Primary Care Management of the Patient with Cirrhosis

Case Study and Commentary, Kenneth D. Ingram, PA, and Atif Zaman, MD, MPH

In 2000, cirrhosis of the liver was the 12th leading cause of death in the United States, accounting for more than 25,000 deaths [1]. In the majority of cirrhosis cases, the cause is chronic hepatitis C virus (HCV) infection, alcoholism, or a combination of the 2, but there are a variety of other less common causes (Table 1) [2]. The incidence of cirrhosis is expected to continue to rise in coming decades related to increased numbers of individuals with HCV who have a duration of infection greater than 20 to 30 years, when risk for development of cirrhosis peaks [3]. Direct medical costs for HCV-related disease are expected to reach $11 billion for the period of 2010–2019 [4]. Furthermore, the incidence of nonalcoholic steatohepatitis is expected to rise as U.S. rates of obesity and diabetes escalate; a proportion of these individuals are also likely to develop cirrhosis. Given these projections, it is imperative that primary care providers are cognizant of the clinical findings associated with cirrhosis and the preventive health measures that may reduce morbidity and mortality among patients with chronic liver disease.

CASE STUDY
Initial Presentation

A 46-year-old man with chronic HCV infection presents for the first time to his new primary care physician to establish care after recently moving to the area for a new job.

History

Currently, the patient feels well and has no symptoms except for occasional mild joint and muscle pain. His risk factor for HCV is a remote history of brief intravenous drug use approximately 28 years ago. He has had no use in the last 25 years. Previously he drank 2 to 3 beers a day for about 15 years but stopped completely after being diagnosed with HCV about 10 years ago. He is married and works in the sales industry. He has not had previous workup or treatment of his HCV beyond advice to stop drinking alcohol.
Physical Examination

On physical examination, the patient is a well-nourished adult male without muscle wasting. There is no scleral icterus. Lungs are clear to auscultation bilaterally. Heart rate and rhythm are regular with no murmurs. Abdomen is convex, with normal bowel sounds and no tenderness to palpation. Liver edge is palpated at 1 cm below the right costal margin on inspiration; there is no splenomegaly or fluid wave and no other masses are noted. The patient is alert and oriented and without asterixis. No edema or discoloration are observed on extremities. Skin is warm and dry with a few small spider telangiectasia noted on the upper trunk.

Laboratory Evaluation

Laboratory testing reveals the following:

- **Sodium**: 138 mEq/L
- **Creatinine**: 0.7 mg/dL
- **Glucose**: 94 mg/dL
- **Bilirubin, total**: 0.7 mg/dL
- **Aspartate aminotransferase**: 46 U/L
- **Alanine aminotransferase**: 30 U/L
- **Albumin**: 3.6 g/dL
- **Alkaline phosphatase**: 100 U/L
- **Hemoglobin**: 14.6 g/dL
- **White blood cell count**: $3.3 \times 10^3/\mu L$
- **Platelet count**: $134 \times 10^3/\mu L$
- **Prothrombin time/international normalized ratio**: 1.18
- **HCV genotype**: 1b
- **HCV polymerase chain reaction**: $1.8 \times 10^9$
- **Hepatitis A virus antibody**: Negative
- **Hepatitis B virus (HBV) surface antibody**: Negative
- **HBV core antibody**: Negative
- **HBV surface antigen**: Negative

Radiographic Evaluation

Computed tomography of the abdomen with contrast shows a slightly smaller than normal liver that is nodular in appearance with a prominent left lobe. There is associated marginal splenomegaly.

**Table 1. Common Causes of Cirrhosis**

<table>
<thead>
<tr>
<th>Category</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Chronic hepatitis B</td>
</tr>
<tr>
<td></td>
<td>Chronic hepatitis C</td>
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<tr>
<td>Toxins</td>
<td>Alcohol</td>
</tr>
<tr>
<td></td>
<td>Autoimmune hepatitis</td>
</tr>
<tr>
<td>Cholestatic</td>
<td>Primary biliary cirrhosis</td>
</tr>
<tr>
<td></td>
<td>Primary sclerosing cholangitis</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Nonalcoholic steatohepatitis</td>
</tr>
<tr>
<td></td>
<td>Hemochromatosis</td>
</tr>
<tr>
<td></td>
<td>Wilson's disease</td>
</tr>
<tr>
<td></td>
<td>α1-Antitrypsin deficiency</td>
</tr>
<tr>
<td>Vascular</td>
<td>Budd-Chiari syndrome</td>
</tr>
<tr>
<td></td>
<td>Cardiac cirrhosis</td>
</tr>
<tr>
<td></td>
<td>Veno-occlusive disease</td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>Hepatitis A</td>
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<tr>
<td></td>
<td>Hepatitis B</td>
</tr>
<tr>
<td></td>
<td>Hepatitis C</td>
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</tbody>
</table>

Cirrhosis of the liver is the convergent final pathway for patients with progressive chronic liver disease. It is characterized histologically by extensive porto-portal and porto-central fibrosis with the presence of regenerative nodules [5]. The development of fibrosis is triggered by inflammation resulting in cytokine-mediated activation of hepatic stellate cells, which results in fibrogenesis [6]. Although the rate of the development of cirrhosis varies widely depending on a number of factors, an estimate of 15% to 20% following 10 to 30 years’ duration of infection is an approximate rate for the most common liver diseases encountered in the United States.

Common physical examination findings associated with cirrhosis include muscle wasting, spider telangiectasia, and splenomegaly (Table 2) [7]. However, many patients with chronic liver disease exhibit no signs or symptoms prior to the development of cirrhosis or even decompensated disease.

**Table 2. Common Physical Examination Findings Associated with Cirrhosis**

- Muscle wasting
- Spider telangiectasia
- Splenomegaly
- Ascites
- Variceal hemorrhage
- Hepatic encephalopathy

With the patient’s history of HCV infection with a likely duration of infection of 25 to 30 years, spider telangiectasia, and thrombocytopenia, the clinical suspicion for underlying cirrhosis is high. The diagnosis is confirmed by typical findings of cirrhosis and portal hypertension on computed tomography.

**What clinical findings point to a diagnosis of cirrhosis in this patient?**

**What is the natural history of cirrhosis?**

The natural history of cirrhosis is complex and variable. The best prognostic indicator of risk is the development of complications related to portal hypertension: ascites, variceal hemorrhage, and hepatic encephalopathy. The development
and severity of these complications are inversely proportional to survival.

The Child-Turcotte-Pugh (CTP) classification system (Table 3) is a well-validated model for measuring hepatic reserve. Based on a composite score, patients are classified into class A, class B, or class C, which correspond with 30%, 50%, and 90% reductions in hepatic function, respectively [8]. Patients with CTP class A cirrhosis are considered well compensated while those with CTP class B or C disease have developed decompensated disease. The natural history of these 2 clinical entities are widely divergent; compensated cirrhotics have an overall 10- to 20-year survival rate greater than 80% [9]. In contrast, survival once decompensated is 50% at 5 years [10].

Unfortunately, patients are frequently clinically asymptomatic until they have experienced severe reductions in hepatic function, and presentation with CTP class B or C cirrhosis is not uncommon. At this point, the disease may progress rapidly, limiting opportunities for successful intervention. Whenever possible, primary care providers and community gastroenterologists involved in the care of patients with chronic liver disease should be aware of this phenomenon to allow ample time for consideration of liver transplant evaluation where appropriate. Frequently, psychosocial issues and medical comorbidities are identified during the evaluation that must be addressed prior to the patient being deemed a suitable transplant candidate. These issues are often time-consuming and patient demise can occur in this phase of management.

- What lifestyle issues must be addressed in the patient with cirrhosis?

### Table 2. Physical Signs of Liver Disease

<table>
<thead>
<tr>
<th>Abdominal</th>
<th>Musculoskeletal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatomegaly (followed by small liver span)</td>
<td>Muscle wasting</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>Palmar erythema</td>
</tr>
<tr>
<td>Ascites</td>
<td>Dupuytren's contracture</td>
</tr>
<tr>
<td>Dilated abdominal vasculature</td>
<td>Neurologic</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Endocrine</th>
<th></th>
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<tbody>
<tr>
<td>Gynecomastia</td>
<td>Altered mental status</td>
</tr>
<tr>
<td>Testicular atrophy</td>
<td>Asterixis</td>
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<tr>
<th>HEENT</th>
<th></th>
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<tbody>
<tr>
<td>Scleral icterus</td>
<td>Spider telangiectasia</td>
</tr>
<tr>
<td>Xanthelasma</td>
<td>Jaundice</td>
</tr>
<tr>
<td>Parotid swelling</td>
<td>Bruising</td>
</tr>
<tr>
<td>Kayser-Fleischer rings</td>
<td>Leukonychia</td>
</tr>
<tr>
<td>Fetor hepaticus</td>
<td>Linear scratch marks</td>
</tr>
</tbody>
</table>

### Table 3. Child-Turcotte-Pugh Classification System

<table>
<thead>
<tr>
<th>Measure</th>
<th>1 Pt</th>
<th>2 Pts</th>
<th>3 Pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encephalopathy</td>
<td>None</td>
<td>Grade 1–2</td>
<td>Grade 3–4</td>
</tr>
<tr>
<td>Ascites</td>
<td>Absent</td>
<td>Slight</td>
<td>Moderate</td>
</tr>
<tr>
<td>Total bilirubin, mg/dL</td>
<td>&lt; 2</td>
<td>2–3</td>
<td>&gt; 3</td>
</tr>
<tr>
<td>Albumin, g/dL</td>
<td>&gt; 3.5</td>
<td>2.8–3.5</td>
<td>&lt; 2.8</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>&lt; 1.7</td>
<td>1.7–2.3</td>
<td>&gt; 2.3</td>
</tr>
</tbody>
</table>

Scoring: 5–6 points, Class A; 7–9 points, Class B; 10–15 points, Class C.

### Alcohol Use

Many lifestyle issues need to be addressed in cirrhotic patients. The most urgent of these is the need for discontinuation of all alcohol consumption on a permanent basis. Individuals with a history of significant alcohol intake, generally more than 2 to 3 drinks daily for men or 1 to 2 drinks daily for women for longer than 10 years, are more likely to progress to cirrhosis [11] and continued use increases risk of decompensation of liver disease. It is frequently helpful to review with the patient that all forms of alcohol have equal potential for causing harm and that routine beer drinking, although it may be viewed as less harmful and more socially acceptable among some peer groups, carries the same risk of hepatotoxicity as other types of alcohol consumption. Persons with a history of extensive use as previously noted or those for whom drug and alcohol use has caused serious difficulty with employment or personal relationships or resulted in legal consequences should be referred for substance use evaluation and treatment as needed.

### Diet

Because cirrhosis is a catabolic state, patients are at increased risk for nutritional deficiency and should be counseled to eat a well-balanced diet, with emphasis on avoiding protein restriction to limit loss of muscle mass [12]. Supplementation with a daily multivitamin without iron is also recommended. Patients with cirrhosis should receive annual influenza vaccinations, the pneumococcal vaccine, and complete vaccinations for hepatitis A and B if previous immunity is not documented [13,14]. To avoid the risk of concomitant fatty liver disease, cirrhotic patients should avoid weight gain. Patients who are overweight or obese should work toward gradual weight loss to achieve a normal body mass index through nutritional modification and increased moderate physical activity [14–16]. Additionally, patients with diabetes should be encouraged to maintain tight control of their glucose levels. In patients with dyslipidemia and fatty liver disease, the use of statin medications is safe and well tolerated and may be beneficial in patients with liver disease [17].
Currently, most liver transplant centers consider body mass index as a part of transplant evaluation due to increased surgical morbidity and mortality in obese patients.

**Case Continued**

The patient is instructed to continue to avoid all alcohol use, vaccination against hepatitis A and B are recommended, and he is instructed to maintain a healthy weight by following a low-fat, low-sodium diet with regular physical activity. The patient reports that exercise sometimes causes joint and muscle pain, and he wants to know which pain medications he can use to control the pain.

• What are principles of analgesic use in the cirrhotic patient?

The topic of analgesic use frequently surfaces during management of the patient with cirrhosis. It is important to balance the issues of pain management with competing concerns such as substance abuse, decreased hepatic clearance leading to accumulation in the blood and resulting in supratherapeutic effects and potentially worsening hepatic encephalopathy, use of medications that have known potential for hepatotoxicity, and agents that contribute to coagulopathy, potentially elevating bleeding risk for patients who are already at increased risk. Primary pain management options include opioids and acetaminophen.

In general, opioids are hepatically cleared and can accumulate in patients with decreased hepatic clearance function, and this can contribute to hepatic encephalopathy. Abdominal pain and constipation are also common side effects of opioids in this patient population. If used, reduced dosing and increased intervals between doses may be required.

Nonsteroidal anti-inflammatory drugs should be avoided in cirrhotic patients due to their ability to block prostaglandin synthesis. In the hyperdynamic circulatory states associated with portal hypertension, prostaglandins play an important role in maintaining renal blood flow and decreases in prostaglandins may precipitate acute renal failure and hepatorenal syndrome. Nonsteroidal anti-inflammatory drugs also carry a small but real risk of gastrointestinal bleeding, which may carry increased risk for cirrhotic patients due to underlying coagulopathy.

Due to the risk of acute liver failure in certain situations [18], there is a misconception that acetaminophen should be avoided in all patients with liver disease. Acetaminophen has been shown to be safe in patients with cirrhosis at recommended doses up to 4 g per day [19,20]. Many hepatologists recommend limiting acetaminophen to 2 g daily in patients with cirrhosis who are not drinking alcohol to stay well below toxic levels. Patients should be instructed to look for the presence of acetaminophen in combination medication products to avoid unintentional increases in acetaminophen consumption. Patients who are actively drinking, however, should be instructed to avoid acetaminophen use [20]. Chronic alcohol use leads to nutritional deficiency and induction of cytochrome P450 enzymes that result in the production of hepatotoxic metabolites, which can lead to severe hepatic injury. In healthy patients who do not drink alcohol, even those with cirrhosis, this pathway only plays a minor role in acetaminophen metabolism and does not impact the safety of acetaminophen use. Eating a small amount of food with each dose may further reduce risk.

• What complications can occur in cirrhosis?

Patients with chronic liver disease and well-compensated cirrhosis of the liver transition to decompensation at rates similar to the rate at which patients with chronic liver disease progression to cirrhosis—approximately 15% to 20% in 10 to 20 years [21]. Ascites is the most common clinical manifestation to herald the onset of decompensation, but variceal hemorrhage is the most deadly [22]. Both ascites and gastroesophageal varices are related to the development of portal hypertension. Increased pressure within the portal system is multifactorial, resulting from an increase in intrahepatic resistance. The 2 primary etiologies are (1) the development of fibrosis and regenerative nodules in the liver, resulting in architectural distortion and (2) increases in intrahepatic vasoconstriction secondary to decreased in nitric oxide production in the liver. These mechanisms are thought to contribute approximately 75% and 25%, respectively, to the reduction in blood flow to the liver [23]. In addition to increased resistance in the liver, hyperdynamic circulation results from vasodilation in the splanchnic arterioles due to increased nitric oxide levels, which in turn results in increased blood flow into the portal vein.

**Varices**

The hepatic portal venous gradient (HPVG) is the best clinical measurement of portal pressure. Normal portal pressure is less than 5 mm Hg; pressures of 10 to 12 mm Hg are required for the development of varices [24]. Varices develop in patients with cirrhosis at the rate of 8% per year [25], and approximately 50% of patients have varices when cirrhosis is diagnosed. The presence of varices correlates with the severity of cirrhosis; only 40% of patients with CTP class A cirrhosis have varices, while 85% of patients with CTP class C cirrhosis have varices. Esophagogastroduodenoscopy (EGD) is the gold standard for diagnosis of varices, but nonendoscopic
CIRRHOSIS

Table 4. Recommendations for Surveillance of Varices in Patients with Cirrhosis

Screening esophagogastroduodenoscopy (EGD) should be performed at the diagnosis of cirrhosis
Patients with no varices should have EGD repeated in 2–3 years for surveillance
If small varices are detected, EGD should be repeated in 1 year
Therapy should be initiated with nonselective β blocker or endoscopic band ligation if large or medium varices are detected

Diagnosis may play a role in the future. The risk of variceal bleeding correlates with the size of the varices; small varices (<5 mm) carry a bleeding risk of less than 5% annually, while the risk of spontaneous bleeding is threefold higher among patients with large varices (>5 mm). The rate of progression from small to large varices is also 8% per year. Significant mortality is associated with variceal hemorrhage, with a 20% mortality rate within 6 weeks of the initial event [26–28].

In a large meta-analysis consisting of 11 trials and nearly 1200 patients, nonselective β blockers (nadolol, propranolol, timolol) have been shown to reduce initial episodes of variceal bleeding (primary prophylaxis) in patients with large varices by more than 50% compared with patients receiving placebo (30% vs. 14%) [29]. Other studies have not shown significant reductions in bleeding rates among lower-risk patients with small varices. Additionally, the administration of nonselective β blockers does not prevent the development of small varices in patients with normal HVPG [25]. Further, nearly 50% of patients in the treatment group experienced moderate to severe adverse events compared with 32% in the placebo group. Serious symptomatic adverse events were reported in 18% of the treatment group compared with 6% in controls. Based on these findings, the American Association for the Study of Liver Diseases (AASLD) and the American College of Gastroenterology issued and endorsed recommendations regarding surveillance for and initial management of gastroesophageal varices in cirrhotic patients without previous bleeding episodes [30]. These are summarized in Table 4.

Ascites

The mainstays of management of ascites are careful adherence to a 2000 mg/day sodium restriction and the use of combinations of potassium-sparing and loop diuretics with monitoring of renal function and electrolytes [31]. Fluid restriction is generally not helpful in these patients and should be avoided except when serum sodium falls below 125 mEq/L.

Hepatocellular Carcinoma

Primary liver cancer, hepatocellular carcinoma (HCC), is an important concern in patients with cirrhosis of the liver. During the last decade, the incidence of HCC has been rising in the United States and throughout the world [32]. This increase correlates with the rising number of individuals with underlying cirrhosis of the liver from viral hepatitis, primarily hepatitis C, with infection 20 years or longer. The incidence of HCC in patients with cirrhosis due to HCV is between 2% to 8% per year, 2.5% per year in HBV-related cirrhosis, and although less well studied is 1.5% per year or greater in cirrhosis from most other etiologies of chronic liver disease. This is important, as screening for HCC has been proposed to be cost-effective given an incidence of greater than 1.5% annually [33,34].

Outcomes in patients with symptomatic tumors at the time of diagnosis are extremely poor, with 0% to 10% survival at 5 years [35]. This is in contrast to a greater than 50% 5-year cure rate seen in patients undergoing hepatic resection or liver transplantation [35]. Other treatment modalities, including transarterial chemoembolization, radio-frequency ablation, and percutaneous ethanol injection, may be offered as a bridge to transplantation or as palliation in those patients not eligible for transplantation. Although randomized trials comparing these alternate therapies with liver transplantation do not exist, survival benefit and potential cure in small tumors may occur and these procedures are frequently offered off-protocol.

The goal of surveillance for HCC is to identify tumors at an earlier stage. This should improve patient outcomes by detecting tumors that fall within criteria designed to identify patients with a lower risk of recurrent HCC after liver transplantation. Patients fulfilling these criteria are given preference in transplant listing, which reduces the waiting time prior to transplantation. The criteria consider the size and number of tumors present. Patients with solitary tumors less than 5 cm in diameter or those with no more than 3 lesions all 3 cm in diameter or smaller are considered for preferential transplant listing. Currently at most centers, patients exceeding these criteria are excluded from transplantation due to increased risk of recurrence after transplantation. Outcomes are being examined at selected centers to see if selection criteria can be expanded without decreasing survival. It is important to balance cost-effectiveness of surveillance with the need to detect tumors at a stage that is early enough to allow for intervention with improved outcomes. With this in mind, the AASLD has issued guidelines for surveillance of HCC in patients at high risk for development of HCC, including patients with cirrhosis [36]. Patients at high risk should be screened at 6- to 12-month intervals; this is based on the estimated doubling time of HCC, which is incompletely understood. The recommended screening modality is ultrasound, given that sufficient local operator expertise is available. Alpha fetoprotein level measurement is frequently used in combination with ultrasound but should not be
used alone for surveillance unless ultrasound is not available. Similarly, computed tomography scanning, although very useful as a diagnostic tool, is not recommended as a primary screening tool due to a number of issues including cost, increased false-positive rates, and cumulative radiation exposure for patients undergoing surveillance every 6 to 12 months.

Surgical Risk

Surgery in the cirrhotic patient is associated with increased morbidity and mortality. Whenever possible, referral to surgeons experienced in the management of cirrhotic patients is preferable to minimize complications. The CTP classification scheme is useful in determining mortality associated with elective surgical procedures. Abdominal surgery is associated with worse outcomes than nonabdominal procedures. CTP class A, B and C patients have expected mortality rates of approximately 10%, 30%, and 80%, respectively. The MELD (Model for End-stage Liver Disease) score has also been validated as predictor of 30- and 90-day mortality following surgery [37]. Efforts for optimizing medical management, including good nutrition prior to surgery, should be undertaken. Overall, surgery is well tolerated in patients with CTP class A cirrhosis but care must be taken to evaluate and manage decompensation in the weeks following surgery.

Case Continued

At this time, the patient has been vaccinated against hepatitis A and B and continues to avoid alcohol use. Screening EGD is normal, and screening for HCC is ongoing. Plans for initiation of antiviral treatment with pegylated interferon and ribavirin-based therapy are being considered in consultation with his gastroenterologist at this time.

- When should patients be evaluated for possible liver transplantation?

As previously outlined, a proportion of patients with cirrhosis will have progression of disease and will require evaluation for liver transplantation. Organs for liver transplantation in the United States are allocated by geographic region and specific requirements for eligibility vary somewhat among transplant centers. Primary care providers should be familiar with requirements of their local referral centers. Adequate social support resources and abstinence from alcohol, tobacco, marijuana, and other illicit drug use are commonly required for evaluation, and prior management of these issues can reduce time needed during evaluation.

In February 2002, the United Network for Organ Sharing (UNOS) adopted a modified version of the MELD score as the method for allocating deceased donor livers to adult patients in the United States [38]. The MELD score is a validated prognostic model that predicts 3-month survival in patients with end-stage liver disease awaiting liver transplantation. Three laboratory parameters are included in the model: serum creatinine, serum bilirubin, and international normalized ratio of prothrombin time. Based on these values, a MELD score between 6 (mild disease) and 40 (most severe disease) is assigned. MELD scores of 15 or less, 30, and 40 are associated with 3-month survival rates of approximately 95%, 65%, and 10%, respectively. Currently, patients with HCC meeting criteria that predicts acceptable outcome following surgical resection are awarded MELD exception points to adjust for their increased risk of mortality from their disease [39]. The AASLD has recommended that patients be referred for consideration for liver transplant evaluation if they have a CTP score of 7 or greater, a MELD score of 10 or greater, or at the onset of variceal bleeding, ascites, or hepatic encephalopathy [40].

**SUMMARY**

Cirrhosis of the liver is a leading cause of death in the United States. Patients with cirrhosis are also at increased risk of morbidity resulting from their disease. Diligence towards the identification and management of underlying chronic liver disease must be sought to prevent or slow progression of cirrhosis. Lifestyle modification, therapeutic interventions against other illnesses, and referral to gastroenterologists/hepatologists for the initiation of disease-specific screenings for HCC and

**Table 5. Summary of Recommendations for Cirrhotic Patients**

<table>
<thead>
<tr>
<th>Identification of at-risk patients</th>
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<tbody>
<tr>
<td>Cirrhosis can be silent or subtle; historical, laboratory, and examination findings should be sought in patients with known or suspected chronic liver disease</td>
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<table>
<thead>
<tr>
<th>Primary care interventions and lifestyle modification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoidance of all alcohol consumption</td>
</tr>
<tr>
<td>Tobacco cessation</td>
</tr>
<tr>
<td>Healthy dietary and physical activity habits</td>
</tr>
<tr>
<td>Achieve or maintain healthy weight</td>
</tr>
<tr>
<td>Daily multivitamin without iron</td>
</tr>
<tr>
<td>Vaccination against hepatitis A and B infection</td>
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</table>

<table>
<thead>
<tr>
<th>Gastroenterology/hepatology interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat underlying disease as appropriate</td>
</tr>
<tr>
<td>Initiate screening for hepatoma</td>
</tr>
<tr>
<td>Esophagogastroduodenoscopy for surveillance of esophageal and gastric varices</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Consider evaluation for liver transplant listing as appropriate</th>
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esophageal varices are important components of the role that primary care providers play in management of this commonly encountered patient (Table 5). Unfortunately, the initiation of all of these measures will not prevent progression to liver transplant in all patients and thus primary care providers must recognize the appropriate timing for referral and be ready to collaborate in the management of the often complex medical needs of their patients with end-stage liver disease.

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References


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CME EVALUATION: Primary Care Management of the Patient with Cirrhosis

DIRECTIONS: Each of the questions below is followed by several possible answers. Select the ONE lettered answer that is BEST in each case and circle the corresponding letter on the answer sheet.

1. When should screening for hepatocellular carcinoma be instituted in patients with chronic hepatitis C infection?
   A. At the diagnosis of hepatitis C
   B. At the diagnosis of cirrhosis
   C. At the diagnosis of ascites
   D. Screening is not indicated

2. Which of the following lifestyle modifications should be considered in patients with well-compensated cirrhosis of the liver?
   A. Discontinuation of all alcohol use
   B. Following a low-sodium diet
   C. Smoking cessation
   D. Maintaining a healthy body mass index through diet and moderate physical activity
   E. All of the above

3. Which of the following analgesics should be avoided in cirrhotic patients?
   A. Acetaminophen
   B. Oxycodone
   C. Methadone
   D. Ibuprofen

4. What is the most common complication resulting from portal hypertension in patients with cirrhosis?
   A. Esophageal varices
   B. Hepatic encephalopathy
   C. Ascites
   D. Hepatocellular carcinoma

5. In a cirrhotic patient, all of the following preventive health measures are appropriate EXCEPT
   A. Protein-restricted diet to prevent development of hepatic encephalopathy
   B. Vaccination against hepatitis A and B
   C. Esophagogastroduodenoscopy for surveillance of varices
   D. Discontinuation of alcohol use
   E. Acetaminophen use up to 2 g in 24 hours in patients who do not drink alcohol
EVALUATION FORM: Primary Care Management of the Patient with Cirrhosis

Participants may earn 1 credit by reading the article named above and correctly answering at least 70% of the accompanying test questions. A certificate of credit and the correct answers will be mailed within 6 weeks of receipt of this page to those who successfully complete the test.

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3. A   B   C   D
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2. This article was fair, balanced, free of commercial bias, and fully supported by scientific evidence.
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3. Please rate the clarity of the material presented in the article.
   __ Very clear  __ Somewhat clear  __ Not at all clear

4. How helpful to your clinical practice was this article?
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   ______________________________________________________

6. What topics would you like to see presented in the future?
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