystectomy, gastric resection, colectomy, and hepatic resection. Data suggest that patients with MELD scores below 10 can undergo elective surgery with minimal risk. However, caution should be exercised with elective surgery in patients with MELD scores between 10 and 15. In general, the risks and benefits of elective surgery in patients with MELD scores above 15 should be carefully evaluated, as perioperative morbidity and mortality are greatly increased in this population.

**Pulmonary Conditions Associated With Cirrhosis**

A variety of pulmonary disorders are encountered in patients with liver disease and portal hypertension (Table 2). Patients with risk factors for liver disease such as alcohol and drug use frequently have a history of tobacco use with concomitant COPD. Emphysema is seen in patients with cirrhosis secondary to α-1 antitrypsin deficiency. Liver disease can result in altered pulmonary mechanics consistent with restrictive lung disease. A decrease in forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁), functional residual capacity, and oxygen saturation often is seen in patients with ascites. Hypoalbuminemia from impaired hepatic synthesis of albumin and increased intraabdominal pressure due to ascites predisposes patients to pleural effusions, atelectasis, and pulmonary edema. Hepatic hydrothorax, defined as the presence of pleural fluid in a patient with cirrhosis after ruling out cardiac or pulmonary etiology, is seen in approximately 6% to 10% of patients with advanced liver disease. The risk for aspiration increases when patients demonstrate signs of encephalopathy. Finally, liver disease can disrupt the proper function of intrapulmonary vascular system.

**Hepatopulmonary Syndrome**

HPS is an intrapulmonary vascular disorder associated with liver disease and portal hypertension. The condition is defined as hypoxemia (PaO₂ <70 mm Hg or alveolar-arterial [A-a] oxygen gradient >20 mm Hg) and pulmonary vascular dilation in patients with chronic liver disease or portal hypertension. HPS is not associated with a specific etiology of chronic liver disease or with the degree of hepatic dysfunction. The prevalence of HPS is reported as 15% to 30% in patients with cirrhosis undergoing evaluation for liver transplantation.
Pathophysiology

HPS is hypoxia caused by dilation of the pulmonary capillary bed in response to vasodilatory substances. Nitric oxide (NO) appears to be the major mediator of pulmonary vasodilation.\(^7,8,10\) Pulmonary vascular dilatation leads to shunting of blood from the venous to the arterial circulation by either microscopic or macroscopic shunts. This intrapulmonary shunting ultimately results in ventilation/perfusion (V/Q) mismatch and impaired gas exchange.\(^8\)

Type 1 HPS is characterized by microscopic diffuse pulmonary vascular dilatation and a decrease in alveolar transit time. The rapid flow of blood through dilated pulmonary capillaries results in failure of red cell oxygenation.\(^9\) Type 2 HPS results from macroscopic pulmonary vascular dilatation and formation of arteriovenous (AV) connections leading to discrete shunts that bypass the alveoli.\(^11\) Increasing the alveolar oxygen therefore does not improve hypoxia in patients with type 2 HPS.\(^7,9\)

Clinical Presentation

Most patients with HPS exhibit signs and symptoms of chronic liver disease (esophageal varices, ascites, palmar erythema, splenomegaly). Although most patients with HPS are asymptomatic or report mild dyspnea, clinical features can encompass a spectrum of findings including digital clubbing, cyanosis, and hypoxia.\(^10\) Platypnea (dyspnea induced by the upright position and relieved by recumbency) and orthodeoxia (arterial deoxgenation increased in the upright position and relieved by recumbency) are characteristic but not pathognomonic and occur in 25% of patients with HPS.\(^7,8,10\) The phenomena are explained by the worsening of diffusion–perfusion matching and an increase of the shunt fraction in the upright position because of increased perfusion of the lower lobes.\(^10,12\) Telangiectasia is an indicator of systemic and pulmonary vasodilation.\(^7\)

Morbidity and Mortality

The estimated median survival of patients with HPS without liver transplant is 2 years, but shorter for those with a PaO\(_2\) below 50 mm Hg. However, death appears to be related more to complications of liver disease or portal hypertension rather than hypoxemic respiratory failure.\(^8\)

Diagnosis

Pulse oximetry is an inexpensive and effective screening tool for HPS and should be performed in both the upright and supine positions. If oxygen saturation is measured only in the supine position, the sensitivity for the detection of HPS is decreased by V/Q mismatch.\(^10\) Decreased oxygen saturation on pulse oximetry should be evaluated with arterial blood gases drawn on room air in both supine and standing positions. A PaO\(_2\) of less than 80 mm Hg or A-a gradient below 15 mm Hg suggest impaired oxygenation. In patients older than age 65 years, a PaO\(_2\) of less than 70 mm Hg or A-a gradient above 20 mm Hg on room air is used as the diagnostic threshold. An elevated A-a gradient is the most sensitive indicator of impaired oxygenation because it accounts for hyperventilation that is common in patients with liver disease. The right-to-left shunt fraction can be calculated by repeating the test on 100% oxygen.\(^9,12\)

Contrast echocardiography is the most sensitive test for identifying intrapulmonary shunting. Contrast is administered in the form of agitated saline. In the presence of an intracardiac shunt, microbubbles are seen almost immediately in the left atrium after venous injection of contrast. With intrapulmonary
shunting, microbubbles appear 3 or more cardiac cycles after injection, as the agitated saline must first traverse the pulmonary circulation. In general, transesophageal echo-cardiography is more sensitive than transthoracic echocardiography in demonstrating intrapulmonary shunting. Although contrast echocardiography is a sensitive test for intrapulmonary shunting, it lacks the ability to quantify the degree of the condition. Scans of lung perfusion using technetium-99 macroaggregated albumin (Tc-99m MAA) have proven to be beneficial in the diagnosis.\(^8\) MAA is characteristically trapped in the pulmonary vascular bed. However, dilation of the intrapulmonary vessels seen in HPS allow albumin to pass through the lungs resulting in abnormal uptake in the kidneys and brain. The proportion of radionucleotide taken up by the kidneys and brain can be used to quantify the degree of shunting.\(^7,12\) Unlike contrast echocardiography, nuclear scanning cannot differentiate between intracardiac and intrapulmonary shunting.

Pulmonary angiography is useful for identifying large AV communications that may be amenable to embolization in patients with type 2 HPS.\(^12\) However, it is seldom performed due to the invasive nature of the procedure.

**Treatment**

Currently, no effective medical treatment exists for HPS. Supplemental oxygen is the most frequently recommended therapy. Although oxygen improves symptoms related to hypoxemia, mortality is unchanged. Ideally, HPS could be treated medically by targeting mediators of pulmonary vasodilation. Somatostatin analogs, methylene blue, cyclooxygenase inhibitors, and a diet low in L-arginine have been proposed to decrease the production or action of vasodilators. However, the evidence supporting these treatments is anecdotal.\(^12\)

The only definitive treatment is surgical. In patients with type 1 HPS, hypoxia responds well to 100% oxygen and orthotopic liver transplantation (OLT) is curative with reversal of clinical features and resolution of preoperative oxygen dependency within several months.\(^8\) Furthermore, normalization of NO levels has been documented post-OLT.\(^10\) Conversely, type 2 HPS does not respond to 100% oxygen administration and some experts consider it a contraindication to general anesthesia and transplant surgery.\(^6\)

**Anesthetic Considerations**

Although studies have found increased perioperative morbidity and mortality in patients with advanced liver disease, minimal data exist regarding the perioperative risk in patients with HPS undergoing elective surgery. Patients with HPS undergoing OLT do experience increased morbidity and mortality in the early postoperative period before resolution of the gas exchange abnormalities.\(^13\) In cases where general anesthesia is required, clinicians should take care to avoid exacerbation of hypoxemia. Volatile anesthetics and positive pressure ventilation may worsen hypoxemia by inhibiting hypoxic pulmonary vasoconstriction and amplifying the V/Q mismatch, respectively. In order to maximize oxygenation, high concentrations of fractional inspired oxygen and supine or Trendelenburg positions should be maintained. An arterial line is useful for serial monitoring of arterial PaO\(_2\). Neuraxial and regional anesthesia should be considered as an alternative to general anesthesia in suitable candidates. However, it is imperative to evaluate coagulation, as patients with advanced liver disease have impaired synthesis of clotting factors and thrombocytopenia due to splenomegaly.
Portopulmonary Hypertension

POPH is a condition of increased pulmonary vascular resistance in the setting of portal hypertension.\textsuperscript{14} In the absence of other causes for pulmonary hypertension, the diagnosis of POPH is based on a variety of criteria (Table 3).\textsuperscript{7,9,14} POPH often is characterized as mild, moderate, or severe (Table 4).\textsuperscript{7,12} When the mean pulmonary artery pressure (mPAP) exceeds 50 mm Hg, the condition becomes life-threatening and can lead to more severe hypoxemia and right ventricular failure.\textsuperscript{6} The development of POPH appears to be independent of the etiology of portal hypertension.\textsuperscript{12} The reported prevalence of POPH in patients with cirrhosis ranges between 2\% and 10\%.\textsuperscript{7,8}

Pathophysiology

A wide spectrum of pathology related to POPH is discussed in the literature; however, the exact mechanism remains poorly understood. Vascular changes associated with POPH include intimal proliferation, medial hypertrophy of smooth muscle cells, and fibrosis within small pulmonary arteries.\textsuperscript{9} In the presence of portal hypertension, autopsy analyses also have confirmed the contribution of microscopic pulmonary artery thromboembolism and platelet aggregates to the development of POPH.\textsuperscript{15} It is suggested that vascular mediators normally metabolized by a healthy liver are shunted from abnormal mesenteric and hepatic vessels to the pulmonary arterial bed in the setting of portal hypertension. Potential humeral substances include serotonin, interleukin-1, endothelin-1, glucagon, secretin, thromboxane B\textsubscript{2}, and vasoactive intestinal peptide.\textsuperscript{7} Finally, because a minority of patients with cirrhosis develop POPH, a proliferative pulmonary vasculopathy may be the result of genetic defect. However, a specific gene has yet to be identified in this population of patients.

Clinical Presentation

POPH has an insidious onset with the clinical presentation ranging from asymptomatic to multiple physical findings consistent with right ventricular failure. The most common presenting symptom is dyspnea on exertion.\textsuperscript{12} Additional nonspecific symptoms include fatigue, palpitations, syncope, or chest pain. On examination, evidence of right-sided heart failure may be detected with evidence of an accentuated pulmonary component of the second heart sound, a murmur consistent with tricuspid regurgitation, jugular venous distension, lower extremity edema, and ascites.\textsuperscript{7-9}

Morbidity and Mortality

No direct correlation exists between the severity of liver disease and the severity of POPH. Patients with severe POPH have a poor prognosis. The overall 3- to 5-year survival ranges from 30\% to 50\%, with death occurring from complications of liver disease or POPH in an equal number of cases.\textsuperscript{8}
Diagnosis

In addition to a detailed history and physical examination, evaluation of a patient with cirrhosis and suspected POPH should consist of an electrocardiogram (ECG) and chest radiograph. ECG findings may include right ventricular hypertrophy, right-axis deviation, and right bundle branch block. Chest radiograph demonstrates cardiomyopathy and enlarged pulmonary vasculature. Arterial blood gas analyses may reveal decreased PaCO₂, increased A-a oxygen gradient, and mild hypoxemia.

Because patients with POPH often are asymptomatic, diagnosis frequently is made during screening for liver transplantation. The recommended screening test is transthoracic echocardiography (TTE). TTE findings suggestive of POPH include the presence of tricuspid jet velocity, pulmonic valve insufficiency, right ventricular wall hypertrophy and/or dilation, and an increased estimated right ventricular systolic pressure. A diagnosis of POPH must be confirmed with right heart catheterization. Given that echocardiography cannot estimate pulmonary vascular resistance (PVR), approximately 30% to 40% of patients with an estimated elevation of right ventricular systolic pressure have a finding of normal PVR during right heart catheterization.

Of note, patients with liver disease have hyperdynamic circulation that leads to increased cardiac output in the setting of low systemic vascular resistance. Approximately 20% of patients with cirrhosis have an increased pulmonary artery pressure but not true POPH. The increase in pulmonary artery pressure is likely due to elevated right ventricular output across a normal PVR. Therefore, it is essential to accurately characterize hemodynamic parameters to avoid misdiagnosis.

Finally, other common causes of pulmonary hypertension must be excluded. Patients with chronic liver dysfunction may have coexisting conditions that could result in pulmonary hypertension, such as left heart disease, valvular heart disease, obstructive lung disease, sleep apnea, and chronic thromboembolism.

Treatment

If hypoxemia is present, supplemental oxygen is initiated. Medication therapy in the form of vasodilators is the mainstay of treatment. Bosentan, a competitive antagonist of endothelin-1 at the endothelin-A and endothelin-B receptors, decreases PVR and can be administered orally. Sildenafil is a selective inhibitor of phosphodiesterase type 5 and enhances the vasodilatory effects of NO by preventing the degradation of cyclic guanosine monophosphate, which promotes relaxation of vascular smooth muscle and increases blood flow. Epoprostenol, a prostacyclin analog, is a potent dilator of pulmonary vasculature. Treatment with epoprostenol entails significant drawbacks, specifically the requirement of permanent central venous access and uninterrupted infusion of the medication.

Mild to moderate POPH can potentially be reversed after OLT. However, most experts agree that OLT is contraindicated in patients with severe POPH due to significant perioperative morbidity and mortality. Severe POPH not only carries a pulmonary risk, but also can lead to compromised perfusion of the liver graft. Congestion of the hepatic veins due to decreased right ventricular function carries a substantial risk for primary graft dysfunction. Table 5 provides a comparison of HPS and POPH.
Anesthetic Considerations

Patients with POPH present a unique challenge for the anesthesiologist. It is important to avoid hypercapnia, hypothermia, and acidosis, as each may worsen pulmonary artery hypertension. A patient with severe POPH may require invasive cardiopulmonary monitoring with a pulmonary artery catheter or TTE. Patients receiving vasodilating agents should remain on these medications during the intraoperative course. Even a brief interruption of a vasodilatory medication (particularly epoprostenol) can result in severe rebound pulmonary hypertension. Neuraxial and regional anesthesia should be considered as an alternative to general anesthesia in suitable candidates.

Management of Case Presented

After completion of the initial preoperative history and physical examination, further testing was ordered to determine the etiology of the patient’s hypoxia and dyspnea. ECG revealed normal sinus rhythm with left ventricular hypertrophy. Evidence of cardiomegaly was present on a chest radiograph. Pulmonary function tests revealed FEV₁ of 51% predicted, FVC of 66% predicted, and an FEV₁/FVC ratio of 59%. Arterial blood gas demonstrated hypoxemia, with a PaO₂ of 53 mm Hg. A contrast echocardiogram was performed, which confirmed a right to left shunt with delayed appearance of bubbles in the left ventricle (4 to 5 beats after the appearance of bubbles in the right ventricle), suggesting HPS. A right heart catheterization demonstrated severe pulmonary hypertension with an elevated mPAP of 47 mm Hg. Pulmonary vascular resistance was 192 dyne/s/cm⁻⁵ and pulmonary capillary wedge pressure was elevated, at 29 mm Hg. Pulmonary angiography performed at the time of the right heart catheterization revealed dilated pulmonary arteries without AV malformations.

Despite the presence of pulmonary hypertension in the setting of portal hypertension, the patient did not meet the criteria for POPH. Rather, the etiology of pulmonary hypertension was likely multifactorial and resulted from long-standing COPD and obstructive sleep apnea, as well as underlying left heart disease and volume overload.

Based on the results of the studies, the patient’s symptoms were medically optimized under the
direction of his cardiologist and pulmonologist. He was started on an inhaled long-acting 
anticholinergic bronchodilator and β-2 agonist with home oxygen therapy and initiation of continuous 
positive airway pressure. Furosemide was titrated along with the addition of spironolactone for volume 
overload.

On the day of surgery, the patient reported less dyspnea on exertion. His lungs were clear to 
auscultation bilaterally and oxygen saturation was 94% on 2 L flow through a nasal cannula in the 
sitting position. Platelet count and INR were 125,000 and 1.3, respectively. In an effort to avoid positive 
pressure ventilation with exacerbation of intrapulmonary shunting as well as increased PVR from 
hypoxia, hypercarbia, and acidosis, the anesthetic plan was spinal anesthesia. Intraoperative and 
immediate postoperative course was unremarkable. The patient was discharged home later the same 
day.

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REFERENCES

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**Post-test**

1. The 3 biochemical measurements used to determine Model for End-Stage Liver Disease (MELD) score are_____.
   a. bilirubin, albumin, international normalized ratio (INR)
   b. bilirubin, INR, platelet count
   c. bilirubin, serum creatinine, INR
   d. albumin, serum creatinine, INR

2. Which of the following is FALSE regarding diagnosis of hepatopulmonary syndrome (HPS)?
   a. Technetium-99 macroaggregated albumin (Tc-99m MAA) lung perfusion scan can be used to differentiate intrapulmonary from intracardiac shunting.
   b. Pulmonary angiography is useful for identifying arteriovenous communications in type 2 HPS.
   c. Transesophageal echocardiogram with contrast is the most sensitive test for intrapulmonary shunt.
   d. Tc-99m MAA lung perfusion scan can be used to quantify the shunt fraction.

3. _____ is considered a curative treatment for patients with HPS.
   a. Supplemental oxygen
   b. Methylene blue
   c. Orthotopic liver transplant
   d. IV epoprostenol

4. Which of the following statement pertaining to perioperative risk in patients with cirrhosis is FALSE?
   a. Morbidity and mortality risks are especially high in patients undergoing cardiac and open abdominal procedures.
   b. The magnitude of perioperative risk depends on the severity of hepatic dysfunction and the type of surgery.
   c. A MELD score of 25 is associated with a similar perioperative mortality risk compared with a MELD score of 8.
   d. Patients with cirrhosis have a greater mortality when undergoing emergency surgery compared with those without liver disease.
5. Which of the following tests confirms the diagnosis of portopulmonary hypertension (POPH)?

a. Transthoracic echocardiography  
b. Arterial blood gas analyses  
c. Right heart catheterization  
d. Pulmonary function testing

6. Which of the following is not a diagnostic criterion for POPH?

a. Mean pulmonary artery pressure (mPAP) >25 mm Hg at rest or >30 mm Hg during exercise  
b. Pulmonary capillary wedge pressure <15 mm Hg  
c. Pulmonary vascular resistance (PVR) >240 dyne/s/cm⁵  
d. Alveolar-arterial oxygen gradient >20 mm Hg

7. Severe POPH is classified as _____.

a. mPAP > 45 mm Hg  
b. PVR 240-500 dyne/s/cm⁵  
c. PVR 500-800 dyne/s/cm⁵  
d. mPAP 25-35 mm Hg

8. The most common presenting symptom of POPH is _____.

a. fatigue  
b. dyspnea on exertion  
c. chest pain  
d. syncope

9. Which of the following statements pertaining to pulmonary physiology associated with chronic liver disease is TRUE?

a. A decrease in FVC, FEV₁, FRC, and oxygen saturation is seen in patients with ascites.  
b. Upright position will result in improvement of ventilation/perfusion (V/Q) mismatch in patients with HPS.  
c. Patients with cirrhosis are NOT at increased risk for aspiration.  
d. A direct correlation exists between the severity of liver disease and severity of POPH.

10. Which of the following is not an anesthetic consideration for patients with HPS or POPH?

a. Neuraxial and regional anesthesia should be considered as an alternative to general anesthesia in suitable candidates.  
b. Epoprostenol can be stopped safely during the intraoperative period in patients with POPH.  
c. Positive pressure ventilation may worsen hypoxemia by amplifying the V/Q mismatch in patients with HPS.  
d. An increase in pulmonary artery hypertension can be seen with hypercapnia, hypothermia, and acidosis.