### Summary

#### Goals
- Diagnose F3/F4 Cirrhosis early and screen for HCC*  
- Diagnose and treat complications  
- Delay decompensation  
- Early identification of patients who are appropriate for Palliative Care/Hospice – Ensure POLST done

#### Alerts
- Abdominal Pain: Consider Spontaneous Bacterial Peritonitis (SBP)  
- Mental status changes – consider encephalopathy  
- Hematemesi/Melena  
- Fever- Consider SBP  
- Oliguria/Anuria  
- Rapid weight gain or loss – fluid gain/loss

### Diagnostic Criteria for Cirrhosis and Decompensated Cirrhosis

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Details</th>
</tr>
</thead>
</table>
| **Cirrhosis** | Is best predicted by these findings:  
  - Ascites (likelihood ratio for cirrhosis [LR] 7.2)  
  - Platelet count < 160,000/mm³ (LR 6.3)  
  - Severe thrombocytopenia often precedes other manifestations  
  - Spider angiomata on physical exam (LR 4.3) |
| **Cirrhosis diagnosed** (liver fibrosis stage 4) | Is one or more of the following:  
  - Imaging: hepatic ultrasound, CT, MRI  
  - Calculations: FIB4 online calculator  
  - Procedure: liver biopsy, transient elastography (FibroScan™)  
  - Physical exam |
| ** Decompensated Cirrhosis** | Is defined by the presence of:  
  - Ascites  
  - Hepatic encephalopathy (HE)  
  - Hepatocellular carcinoma (HCC)  
  - Hepatorenal syndrome  
  - Hepatopulmonary syndrome  
  - Child-Pugh class B and C (See page 5)  
  - SBP  
  - Variceal bleeding |

### Evaluation

#### Complete clinical history and physical exam
- History: Especially risk factors for hepatitis; symptoms of significant liver disease: hematochezia, melena, hematemesis, weight gain, abdominal distension  
- Physical Exam: Particularly mental status changes, skin changes, hepatosplenomegaly, spider angiomata, weight changes, hematemesis, jaundice and edema in addition to usual review of symptoms components  
- Pay attention to the presence of complications of liver disease (i.e., ascites, esophageal varices, hepatic encephalopathy, SBP) indicative of decompensated cirrhosis

#### Medication List Review
- Avoid hepatotoxins and chronic NSAID use  
- Multiple drugs have altered kinetics in patients with severe liver disease; dose alterations frequently required

#### Lab/Diagnostics
- CBC, CMP, PT/INR, hepatitis serologies, HIV testing  
- Cirrhosis/F4: EGD (baseline) to screen for esophageal varices: follow-up based on clinical findings  
- F3 and F4 fibrosis: US to screen for HCC every 6 months  
-AFP not recommended as the only tool to screen for HCC

### Treatment (see pages 6-11)

- Vaccinations: influenza annually, pneumococcal vaccines, if not immune, consider vaccinating for: HAV, HBV
- Medications or other therapies based on specific patient findings (See below and pages 6-11)  
  - Ascites: optimize volume management (diuretics and salt restriction); consider midodrine for refractory ascites  
  - Esophageal varices: determine if nonselective beta-blocker is indicated; order baseline EGD with follow-up as needed  
  - Hepatocellular carcinoma diagnosed: obtain consultation  
  - Hepatic encephalopathy: optimize lactulose and minimize potential for exacerabation  
  - Hepatitis C: consider treatment if no HCC and prognosis > 1 year – See CCHCS Hepatitis C Care Guide  
  - Liver transplantation: consult with CME or Regional DME for potential transplant candidates  
  - SBP: antibiotic therapy and prophylaxis

### Monitoring (see pages 6-11)

#### Follow-up visit
- Chronic Care visit as clinically indicated, typically at least every 180 days, but more frequently if unstable or decompensated cirrhosis  
- Monitor changes in: mental status, weight, vital signs, skin

#### Labs
- Consider CBC, CMP, PT/INR annually or more frequently as indicated (especially if the patient has ascites and is on diuretics)

#### Ultrasound
- Every 6 months (HCC screening) for F3 and F4 fibrosis

#### EGD
- EGD (F4 only) at baseline, then as recommended by Gastroenterologist (GI), generally within 2-3 years (see page 9 for more details)

---

**Table of Contents**

- Liver Disease Algorithm ........................................ 2  
- Evaluation .................................................. 3-5  
- Child-Pugh and MELD ......................................... 5  
- Treatment: General Management .......................... 6  
- Managing Complications .................................. 7-11  
- Ascites ......................................................... 7  
- Refractory Ascites ........................................... 8  
- SBP .............................................................. 8  
- Hepatic Encephalopathy ..................................... 9  
- Esophageal Varices .......................................... 9  
- HCC ............................................................ 10  
- HPS ............................................................ 10  
- HRS ............................................................ 10  
- Liver Mass Evaluation ...................................... 11  
- References ................................................... 11  
- Medications .................................................. 12-13  
- Patient Education .......................................... PE-1  
- Patient Education (Spanish) ................................ PE-2

---

CCHCS Care Guide: Advanced Liver Disease

April 2019

Liver Disease Algorithm

Patient Presents with Liver Disease

1. Complete a thorough History/Physical Evaluation Examination
2. Review medications (avoid hepatotoxins and chronic NSAIDs)
3. Order baseline labs and HCV, HBV, HIV
4. Update vaccinations

Determine existence and staging of liver fibrosis/cirrhosis by determining FIB4 Score.

If FIB4 score < 1.45:
• Not likely cirrhotic
• Patient Education: no ETOH use, weight management
• Labs and Chronic Care F/U annually or as clinically indicated

If FIB4 score 1.45-3.25:
• Fibroscan (or liver biopsy) needed to determine fibrosis level

Cirrhosis present
• EGD baseline for varices (See page 9 for info on varices)
• Abd US for HCC q 6 month
• Consider Serial AFP q 6 month with Abd US
• Consider POLST

*Interpreting FibroScan Results

<table>
<thead>
<tr>
<th>FibroScan Result (kPa)</th>
<th>≤ 7.0</th>
<th>&gt; 7.0</th>
<th>≥ 9.5</th>
<th>≥ 12.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equivalent Stage of Fibrosis (HCV)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F0-F1</td>
<td>Absent or mild fibrosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F2</td>
<td>Significant fibrosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F3</td>
<td>Severe fibrosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F4</td>
<td>Cirrhosis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Above table refers to HCV only; if other condition present, refer to appropriate tables

Decompensated Cirrhosis?

• Complete/Update POLST
• Patient Education: no ETOH use, weight management, avoid NSAIDs
• Close Chronic Care F/U with labs as clinically indicated

Severe fibrosis present
• Abdominal US for HCC q 6 months
• Consider Serial AFP q 6 month with Abd US
• Labs and Chronic Care F/U annually or as clinically indicated
• Patient Education: avoid ETOH use, weight management; avoid chronic NSAIDs

Poor 6 mos Prognosis??

• Discuss goals of care and code status with patient;
• Complete/Update POLST
• Patient Education: no ETOH use, weight management, avoid NSAIDs
• Close Chronic Care F/U with labs as clinically indicated

***Poor 6 month prognosis, if any of the following are present
• Recurrent SBP
• Recurrent Variceal Bleed
• Refractory Ascites
• MELD ≥ 20
• Child-Pugh C
• Poor functional status

**Interpreting FibroScan Results

• Child-Pugh ≥ 7 (≥ 6 for HIV/HCV co-infection)
• Encephalopathy present
• Ascites
• H/O SBP
• Variceal Hemorrhage
• Hepatopulmonary/Hepatorenal Syndrome (HPS/HRS)
During the initial evaluation (and subsequent evaluations), it is important to recognize that liver disease is likely not the only significant chronic medical condition in your patient. Coexisting medical conditions especially heart failure, chronic kidney disease/end stage renal disease, COPD, dementia, diabetes, HIV and malignancy can significantly alter the treatment plan, as well as the overall prognosis of your patient.

- Patients often present late in their disease progression and can already be cirrhotic at initial diagnosis.
- If cirrhosis is present, it is important to identify the patients with decompensated cirrhosis early.
- The overall prognosis, surveillance plan, and management of patients with decompensated cirrhosis is vastly different.
- Obesity has been shown to predict worsening of liver fibrosis, and cirrhosis decompensation.

### History, Physical Exam and Medication Review

#### History:
Especially noting risk factors for hepatitis (alcohol, substance abuse, and tattoos); symptoms of significant liver disease (see below). Obtain vaccination history (for HAV, HBV) and family history.

#### Review of systems (ROS):
Ask about anorexia, weight loss, weakness, fatigue, muscle cramps, and easy bruising. Patients with **decompensated liver cirrhosis** can present with: jaundice, dark urine, pruritus, hematemesis/melena/hematochezia, abdominal distension, lower extremity edema, confusion, or sleep disturbances.

#### Physical Exam:
Pay particular attention to mental status changes, skin changes, hepatosplenomegaly, spider angiomata, jaundice, edema, and distended abdomen with shifting dullness and/or positive fluid wave.
- Other physical examination findings may include: gynecomastia, palmar erythema, digital clubbing, and asterixis.
- Check weight and monitor for weight changes.

Note: Ascites and spider angiomata are strong predictors for the presence of cirrhosis:
- Ascites: likelihood ratio for cirrhosis (LR 7.2)
- Spider Angiomata: (LR 4.3)

#### Review Medication List:
Review on a continuing basis. Be aware of hepatotoxic medications. Avoid hepatotoxins and chronic NSAIDs if liver disease is present. Discontinue or dose adjust medications as clinically indicated. Discontinue beta-blockers in patients with decompensated disease.

### Laboratory Evaluation

#### Lab/Diagnostics:
Laboratory abnormalities may include elevated serum bilirubin, abnormal aminotransferases, elevated alkaline phosphatase/gamma-glutamyl transpeptidase, a prolonged prothrombin time/elevated international normalized ratio (INR), hyponatremia, hypoalbuminemia, and thrombocytopenia.

- **At baseline:** hepatitis serologies (anti-hepatitis A IgM [for acute infections], hepatitis B surface antigen, anti-hepatitis B core IgM, anti-hepatitis C virus antibody), HCV RNA and genotype (if infected) and HIV
- **Generally at least annually:** CBC, CMP, PT/INR, Test for HCV RNA, and other diagnostic labs as clinically indicated

Note: **Thrombocytopenia** is a strong predictor for the presence of cirrhosis: Platelet count < 160,000/mm³ (LR 6.3)

- **Annually:** Calculate Fibrosis-4 (FIB4): Based on age, AST, ALT, platelets. (Can use online calculator, value is on Quality Management HCV Registry)
- Treat the patient with FIB4 > 3.25 as cirrhotic

\[
FIB4 = \frac{[\text{Age}(y) \times \text{AST(U/L)}]}{[\text{PLT}(10^9/L) \times \text{ALT(U/L)}^{1/2}]} 
\]

<table>
<thead>
<tr>
<th>FIB4</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1.45</td>
<td>unlikely to have significant fibrosis</td>
</tr>
<tr>
<td>1.45-3.25</td>
<td>not accurate at this range; other staging method required</td>
</tr>
<tr>
<td>&gt; 3.25</td>
<td>likely to have advanced fibrosis/cirrhosis (Fibrosis stage 3–4)</td>
</tr>
</tbody>
</table>

Online calculator: [http://www.hepatitisc.uw.edu/page/clinical-calculators/fib-4](http://www.hepatitisc.uw.edu/page/clinical-calculators/fib-4)

---

Summary

For Patients with FIB4 scores of 1.45 to 3.25: Obtain FIBROSCAN™

FibroScan™ uses transient elastography to measure liver stiffness. The shear wave velocity has been correlated with stages of fibrosis in HCV patients in the following manner:

<table>
<thead>
<tr>
<th>FibroScan Result (kpa)</th>
<th>≤ 7.0</th>
<th>&gt; 7.0</th>
<th>≥ 9.5</th>
<th>≥ 12.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equivalent Stage of Fibrosis</td>
<td>F0-F1</td>
<td>F2</td>
<td>F3</td>
<td>F4</td>
</tr>
</tbody>
</table>

Metavir Fibrosis:
- F0-F1: Absent or mild fibrosis
- F2: Significant fibrosis
- F3: Severe fibrosis
- F4: Cirrhosis

Screening:
- F3 and F4 Fibrosis Patients: Ultrasound every 6 mos to screen for HCC (AFP not recommended for HCC screening).
- F4 Fibrosis/cirrhosis Patients: EGD (baseline) to screen for esophageal varices.

Other Causes of Liver Disease

There are numerous causes of liver disease that can result in cirrhosis, either by causing chronic hepatic inflammation or cholestasis. The most common causes of cirrhosis in the United States are hepatitis C, alcoholic liver disease, and cryptogenic causes.

Other Causes of Liver Disease:
- Nonalcoholic Fatty Liver Disease: Diagnosis of exclusion and associated with obesity, HTN, DM, and dyslipidemia. Fatty liver on imaging
- Wilson Disease: Young patient with a family history. Can have neurologic and psychiatric symptoms, thrombocytopenia, and anemia. Check serum ceruloplasmin level and copper concentration
- Hereditary Hemochromatosis: Family history and associated with DM, cardiomyopathy (45% of deaths due to HCC). Check transferrin saturation
- Autoimmune Hepatitis: Initial labs: antinuclear ab, anti-smooth muscle ab, ALKM-1, AMA, IgG level. Watch for other autoimmune liver diseases such as Primary Biliary Cirrhosis and Primary Sclerosing Cholangitis
- Drug Induced/Ingested Toxins: Acetaminophen, herbal supplements, mushroom poisoning, and antibiotics (Amoxicillin-Clavulanate)

Severity of Cirrhosis/Prognosis

Cirrhosis represents a late stage of progressive hepatic fibrosis characterized by distortion of the hepatic architecture and the formation of regenerative nodules. It is generally considered to be irreversible in its advanced stages. In earlier stages, specific treatments aimed at the underlying cause of liver disease may improve or even reverse cirrhosis.2

- Compensated Cirrhosis: median survival is > 12 years
  - Patients with varices but who have not developed variceal bleeding are considered to have compensated cirrhosis, though their prognosis is worse than that of patients who have compensated cirrhosis without varices (3.4 versus 1.0 percent one-year mortality rates).
- Decompensated Cirrhosis: median survival was ≤ 6 months (and a Child-Pugh score ≥ 12 or a MELD score ≥ 21
  - In addition, patients with decompensated cirrhosis who had been hospitalized with an acute liver-related illness (e.g., variceal hemorrhage or spontaneous bacterial peritonitis) had a median survival of ≤ 6 months if the Child-Pugh score was ≥ 12 or the MELD score was ≥ 18.
  - Tools to help assess severity of disease (and therefore prognosis) include the Child-Pugh and MELD score (see page 5).
- Risk Factors for Poor 6 Month Prognosis: Recurrent SBP, Recurrent Variceal Hemorrhage, Refractory Ascites, MELD ≥ 20, Heart failure and/or other significant co-morbid conditions, any hospitalization within 30 days or > 2 within 60 days, poor functional status, HCC/other cancer, HPS/HRS, dialysis patient, Child Score > 10 (Class C).

---

3 Goldberg, E. Cirrhosis in adults: Overview of complications, general management, and prognosis, Up to Date June 2018.
EVALUATION CONTINUED

Severity of Cirrhosis/Prognosis Continued

Decompensated Cirrhosis is defined by the presence of any of the following:
- Ascites, HE, HCC, Variceal bleeding, Hepatorenal syndrome, Hepatopulmonary syndrome, Child-Pugh score ≥ 7 (and ≥ 6 in patient with HIV) and/or SBP.

Recognize the poor prognosis and discuss end of life preferences with the patient. Obtain a POLST and identify/document the patient’s preferred surrogate decision-makers using an Advance Directive.

Child-Pugh

Child-Pugh is a tool used to help assess prognosis in the patients with liver disease. Variations in the timing and subjectivity inherent in the scoring (e.g., in grading ascites or encephalopathy) are its major limitations.

<table>
<thead>
<tr>
<th>CHILD-PUGH POINTS</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encephalopathy</td>
<td>None</td>
<td>Grade 1-2</td>
<td>Grade 3-4 (or chronic)</td>
</tr>
<tr>
<td>Ascites</td>
<td>None</td>
<td>Mild/Moderate (diuretic-responsive)</td>
<td>Severe (diuretic-refractory)</td>
</tr>
<tr>
<td>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>INR</td>
<td>&lt; 1.7</td>
<td>1.7-2.3</td>
<td>&gt; 2.3</td>
</tr>
</tbody>
</table>

Encephalopathy Grading:
- Grade 1: Mild confusion, anxiety, restlessness, fine tremor, slowed coordination
- Grade 2: Drowsiness, disorientation, asterixis
- Grade 3: Somnolent but arousable, marked confusion, incomprehensible speech, incontinence, hyperventilation
- Grade 4: Coma, decerebrate posturing, flaccidity

Model for End-Stage Liver Disease (MELD)

MELD: Originally derived from the patients with cirrhosis undergoing elective Transjugular Intrahepatic Portosystemic Shunt (TIPS) procedures to predict 3 month mortality post procedure. Adopted by the United Network for Organ Sharing in 2002 for the prioritization of the patients waiting for liver transplants.

- Note: There are some conditions associated with chronic liver disease that may result in impaired survival but are not directly accounted for in the MELD scoring system; such as: HCC, Hepatopulmonary Syndrome; therefore these should not be the only tools used for accessing overall prognosis.

- MELD formula:
  - \( MELD = 3.78 \times \ln[\text{serum bilirubin (mg/dL)}] + 11.2 \times \ln[\text{INR}] + 9.57 \times \ln[\text{serum creatinine (mg/dL)}] + 6.43 \)
  - \( \ln = \text{natural logarithm} \)

- MELD Score Three Month Mortality:

<table>
<thead>
<tr>
<th>MELD Score</th>
<th>3 Month Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 or more</td>
<td>71.3% mortality</td>
</tr>
<tr>
<td>30-39</td>
<td>52.6% mortality</td>
</tr>
<tr>
<td>20-29</td>
<td>19.6% mortality</td>
</tr>
<tr>
<td>10-19</td>
<td>6.0% mortality</td>
</tr>
<tr>
<td>&lt; 9</td>
<td>1.9% mortality</td>
</tr>
</tbody>
</table>

- Online Calculator: [https://optn.transplant.hrsa.gov/resources/](https://optn.transplant.hrsa.gov/resources/)
# Treatment: General Management

<table>
<thead>
<tr>
<th>Major Pillars in Management</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Slow or reverse the progression of liver disease</strong></td>
<td>Some chronic liver diseases respond to treatment even when the liver disease has progressed to cirrhosis. Specific therapies directed against the underlying cause of the cirrhosis should be instituted (such as HCV).</td>
</tr>
<tr>
<td><strong>Prevent superimposed insults to the liver and minimize risks for acute exacerbations</strong></td>
<td>Vaccinations: influenza, pneumococcal vaccines; if not immune: HAV, HBV. HCV, HBV infections. Alcohol cessation.</td>
</tr>
<tr>
<td><strong>Identify medications that require dose adjustments, discontinuation, or should be avoided entirely</strong></td>
<td>Avoidance of hepatotoxins. Continued review of medication lists.</td>
</tr>
<tr>
<td><strong>Manage symptoms and laboratory abnormalities (for ascites, encephalopathy and variceal bleeding; see pages 7 and 9)</strong></td>
<td>Muscle Cramps: Patients with cirrhosis may experience muscle cramps which can be severe. It is important to confirm that the muscle cramps are related to cirrhosis, check electrolyte levels and replace if low, treat if symptoms persist. Umbilical Hernias: Umbilical hernias pose a management dilemma in the patients with cirrhosis, since they often develop in the patients with severe liver disease and ascites who are at high risk of complications with surgical repair. Asymptomatic hernias should be managed conservatively. Ruptured/incarcerated hernias should be referred for immediate repair. Hyponatremia: Common problem in the patients with advanced cirrhosis; the pathogenesis of hyponatremia is directly related to the hemodynamic changes and secondary neurohumoral adaptations that occur in the setting of cirrhosis, resulting in an impaired ability to excrete ingested water. The severity of the hyponatremia is related to the severity of the cirrhosis. Free water restriction is often not necessary unless serum Na is less than 125mmol/L.</td>
</tr>
<tr>
<td><strong>Prevent, identify, and treat complications of cirrhosis</strong></td>
<td>Patients should be monitored for the development of complications and when possible, steps should be taken to prevent their development. Presence of any complication is a sign of worsening long-term prognosis. See pages 7-11 for treatment of the complications of cirrhosis.</td>
</tr>
<tr>
<td><strong>Determine the appropriateness and optimal timing for liver transplantation</strong></td>
<td>Consult with CME or Regional CME.</td>
</tr>
<tr>
<td><strong>Identify and treat/manage other chronic illnesses</strong></td>
<td>For example: diabetes, heart failure, CKD/ESRD, HCV, HIV.</td>
</tr>
<tr>
<td><strong>Patient Education</strong></td>
<td>It is important to ensure your patient understands that there are things they can do, or refrain from doing, that can help protect their liver from further damage. Alcohol and other illicit substance use should be stopped. Healthy diet: sodium restriction 2gm daily. Weight Management: patients should be encouraged to participate in lifestyle modification activities to improve their health; these include eating healthy and engaging in physical activity regularly.</td>
</tr>
<tr>
<td><strong>Early identification of the patients with poor prognosis</strong></td>
<td>Develop an overarching management plan that takes into account the patient’s cirrhosis, other comorbid conditions, and his/her wishes for care towards the last year of life. This discussion should be continued on a regular basis and include (but not limited to): Code Status, goals/end of life care, and completion of the POLST form.</td>
</tr>
</tbody>
</table>
Students should be instructed when appropriate imaging study or physical exam
Differential diagnosis: ascites may be caused by conditions other than liver disease (or in addition to liver disease); about 15% are due to heart failure, nephrotic syndrome, cancer, tuberculosis, or other conditions
Paracentesis (if indicated under ultrasound guidance) for diagnosis may be indicated; especially with new onset ascites

<table>
<thead>
<tr>
<th>ROUTINE TESTS ON ASCITIC FLUID</th>
<th>OPTIONAL TESTS</th>
<th>UNUSUAL TESTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell count and differential</td>
<td>Glucose level</td>
<td>Tuberculosis smear and culture</td>
</tr>
<tr>
<td>Albumin level</td>
<td>LDH level</td>
<td>Cytology</td>
</tr>
<tr>
<td>Total protein level</td>
<td>Gram stain</td>
<td>Triglyceride level</td>
</tr>
<tr>
<td>Culture in blood culture bottles</td>
<td>Amylase level</td>
<td>Bilirubin level</td>
</tr>
</tbody>
</table>

- Serum to Ascitic Albumin Fluid Gradient (SAAG) > 1.1 indicates portal hypertension with 97% accuracy; SAAG < 1.1 suggests ascites from other causes. To calculate SAAG, the serum albumin should be drawn the same day as the paracentesis. (SAAG = Serum Albumin minus Ascitic Albumin level)
- Patient may require large volume paracentesis (> 5 liters). Albumin infusion (between 6-8 g of albumin per liter of fluid removed) is recommended.

**Focus should be on Diuretic and Diet Therapy**

- **Diuretics:** Start at low dose and titrate up. Optimal ratio spironolactone to furosemide is 100 mg to 40 mg;
  - Spironolactone: 100 mg/day or 50 mg/day for patients ≤ 50kg WITH
  - Furosemide: 40 mg/day (or 20 mg/day for patients ≤ 50 kg)
  - Increase doses of both agents every 3-5 days if tolerated
  - Usual Daily Max dose: Spironolactone 400 mg, furosemide 160 mg
  - Alternative agents: Amiloride starting at 5-10 mg/day can be used as substitute for spironolactone if side effects (e.g., gynecomastia) noted
- **Dietary sodium restriction:** 2 gm/day (consider dietary consult or handout)
- **Free Water Restriction is often not necessary unless serum Na is less than 125mmol/L**
- **Avoid:** alcohol, ACE inhibitors, ARBs, NSAIDs

**MONITORING**

- Monitor patient weight and abdominal girth
- Monitor for other complications (i.e., encephalopathy, peritonitis, systemic or localized infections, worsening creatinine, worsening urine output, worsening respiratory status
- Obtain CMP every one to two months or as indicated for patients on diuretics; adjust treatment as indicated

Managing Complications (continued)

REFRACTORY ASCITES

### DIAGNOSIS
- Presence of ascites (See previous page)
- Patients are considered refractory ONLY if they fail max dose (or cannot tolerate) diuretic therapy, AND if on 2gm/day sodium restriction diet

### EVALUATION/ TREATMENT AND PROPHYLAXIS
- Discontinue beta-blockers
- Consider Oral midodrine starting at 5 mg three times daily; recommended dosing is 7.5mg 3x daily
- Serial paracentesis
- TIPS (may precipitate encephalopathy)
- Continue diuretic therapy and dietary sodium restriction

Refractory Ascites carries a 21% 6 month mortality rate. Recommend POLST, End of Life, and Goals of Care discussion with your patient.

### MONITORING
- Monitor the patient weight and abdominal girth
- Monitor for other complications (i.e., encephalopathy, peritonitis, systemic or localized infections, worsening creatinine, worsening urine output, worsening respiratory status)
- Obtain CMP every one to two months or as indicated for patients on diuretics; adjust treatment as indicated

SPONTANEOUS BACTERIAL PERITONITIS (SBP)

### DIAGNOSIS
SBP may present without obvious symptoms or may present with fever, abdominal pain, altered mental status. Any or all symptoms may be subtle or absent

Diagnosis: ascitic fluid with ≥ 250 PMNs/ml and/or positive culture without other obvious causes of peritonitis (such as: abdominal abscess, perforated bowel, patients on peritoneal dialysis)
(Most often E. coli, or klebsiella; can be streptococcus or rarely staphylococcus)

### TREATMENT / PROPHYLAXIS
Evaluate and transfer to a higher level of care if clinical suspicion is present.

Treatment:
- Stop beta-blocker prophylaxis indefinitely if history of SBP
- Empiric IV antibiotic with Cefotaxime while waiting for lab results if clinical suspicion present (fever, abdominal pain, altered mental status)
- Usually in hospital with IV Cefotaxime. Use Quinolone for patients with allergy to β-lactamase antibiotics, unless Quinolone used for prophylaxis. Avoid aminoglycosides (due to nephrotoxicity)
- Treatment duration usually 5 days, unless unusual organism, unusual presentation or associated bacteremia which requires extended treatment

Prophylaxis:
All patients with history of prior SBP, significant ascites, or impaired renal function should be treated indefinitely with:
- Ciprofloxacin 500 mg daily or Sulfamethoxazole/Trimethoprim DS one tablet daily. Weekly dosing is not recommended.
- Patients with cirrhosis who are hospitalized with GI bleed should receive antibiotic prophylaxis: either IV Cefotaxime or Sulfamethoxazole/Trimethoprim DS for seven days

Prophylaxis also recommended during GI bleed

### MONITORING
Observe for return of fever, abdominal pain, change in mental status
Follow-up on culture results

---

### Managing Complications (continued)

#### Hepatic Encephalopathy (HE)\(^1\)

| **Diagnosis** | • Presentation may vary from mild subclinical changes in mentation to overt psychiatric symptoms to deep coma  
• Presenting symptoms can include confusion, decreased attention, mental slowing, asterixis, irritability, sleep disorder, lethargy, or unresponsiveness |
| **Treatment / Prophylaxis** | Correct precipitating cause(s):  
• Precipitating factors: GI bleed, infection (including SBP), blood transfusion, HCC, excess protein intake, constipation, dehydration, drugs, poor adherence to medications, and portohepatic shunts  
**Treatment** overt HE:  
• Lactulose - give lactulose when patient is able to take medications orally for treatment and prophylaxis  
Recommended starting dose: 30 ml po BID - TID  
  • Consider NA or DOT administration for recurrent symptoms in selected cases (e.g., nonadherence). Titrate dose to no more than three to four bowel movements per day  
• Rifaximin - only after optimized lactulose treatment. Recommended dose: rifaximin 550 mg BID  
• Patients with significant mental status changes should be referred to a higher level of care  
• Consider lactulose enemas when patient is comatose (inpatient setting only)  
**Prophylaxis:** After 1st episode: lactulose  
  After 2nd episode: add rifaximin (NF) to lactulose\(^3\) |
| **Monitoring** | Medication adherence, bowel movement frequency, mental status, functional status  
Be aware of other causes of altered mental status (i.e., localized and systemic infections, electrolyte imbalance, renal failure, and worsening of other chronic illnesses) |

#### Esophageal Varices\(^2\)

| **Diagnosis** | • Baseline EGD to screen for varices indicated when cirrhosis is first diagnosed  
• EGD to diagnose when varices suspected |
| **Treatment / Prophylaxis** | No varices seen on EGD: beta-blockers not recommended for “pre-primary prophylaxis” (i.e., to prevent EV)  
All “beta-blockers” recommendations are for Non-Selective Beta-Blockers (propranolol and nadolol)  
**Primary Prophylaxis:**  
• Small varices that haven't bled:  
  • If Child-Pugh class A and no red wales on EGD - can use surveillance EGD in place of beta-blockers  
  • If Child-Pugh class B/C or red wales on EGD - consider beta-blockers  
• With beta-blockers: Do not lower systolic BP < 90 or heart rate < 55  
• Medium/large varices that haven't bled:  
  • Non-selective beta-blockers or esophageal variceal ligation (EVL)  
  • If bleeding risk is not high, beta-blockers preferred over EVL  
  • With large varices, EVL preferred  
• These agents are not recommended for primary prophylaxis: nitrates, combination beta-blockers and EVL, shunt therapy, or sclerotherapy  
**Secondary Prophylaxis:**  
• Patients who survive an EV bleed should receive both beta-blockers and EVL  
• Repeat EGD every 1-2 weeks until varices obliterated, then every 1-3 months, then every 6-12 months for surveillance  
• Consider TIPS if bleeding recurs despite combination beta-blockers and EVL  
• Sclerotherapy is not recommended for secondary prophylaxis  
• Consider TIPS in Child-Pugh class A/B patients with recurrent bleeding despite beta-blockers and EVL |
| **Monitoring** | Cirrhosis without varices on EGD → repeat EGD within 3 years  
Small varices and no beta-blocker used → repeat EGD within 2 years  
Small/medium/large and beta-blockers maximized: consider EGD within 2-3 years  
• Medium/large and EVL used: → repeat EGD every 1-2 weeks until varices obliterated, then every 1-3 months, then every 6-12 months  
• Decompensated cirrhosis: → repeat EGD at time of diagnosis and annually or more often as indicated |

---

### HEPATOCELLULAR CARCINOMA (HCC)<sup>1</sup>

**DIAGNOSIS**
- Screen for HCC with ultrasound every 6 months for **Metavir F3 and F4 patients**
- Evaluate mass on ultrasound with contrast enhanced imaging study imaging (dynamic triphasic or quadriphasic CT or MRI with gadolinium)
- Hepatic mass identified on contrast enhanced imaging (See liver mass evaluation page 11)
- Biopsy, as indicated (See liver mass evaluation page 11)
- Consultation recommended with a specialist knowledgeable in the diagnosis and management of HCC

**TREATMENT / PROPHYLAXIS**
Classification and diagnosis complements the Barcelona Clinic Liver Cancer staging and treatment criteria:
- Very early to early stage disease - may be cured with ablation, resection, or liver transplant
- Intermediate stage - usually treated with chemoembolization
- Advanced stage - sorafenib (trade name NexAVAR<sup>®</sup>)
- Terminal stage - Child-Pugh C with liver biopsy evidence of stage 3-4 disease - initiate supportive care, discuss end of life goals, comfort focused care indicated, POLST

**MONITORING**
- Monitor change in tumor size with imaging, new symptoms

### HEPATOPULMONARY SYNDROME (HPS)<sup>2</sup>

**DIAGNOSIS**
- Symptoms:
  - Platypnea: dyspnea that worsens when sitting up from supine
  - Orthodeoxia: arterial deoxyhemoglobin saturation decrease >5% when sitting up from supine
- Diagnosis:
  - Contrast-enhanced echocardiography
  - Pulmonary angiography
  - Nuclear scanning to view intravascular pulmonary dilatations

**TREATMENT / PROPHYLAXIS**
- There are no effective treatments for HPS
- Long term oxygen therapy for hypoxemia
- Transplant may be a treatment option; if recommended, consult with CME or Regional DME

**MONITORING**
- Breathing symptoms as described
- Pulse oximetry as indicated

### HEPATORENAL SYNDROME (HRS)<sup>3</sup>

**DIAGNOSIS**
- Progressive rise in serum creatinine
- Urine sediment often normal with no or minimal proteinuria (less than 500 mg per day)
- Very low rate of sodium excretion (i.e., urine sodium concentration less than 10 mEq/l)
- Oliguria

**TREATMENT / PROPHYLAXIS**
- There are two forms of HRS based on the speed of onset of renal failure:
  - **Type I HRS** is more serious and generally develops in less than two weeks with serum creatinine increasing two fold to > 2.5 mg/dl and Clcr falling to below 20 ml/min
  - **Type II HRS** is less severe renal insufficiency associated with diuretic resistant ascites. Serum creatinine level increases over days to weeks
  - Hepatorenal syndrome is usually treated in a hospital setting as it has high mortality rate and requires specialty care.

**MONITORING**
- Serum creatinine, urine output

---


<sup>2</sup>Lange, P.A., Hepatopulmonary syndrome: Natural history, treatment, and outcomes. UpToDate: March 2015.


<sup>4</sup>Runyon, B.A., Hepatorenal syndrome. UpToDate: March 2015.

MANAGING COMPLICATIONS (CONTINUED)

<table>
<thead>
<tr>
<th>LIVER MASS EVALUATION¹</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIAGNOSIS</strong></td>
</tr>
<tr>
<td>Lesions &lt; 1 cm</td>
</tr>
<tr>
<td>• Repeat ultrasound every 3 months for 24 months</td>
</tr>
<tr>
<td>• If lesion remains &lt; 1 cm, resume every 6 months US screening</td>
</tr>
<tr>
<td>• Not feasible to definitively diagnose liver lesions &lt; 1 cm</td>
</tr>
<tr>
<td>Lesions &gt; 1 cm or multiple masses and at least 1 lesion is &gt; 1 cm</td>
</tr>
<tr>
<td>• Perform contrast enhanced imaging study such as dynamic triphasic or quadriphasic CT or MRI with gadolinium</td>
</tr>
<tr>
<td>• Look for arterial hypervascularization and venous or delayed washout as diagnostic of HCC (See HCC page 10)</td>
</tr>
<tr>
<td>• If CT/MRI is not typical for HCC, a biopsy is needed to diagnose HCC</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TREATMENT / PROPHYLAXIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment of HCC: (See page 10)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MONITORING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imaging</td>
</tr>
</tbody>
</table>


**REFERENCES**

# CCHCS Care Guide: Advanced Liver Disease

## Summary

### Indication: Ascites

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing</th>
<th>Adverse Effects*/Interactions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide (Lasix®)</td>
<td></td>
<td><strong>Electrolyte imbalances:</strong> hypokalemia, possibly severe, hypomagnesemia, hypocalcemia, hyperglycemia, hyperuricemia, metabolic alkalosis</td>
<td><strong>Once a patient with cirrhosis develops clinically apparent ascites, it is unlikely to resolve without specific treatment (e.g., a sodium-restricted diet [2000 mg/day] and diuretics)</strong></td>
</tr>
<tr>
<td>Tablet: 20 mg, 40 mg</td>
<td>Recommended starting dose: 40 mg by mouth daily (with 100 mg spironolactone)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recommended starting dose for patients ≤ 50 kg: 20 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increase every 3-5 days as needed up to 160 mg furosemide with 400 mg spironolactone</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Keep the ratio of 100 mg spironolactone and 40 mg furosemide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone (Aldactone®)</td>
<td></td>
<td><strong>Electrolyte imbalances:</strong> hyperkalemia, possibly severe, hypocalcemia, hypomagnesemia</td>
<td><strong>Once a patient with cirrhosis develops clinically apparent ascites, it is unlikely to resolve without specific treatment (e.g., a sodium-restricted diet [2000 mg/day] and diuretics)</strong></td>
</tr>
<tr>
<td>Tablet: 25 mg, 50 mg, 100 mg</td>
<td>Recommended starting dose: 100 mg by mouth daily with food with 40 mg furosemide</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recommended starting dose for smaller patient ≤ 50 kg: 50 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increase every 3-5 days as needed up to 400 mg spironolactone with 160 mg furosemide</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Renal Impairment: use with caution</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hepatic Impairment with cirrhosis and ascites: initiate spironolactone in the hospital</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiloride (Midamor®)</td>
<td></td>
<td><strong>Hyperkalemia</strong> (black box warning)</td>
<td><strong>Can be used in place of spironolactone in cases of painful gynecomastia; less effective than spironolactone in patients with cirrhosis</strong></td>
</tr>
<tr>
<td>Tablet: 5 mg</td>
<td>Recommended starting dose: 5-10 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Max dose: 40 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Renal dosing CrCl: 10-50 mL/min - reduce dose 50%; however, amiloride should generally be avoided due to risk of hyperkalemia. If use is necessary, monitor potassium closely. &lt; 10 mL/min - contraindicated</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Indication: Refractory Ascites

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosing</th>
<th>Adverse Effects*/Interactions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midodrine</td>
<td></td>
<td><strong>May decrease cardiac output, bradycardia, supine hypertension</strong></td>
<td><strong>Reserve for patients with true Refractory Ascites or patients unable to tolerate increased diuretic dosing, or on max dose of diuretics and sodium restriction at 2 g/day</strong></td>
</tr>
<tr>
<td>Tablet: 2.5 mg, 5 mg, 10 mg</td>
<td>Recommended dose: Start at 5 mg TID. Titrate dose by 2.5 mg for each dose every 24 hours (Max dose 7.5 mg TID) to achieve an increase in systolic blood pressure of approx. 10-15 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Last dose should be taken at least 4 hrs. before bedtime</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Renal Impairment: Start at 2.5 mg TID</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Bold = Formulary

*See prescribing information for complete description of dosing, adverse effects and drug interactions. Hypersensitivity to the medication, medication class or a component of the formulation is a contraindication to use of the drug.*

The cost scale $-$ $$ $$ $$ $$ $$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.
**CCHCS Care Guide: Advanced Liver Disease**

### SUMMARY

<table>
<thead>
<tr>
<th>INDICATION: Hepatic Encephalopathy (HE)</th>
<th>Dosing</th>
<th>Adverse effects*/ Interactions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lactulose (Enulose®)</strong></td>
<td>• Recommended dose: 30-45 ml by mouth, two to three times daily&lt;br&gt;• Titrate dose to achieve two to three soft bowel movements per day</td>
<td>• Abdominal discomfort, cramping, flatulence, nausea, vomiting&lt;br&gt;• With excessive dosing: electrolyte imbalance, diarrhea, metabolic acidosis</td>
<td>• Patients with cirrhosis are often malnourished and protein restrictions are associated with increased mortality, so patients with hepatic encephalopathy should generally not have their protein intake restricted&lt;br&gt;• For patients who have not improved within 48 hours or who can not take lactulose consider treatment with rifaximin</td>
</tr>
<tr>
<td><strong>Soln: 10 g/15ml</strong>&lt;br&gt;$$$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Rifaximin (Xifaxan®)</strong></td>
<td>• Recommended dose: 550 mg by mouth, twice daily&lt;br&gt;• Indicated for breakthrough HE despite optimized lactulose dosing</td>
<td>• Bacterial or fungal superinfection may occur with prolonged use, including C difficile-associated diarrhea&lt;br&gt;• Abdominal pain, nausea, ascites, headache, fatigue, peripheral edema, angioedema, pruritus, rash</td>
<td>• Avoid use in patients with diarrhea and fever or blood in stool&lt;br&gt;• Use with caution in patients with severe hepatic impairment (Child-Pugh C)</td>
</tr>
<tr>
<td><strong>Tablet: 550 mg</strong>&lt;br&gt;$$$$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### INDICATION: Unresectable Hepatocellular Carcinoma (HCC)

| Sorafenib (Nexavar®) | Recommended dose: 400 mg (200 mg x 2) by mouth, twice daily until clinical benefit ceases or unacceptable toxicity occurs<br>• Administer without food (at least 1 hour before or 2 hours after a meal) | • Hand-foot syndrome, severe skin reactions, SJS, TENS, erythema multiforme<br>• GI perforation, pancreatitis, renal failure<br>• MI, CHF, hypertensive crisis, QT prolongation<br>• Rhabdomyolysis<br>• Intestinal lung disease<br>• Skin carcinoma<br>• Hypokalemia, hypoalbuminemia, AST/ALT elevations, hypocalcemia, hypophosphatemia, anemia, lymphopenia, thrombocytopenia, prolonged INR<br>• Headache, fatigue, weight loss<br>• Diarrhea, constipation, abdominal pain, N/V<br>• Anorexia, stomatitis, sensory neuropathy<br>• Alopecia, desquamating rash<br>• Co-administration of certain drugs may need to be avoided or dosage adjustments may be necessary | • Sorafenib is a multikinase inhibitor acting on the vascular endothelial growth factor receptor (VEGFR), among others<br>• Findings from the SHARP trial, showed that sorafenib significantly prolonged survival over supportive care alone in patients with advanced HCC<br>• Oncology co-management required<br>• Blood pressure should be monitored weekly for the first 6 weeks of sorafenib therapy, then monitored and treated as needed thereafter as clinically indicated<br>• Sorafenib has not been studied in patients with severe hepatic impairment (Child-Pugh C) |
| **Tablet: 200 mg**<br>$$$$$ | | | |

### INDICATION: Portal Hypertension (Esophageal Varices Non-Selective Beta-Blockers)

| Nadolol (Corgard®) | Recommended starting dose: 40 mg daily<br>• Titrate to reduce resting heart rate by 25%, but not below 55 beats/min, and to reduce systolic BP, but not below 90 mmHg<br>Renal dose CrCl:<br>31-50 mL/min dose Q24-36h<br>10-30 mL/min dose Q24-48h<br>< 10 mL/min dose Q40-60h | • Side effects common to non-selective beta-blockers:<br>• Cardiac: CHF, heart block, bradycardia, hypotension, impaired myocardial contractility, angina exacerbation or MI with abrupt d/c<br>• Pulmonary: bronchospasm<br>• Other: fatigue, dizziness, Raynud’s phenomenon, pruritus, diarrhea, constipation, nausea<br>• Hypersensitivity reaction<br>• Rash including SJS, TENS (propranolol) | • Approximately half of patients with cirrhosis have esophageal varices, and one-third of all patients with varices will develop variceal hemorrhage<br>• The risk of hemorrhage has been related to the size and appearance of the varices, as well as the degree of hepatic dysfunction<br>• Nonselective beta blockers lower portal pressure and reduce the risk of first bleeding in patients with esophageal varices<br>• D/C with refractory ascites |
| **Tablet: 20 mg, 40 mg, 80 mg**<br>$$$$-$$ | | | |
| Propranolol (Inderal®) | Recommended starting dose: 20 mg twice daily<br>• Titrate to reduce resting heart rate by 25%, but not below 55 beats/min, and to reduce systolic BP, but not below 90 mmHg | | |
| **Tablet: 10 mg, 20 mg, 40 mg, 60 mg**<br>$-$$ | | | |

*Bold = Formulary

*See prescribing information for complete description of dosing, adverse effects and drug interactions. Hypersensitivity to the medication, medication class or a component of the formulation is a contraindication to use of the drug.

The cost scale $-$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.
WHAT IS CIRRHOSIS? (Sir-o-sis)

- Cirrhosis is when a healthy liver becomes damaged by scars and lumps
- Cirrhosis is usually caused by viral infections (like hepatitis B and C), alcoholism, or fatty liver disease
- You can live several years with cirrhosis if you get medical care

HOW DO YOU KNOW IF YOU HAVE CIRRHOSIS?

You may not know if you have cirrhosis because you may not have any symptoms.

Your doctor will determine if you have cirrhosis by examining you and performing tests if needed.

You could have cirrhosis if you have:
- Swollen legs or belly
- Yellow colored skin
- Frequent nosebleeds
- Red palms
- A tendency to bruise easily
- Unexplained weight loss or weight gain
- Belly pain
- Frequent infections
- Trouble thinking clearly or confusion

END STAGE LIVER DISEASE – CIRRHOSIS: WHAT YOU SHOULD DO

- Eat from the CDCR “heart healthy” diet
- Stay away from high salt, high fat food from the canteen and/or packages
- Get regular exercise unless your health care provider tells you not to
- Get vaccinated for Hepatitis A and B and pneumonia
- Get a yearly flu shot
- Do not drink any alcohol, including pruno, while you are in prison or after release
- Discuss all medications with your health care provider
- Take your medication as directed by your health care provider
- Do not take more than 2000 milligrams a day of acetaminophen (brand name Tylenol®)
- Stay away from NSAID medication like Advil®, Motrin®, or Aleve® unless recommended by your health care provider
- Avoid protein and amino acid supplements
- Avoid iron supplements
- Do not take more than the recommended dose of Vitamins A, D, E, or K

TELL YOUR HEALTH CARE PROVIDER IF YOU HAVE ANY OF THESE SYMPTOMS

- Vomiting blood or what looks like “coffee grounds”
- Feeling sleepy for long periods of time
- Trouble thinking or increasing confusion
- Black tarry stools
- You don’t pee as much as you used to
- Fever
- Problems breathing
¿QUÉ ES LA CIRROSIS?
♦ La cirrosis es cuando se daña un hígado sano a causa de cicatrices y nódulos
♦ Es causada principalmente por infecciones virales (como hepatitis B y C), alcoholismo o la enfermedad del hígado graso
♦ Usted puede vivir varios años con cirrosis si recibe atención médica

¿CÓMO SABER SI TIENE CIRROSIS?
Puede que no sepa que tiene cirrosis porque no presenta ningún síntoma.
Su médico determinará si usted tiene cirrosis al examinarlo y practicarle algunos exámenes, de ser necesario.

Usted podría tener cirrosis si presenta:
♦ Hinchazón en las piernas o el vientre
♦ Piel amarillenta
♦ Hemorragias nasales frecuentes
♦ Palmas de las manos rojas
♦ Tendencia a sufrir de hematomas
♦ Pérdida o aumento de peso sin razón aparente
♦ Dolor abdominal
♦ Infecciones recurrentes
♦ Dificultad para pensar con claridad o confusión

ENFERMEDAD HEPÁTICA EN ETAPA TERMINAL – CIRROSIS: LO QUE USTED DEBE SABER

Base su alimentación en la dieta “corazón sano” del CDCR
Evite los alimentos altos en sal y en grasas y/o las comidas empaquetadas
Practique ejercicio de manera regular a menos que su proveedor de cuidados de la salud le indique algo distinto
Vacúñese contra la Hepatitis A y B y contra la neumonía
Vacúñense anualmente contra la gripe
No ingiera nada de alcohol, incluyendo pruno, mientras esté en prisión ni cuando sea puesto en libertad
Consulte cualquier medicación con su proveedor de cuidados de la salud
Tome sus medicamentos como se los recetó su proveedor de cuidados de la salud
No tome más de 2 gramos de acetaminofén al día (la marca Tylenol®)
Evite los medicamentos antiinflamatorios no esteroideos (NSAID) como el Advil®, Motrin® o Aleve® a menos que se lo recomiende su proveedor de cuidados de la salud
Evite los suplementos de proteínas y aminoácidos
Evite los suplementos de hierro
No tome más de la dosis recomendada de vitaminas A, D, E, o K

AVISE A SU PROVEEDOR DE CUIDADOS DE LA SALUD SI PRESENTA ALGUNO DE ESTOS SÍNTOMAS
♦ Vomita sangre o lo que parece ser deshechos de café
♦ Se siente somnoliento durante largos períodos de tiempo
♦ Dificultad para pensar o confusión creciente
♦ Deposiciones negro alquitrandado
♦ No orina tan seguido como solía hacerlo
♦ Fiebre
♦ Dificultad para respirar