Anesthesia for Patients with Concomitant Hepatic and Pulmonary Dysfunction

Geraldine C. Diaz, DO[a,*], Michael F. O'Connor, MD[a], John F. Renz, MD, PhD[b]

KEYWORDS

• Hepatopulmonary syndrome • Portopulmonary hypertension
• Intrapulmonary vascular dilatation • Cirrhosis • Anesthesia

KEY POINTS

- Hepatic function and pulmonary function are interrelated with failure of one organ system affecting the other.
- With improved treatments, patients with concomitant hepatic and pulmonary failure increasingly have a good quality of life and life expectancy. Therefore, more patients are presenting for elective as well as emergent surgical procedures.
- Hepatopulmonary syndrome originates from the development of intrapulmonary vascular dilatations that are common in patients with end-stage liver disease.
- Hypoxemia requires a thorough evaluation in patients with end-stage liver disease. The most common causes respond to appropriate therapy.
- Portopulmonary hypertension and hepatopulmonary syndrome are associated with high perioperative morbidity and mortality.

INTRODUCTION

The prevalence of end-stage liver disease (ESLD) has steadily increased over the past 2 decades to become a common comorbidity encountered by the practicing anesthesiologist. Dramatic improvements in the medical management of cirrhosis by gastroenterologists and liver surgeons, including the use of beta blocker prophylaxis for portal hypertension, endoscopy for the management of esophageal varices, transjugular intrahepatic portosystemic shunt, and effective medications to suppress viral replication, have increased the life expectancy and quality of life for patients with ESLD. Today, patients with ESLD are seeking emergent as well as elective surgical

[a] Department of Anesthesiology/Critical Care, University of Chicago, 5841 South Maryland Avenue, Chicago, IL 60637, USA; [b] Department of Surgery, University of Chicago, 5841 South Maryland Avenue, Chicago, IL 60637, USA
* Corresponding author.
E-mail address: gdiaz@dacc.uchicago.edu

Anesthesiology Clin 34 (2016) 797–808
http://dx.doi.org/10.1016/j.anclin.2016.06.012
anesthesiology.theclinics.com

procedures for conditions that were previously regarded as posing too high a risk for surgery. This change mandates not only a greater understanding of the physiology of cirrhosis but also the systemic manifestations of the failing liver on other organs.

This article examines anesthesia for patients with concomitant hepatic and pulmonary dysfunction. Concomitant hepatic and pulmonary dysfunction can be encountered within 3 broad scenarios. The most frequent scenario is patients with a history of independent pulmonary and hepatic diseases, for example, a patient with alcoholic liver disease and chronic obstructive pulmonary disease secondary to smoking. In this setting, the history and severity of the disease processes will be variable as will the influence of each on the other organ system's physiology. The variability in pathophysiology typically results in one organ system being the dominant source of morbidity that is accentuated by dysfunction of the other. In these situations, anesthetic management is directed toward the principally disaffected organ system. The truly concomitant scenarios of ESLD with associated pulmonary manifestations, such as portopulmonary hypertension (POPH) and hepatopulmonary syndrome (HPS), as well as inherited metabolic disorders affecting both organ systems, such as alpha-1-antitrypsin (A1AT) deficiency and cystic fibrosis (CF), require more complex medical management and are the focus of this article. Current definitions, diagnostic criteria, pathophysiology, algorithms for preoperative screening, and management for these patients in the setting of nontransplant surgery are explored.

PULMONARY FUNCTION IN THE SETTING OF LIVER DISEASE

The failing liver imposes early and significant effects on pulmonary function. The most frequently recognized restrictive pulmonary complications arise from ascites secondary to portal hypertension. Ascites expands the abdominal cavity, impedes mobility of the diaphragm, and can result in pleural effusions.[1] In addition to these restrictive changes, liver disease impairs gas exchange through ventilation-perfusion (VQ) mismatch, impedes oxygen diffusion, and facilitates intrapulmonary shunts.[1] Patients with ESLD demonstrate a widened alveolar-arterial oxygen concentration gradient (A-a gradient), impaired diffusing capacity of the lungs for carbon monoxide (DLCO), and hypoxemia that typically progress with the severity of liver disease.[2,3]

The most commonly encountered pulmonary complications associated with ESLD result from VQ mismatch. Ascites, anasarca, pleural effusions, and hepatomegaly impede chest wall motion, reduce lung recoil, and impair diaphragmatic excursion.[4] These restrictive changes reduce respiratory volumes, decrease functional residual capacity, and increase closing capacity.[5] Increased pulmonary parenchymal pressure from interstitial edema promotes small airway collapse and obstruction.[5] Therefore, hypoxemia evolves with liver disease progression from a principally restrictive process to a combination of restrictive as well as obstructive processes.[6]

Although the physiology of portal hypertension has been widely recognized, the physiologic changes induced on pulmonary vascular tone through hepatic vasoactive mediators are increasingly recognized as a significant contribution to VQ imbalance.[5] As cardiac output and circulating blood volumes increase, systemic venodilation increases blood flow through the pulmonary circulation. This increase creates a relative VQ imbalance of impaired gas exchange through physiologic shunting. Hypoxemia is accentuated by inhibition of the hypoxic pulmonary vasoconstrictive reflex observed in cirrhosis.[7]

Abnormal diffusion capacity can be an early manifestation of pulmonary dysfunction in patients with ESLD with a DLCO of less than 75% predicted observed in most liver transplant candidates.[3,4] A reduced DLCO is hypothesized to result from the