# End-Stage Liver Disease: Challenges and Practice Implications

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**KEYWORDS**

• End-stage liver disease • End of life • Palliative care • Pain

An estimated 5.5 million Americans have chronic liver disease. It is the twelfth leading cause of death in the United States, accounting for more than 27,000 deaths annually.[1] As the seventh leading cause of death among people aged 25 to 64 years, end-stage liver disease (ESLD) affects many Americans in the most productive years of their lives.[2] Because of a large and growing disparity between the availability of donor livers and the number of patients waiting for a transplant, increasing numbers of patients die before receiving a transplant,[3–5] or may not be candidates for liver transplant because of comorbidities.[6] In fact, the vast majority of individuals with ESLD are not liver transplant candidates. Despite the increasing number of individuals who are dying of ESLD, little is documented about their end-of-life challenges as the disease progresses. The purpose of this article is to highlight specific challenges for people with ESLD, their families, and their implications for health care providers: ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, malnutrition, altered drug metabolism, renal insufficiency and hyponatremia, hepatocellular carcinoma, and pain. The authors also present a case study to illustrate disease progression and difficulties facing patients, family members, and providers.

## END-STAGE LIVER DISEASE

Although the average layperson associates liver disease with alcohol abuse, alcohol itself causes only a small percentage of chronic liver disease in the world. Currently,

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hepatitis B is the leading cause of liver disease worldwide, although hepatitis C is becoming an increasing problem.[7] Cryptogenic cirrhosis, which has been associated with nonalcoholic steatohepatitis (NASH) and the metabolic syndrome, is the fastest growing cause of cirrhosis and liver failure. The metabolic syndrome is a constellation of conditions consisting of Type II diabetes, hyperlipidemia, hypertension, and obesity.[8] People with this syndrome are at high risk for cardiovascular events. How alcohol and the metabolic syndrome affect liver damage and fibrosis are still largely unknown. Other etiologies include inborn errors of metabolism, such as hemochromatosis (iron overload); Wilson's disease (copper overload); and alpha-1 antitrypsin deficiency.

Most commonly, cirrhosis is a process of injury and repair that occurs over decades. One exception is hepatitis C infection, which can result in cirrhosis in less than 2 years, especially with recurrent hepatitis C following liver transplant.[9] The progression to cirrhosis is frequently asymptomatic; the affected individual is not aware of the liver disease until a complication of cirrhosis occurs. Cirrhosis can be compensated or decompensated. Compensated cirrhosis occurs when the liver continues to function normally despite the development of progressive damage; it can continue for years. Approximately 80% of patients who are diagnosed with cirrhosis remain compensated for the next 10 years.

Decompensated cirrhosis (ie, a decline in liver function values) is associated with increased mortality from different causes, such as liver failure, renal failure, and infections. The first attempt to predict prognosis for people with declining liver function was the Child classification, which took into account laboratory values (lowered serum albumin and clotting factors) and clinical assessment (degree of encephalopathy, presence of malnutrition, and amount of ascites). This classification has been modified several times and is now known as the Child-Pugh-Turcotte (CPT) score.[10] Based on the sum of the scores for each of the five variables (ascites, encephalopathy, bilirubin, albumin, and prothrombin time) patients are grouped into three classes: A, B, and C. Patients scoring 5 to 6 have ''Class A'' liver failure. Patients scoring 7 to 9 have ''Class B'' liver failure. Patients scoring 10 to 15 have ''Class C'' and far greater mortality (1-year median survival is 45% and 2-year is 38%) than the other two classes.

A more recent scoring system to predict the probability of 3-month mortality is the Model for End-Stage Liver Disease, or MELD.[11] This is a weighted formula using bilirubin, international normalized ratio (INR), and creatinine levels.[12] Currently, patients on the liver transplant waiting list are ranked by their MELD scores. The higher the score (6 to 40) the more severe the disease.[10]

### *Pathophysiology*

End-stage liver disease is a term that is over used and should be reserved for those individuals who have abnormalities of renal function and hyponatremia caused by liver disease. Decompensated cirrhosis with abnormalities of liver synthetic and excretory function (eg, serum albumin and other serum proteins, INR, bilirubin) is more common and occurs when less than 10% of liver-cell mass remains. Death usually results from one of the complications of decompensated cirrhosis, rather than from the additional loss of liver cells.

Cirrhosis is therefore a diffuse process of injury and repair, resulting in a significant increase in fibrous tissue, the loss of normal hepatic architecture, and distortion of vascular and bile flow. Nodules of liver cells form and they are surrounded by fibrous tissue. These nodules have lost their normal blood supply and are nourished through a dense capillary network that envelops the nodules. The normal liver receives up to 85% of nutrition and oxygen through the portal vein, whereas the cirrhotic liver is

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