Objectives

**Identify a patient with chronic liver disease or cirrhosis**
- Key Features on Presentation
- Review of Systems / Medical History

**Risk Factors for chronic liver disease**
- Social
- Medical
- Familial

**Physical exam findings of chronic liver disease**

**Interpret and utilize testing appropriately in liver disease**
- Laboratory tests of liver function vs inflammation
- Diagnostic laboratory tests of liver diseases
- Assessing severity of liver disease
- MELD Score / Child’s Pugh
- Non-invasive measures of fibrosis
- Liver Biopsy
- Upper Endoscopy
- Peritoneal Fluid Analysis

**Management of Patients with Cirrhosis**
- Hepatic Encephalopathy
- Ascites
- Spontaneous Bacterial Peritonitis
- Esophageal Varices

**Utilize Scoring Systems for patients with cirrhosis**

**Discuss Complications of Cirrhosis**
- Portal Hypertension
- Hepatocellular Carcinoma
- Decompensated Cirrhosis
- Acute on Chronic Liver Failure
Initial Evaluation of Patient with Suspected Liver Disease

- Does this patient have liver disease?
  - Laboratory Data
  - Imaging
  - Ascites
- Is it acute or chronic? (6 months)
  - Acute
    - Acute Hepatitis
    - Acute Liver Injury
    - Acute liver failure
  - Chronic
    - Chronic Hepatitis
    - Cirrhosis
- What is the cause?
- HPI
  - Symptoms
  - New Medications/Supplements
  - Risk factors or exposures for liver disease
- Past Medical History
  - Metabolic Syndrome
  - Autoimmune Disorders
- Family History
- Medications
- Physical Exam – Stigmata of Chronic Liver Disease
- Laboratory Testing
- Imaging
Patterns of Liver Injury

1. **Acute Hepatitis**
   - Temporary or new onset inflammation of the liver tissue
   - May or may not be symptomatic
   - Can resolve or become chronic

2. **Acute Liver Failure (Fulminant Liver Failure)**
   - Sudden loss of hepatic function in the absence of pre-existing liver disease
   - Can lead to multi-organ failure and death or recovery, depending on the cause
   - Results in severe liver dysfunction and may require liver transplantation

3. **Chronic Hepatitis → Cirrhosis**
   - Inflammation of the liver tissue that lasts at least 6 months
   - Often without symptoms
   - Can progress to cirrhosis
Laboratory Assessment of the Liver

Provide a noninvasive method to screen for liver disease

Markers of Hepatocellular Damage
- Serum Transaminases
  - ALT (Alanine aminotransferase)
  - AST (Aspartate aminotransferase)

Markers of Cholestasis
- Alkaline Phosphatase
- Gamma Glutamyl Transpeptidase
- Bilirubin

Tests of Liver Function
- Prothrombin time
- Albumin
- Cholesterol
- Bilirubin
Aminotransferases

ALT

- Normal lab values range 29-33 IU/mL for males
- 19-25 IU/mL for females
- Primarily present in the liver
  - More specific marker of hepatocellular injury
  - Correlate with degree of abdominal adiposity

AST

- Present in other organs, including cardiac muscle, skeletal muscle, kidney, and brain
- 10-40 IU/mL for males
- 9-32 IU/mL for females

Most laboratories use >2 SD to define abnormal

- The differences in clinical laboratories abnormal is based on the health of the reference population

A “normal” ALT does not exclude liver disease or histologic damage
Liver Injury Tests can be classified by:

1. **Type**
   - Hepatocellular Injury (ALT/AST elevation)
   - Cholestatic Injury (Alk Phos / T.bili elevation)

2. **Acute or chronic**
   - Acute
     - Abrupt onset
     - Less than 6 months but usually < 1 month
   - Chronic
     - Greater than 6 months (arbitrary)

3. **Magnitude**
   - Mild AST/ALT elevations <200 IU
   - Moderate AST/ALT elevations 200-600 U
   - Severe AST/ALT elevations > 600
## Assessing the Severity of Liver Disease

<table>
<thead>
<tr>
<th>Laboratory Testing</th>
<th>MELD Score / Child’s Pugh</th>
<th>Non-invasive measures of fibrosis</th>
<th>Liver Biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tests of Liver Function (PT/INR, T.bilirubin, Albumin)</td>
<td>Prognostic score in Cirrhosis to determine mortality</td>
<td>Determines if advanced fibrosis/cirrhosis is present in the setting of chronic hepatitis</td>
<td>Determine etiology of liver disease</td>
</tr>
<tr>
<td>Liver dysfunction present – either ALI/ALF or Cirrhosis</td>
<td></td>
<td></td>
<td>Determine degree of fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Measure portal pressures</td>
</tr>
</tbody>
</table>
Acute Hepatitis: Etiology

- Viral Hepatitis
  - Hepatitis A, B, C, E
  - CMV, EBV, Adenovirus, HSV
- Excessive Alcohol Intake
  - Alcoholic Hepatitis
- Drug-Induced Liver Injury
- Autoimmune Hepatitis
- Circulatory Dysfunction
  - Ischemia
  - Budd Chiari
  - Portal vein thrombosis
# Chronic Hepatitis: Etiologies

<table>
<thead>
<tr>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B, C, or D (Hep E, rarely)</td>
</tr>
<tr>
<td>Autoimmune hepatitis</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>Wilson's Disease</td>
</tr>
<tr>
<td>Alpha-1 Antitrypsin Deficiency</td>
</tr>
<tr>
<td>Hemochromatosis</td>
</tr>
<tr>
<td>NASH</td>
</tr>
<tr>
<td>NASH</td>
</tr>
<tr>
<td>Alcohol</td>
</tr>
<tr>
<td>PBC</td>
</tr>
<tr>
<td>PSC</td>
</tr>
</tbody>
</table>
## Historical Clues

<table>
<thead>
<tr>
<th>History Component</th>
<th>Disease Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remote history of jaundice</td>
<td>Viral hepatitis</td>
</tr>
<tr>
<td>Medical history of autoimmune diseases</td>
<td>AIH</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>AIH, PBC</td>
</tr>
<tr>
<td>History of liver disease as a newborn</td>
<td>Alpha-1 antitrypsin deficiency</td>
</tr>
<tr>
<td>Family history of liver disease</td>
<td>HBV, hemochromatosis</td>
</tr>
<tr>
<td>History of alcohol abuse, DUI</td>
<td>Alcohol</td>
</tr>
<tr>
<td>History of IVDA, blood transfusion prior to 1990</td>
<td>HCV</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Hemochromatosis, NAFLD</td>
</tr>
<tr>
<td>Components of Metabolic Syndrome</td>
<td>NAFLD</td>
</tr>
<tr>
<td>Medications, CAM therapy</td>
<td>Drug induced liver injury</td>
</tr>
<tr>
<td>Pruritis</td>
<td>PBC</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td>PSC</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Hemochromatosis, HCV</td>
</tr>
</tbody>
</table>
NONINVASIVE MARKERS OF FIBROSIS

- **Serological Assays**
  - **Indirect Markers**
    - APRI
    - Fib-4
    - NAFLD Fibrosis Score
    - ELF
  - **Direct Markers (hepatic matrix metabolism)**
    - Hyaluronic Acid
    - Procollagen type III
    - Metalloproteinases, MMP-1, MMP-2

- **Imaging Methods**
  - Ultrasonography
  - Magnetic Resonance Imaging
  - Elastography
Indirect Measures of Fibrosis

- **APRI**
  
  \[
  \text{APRI} = \left( \frac{\text{AST (Upper Limit of Normal)}}{\text{Platelet Count (10^9/L)}} \right) \times 100
  \]

- **FIB-4**
  
  \[
  \text{FIB-4} = \frac{\text{Age (years)} \times \text{AST (U/L)}}{\text{Platelet Count (10^9/L)} \times \sqrt{\text{ALT (U/L)}}}
  \]

<table>
<thead>
<tr>
<th>Fibrosis</th>
<th>F0-F2</th>
<th>F3-F4</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>APRI &lt; 0.7515</td>
<td>24</td>
<td>2</td>
<td>26</td>
</tr>
<tr>
<td>APRI ≥ 0.7515</td>
<td>14</td>
<td>19</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>21</td>
<td>59</td>
</tr>
</tbody>
</table>
Fibroscan

- **Transient Elastography**
  - Non-invasive method for the assessment of hepatic fibrosis in patients with chronic liver disease
  - Measures liver stiffness
  - Can easily be performed at the bedside or in the outpatient clinic with immediate results and good reproducibility
  - Surface ultrasound probe that delivers a low frequency pulse or shear wave to a small volume of liver tissue under the rib cage
Fibroscan

Liver stiffness cut-offs in chronic liver diseases

Matavir  F0-F1  F2  F3  F4

Fibrosis  Mild  Sign  Severe  Cirrhosis
Cirrhosis: Etiologies

- Alcoholic Liver disease
- Viral Hepatitis
- Non-Alcoholic Liver Disease (NASH)
- Chronic Biliary Obstruction
- Hemochromatosis
- Wilson’s Disease
- Alpha-1 Antitrypsin Deficiency
- Metabolic Disorders
- Drug-Induced Liver Injury
- Autoimmune Liver Disease
- Primary Sclerosing Cholangitis
- Primary Biliary Cholangitis
- Cardiac Cirrhosis (Passive Congestion)
- Budd Chiari
# Physical Clues

<table>
<thead>
<tr>
<th>Physical Exam Findings</th>
<th>Disease Correlates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spider angiomas</td>
<td>Cirrhosis</td>
</tr>
<tr>
<td>Palmar erythema</td>
<td>Cirrhosis</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>Portal hypertension</td>
</tr>
<tr>
<td>Jaundice</td>
<td>Cirrhosis, Biliary obstruction, hemolysis, Gilbert’s</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>Hemochromatosis</td>
</tr>
<tr>
<td>Kayser-Fleisher rings</td>
<td>Wilsons disease</td>
</tr>
<tr>
<td>Emphysema/Lung disease</td>
<td>Alpha-1 antitrypsin deficiency</td>
</tr>
<tr>
<td>Ascites</td>
<td>Portal hypertension, cirrhosis</td>
</tr>
<tr>
<td>Asterixis</td>
<td>Portal hypertension</td>
</tr>
<tr>
<td>Xanthalasma</td>
<td>PBC</td>
</tr>
</tbody>
</table>
Cirrhosis: Clinical Symptoms

- Lower Extremity Edema
- Abdominal Distension (Ascites)
- Gastrointestinal Bleeding (esophageal varices)
- Confusion (Hepatic Encephalopathy)
- Muscle Wasting and loss of muscle mass
- Muscle Cramping
- Gynecomastia
- Jaundice / Scleral Icterus
Mental Status
- Alert, Drowsy, Obtunded
- Parotid Gland Enlargement
- Spider Angiomas
- Scleral Icterus
Palmar Erythema

Duputyren’s contracture
Prognosis of **Compensated Cirrhosis**

- Median survival = 9-12 years
- Majority of deaths: Non-liver related
  - Cardiovascular, strokes, etc
  - Liver-related deaths: HCC
- Predictors of decompensation
  - HVPG: HR 1.11
  - MELD score: HR 1.15
  - Serum albumin: HR .37
Prognosis of Decompensated Cirrhosis

- Median survival = 2 years
- Causes of deaths:
  - Portal HTN
  - Liver failure
  - Sepsis
  - HCC
- Predictors of death
  - Childs-Turcott-Pugh score
  - MELD score
  - Serum sodium
### Child-Turcotte-Pugh Classification for Severity of Cirrhosis

<table>
<thead>
<tr>
<th>Clinical and Lab Criteria</th>
<th>Points*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>None</td>
</tr>
<tr>
<td>Ascites</td>
<td>None</td>
</tr>
<tr>
<td>Bilirubin (mg/dL)</td>
<td>&lt; 2</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>&gt; 3.5</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>&lt;4</td>
</tr>
<tr>
<td>Seconds prolonged or International normalized ratio</td>
<td>&lt;1.7</td>
</tr>
</tbody>
</table>

*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)

- **Class A** = 5 to 6 points
- **Class B** = 7 to 9 points
- **Class C** = 10 to 15 points

---

**Assessing Severity of Cirrhosis:**
**Child’s Pugh Score**
MELD

Estimates the probability of dying over time in patients with chronic liver disease

Accessible

Validated measure of liver disease severity

Objective

Reproducible
MELD = \[(0.957 \times \ln \text{Cr}) + (0.378 \times \ln \text{Bili})
\quad + (1.12 \times \ln \text{INR}) + (0.643) \times 10\]\]

MELD-Na = MELD - Na+ - (0.025 \times \text{MELD} 
\quad \times (140 - \text{Na+})) + 140

<table>
<thead>
<tr>
<th>Variable</th>
<th>Influencing factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin</td>
<td>Increased indirect bilirubin</td>
</tr>
<tr>
<td></td>
<td>Hemolysis</td>
</tr>
<tr>
<td></td>
<td>Blood transfusion</td>
</tr>
<tr>
<td></td>
<td>Drug or sepsis-induced cholestasis&lt;sup&gt;19, 20&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Acute renal failure&lt;sup&gt;21, 22&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Hepatorenal syndrome</td>
</tr>
<tr>
<td></td>
<td>Other causes: shock, hypovolemia, drug-induced nephropathy, and medication-induced nephropathy</td>
</tr>
<tr>
<td>INR</td>
<td>Anticoagulant therapy: warfarin</td>
</tr>
<tr>
<td></td>
<td>Hemodilution&lt;sup&gt;23&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Bleeding-induced coagulopathy&lt;sup&gt;24&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Disseminated intravascular coagulopathy&lt;sup&gt;25&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Malnutrition</td>
</tr>
</tbody>
</table>

INR- International Normalized Ratio
MELD SCORE PREDICTS MORTALITY

Categories: 1 = MELD ≤ 9, 2 = MELD 10 to 19, 3 = MELD 20 to 29, 4 = MELD ≥ 30

Pairwise comparisons
1 vs 2  p<0.0001
1 vs 3  p<0.0001
1 vs 4  p<0.0001
2 vs 3  p=0.004
2 vs 4  p<0.0001
3 vs 4  p<0.0001

Comparison of observed (A) and Predicted (B) curves
Category 1 (≤ 9)  p=0.95
Category 2 (10-19)  p=0.05
Category 3 (20-29)  p=0.34
Category 4 (≥ 30)  p=0.51
Overall  p=0.18
(non-categorized MELD)
Complications of Cirrhosis

<table>
<thead>
<tr>
<th>Related to Portal Hypertension</th>
<th>Unrelated to Portal Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Ascites</td>
<td>• Hepatocellular Carcinoma</td>
</tr>
<tr>
<td>• Hepatic encephalopathy</td>
<td></td>
</tr>
<tr>
<td>• Spontaneous Bacterial Peritonitis</td>
<td></td>
</tr>
<tr>
<td>• Esophageal Varices</td>
<td></td>
</tr>
<tr>
<td>• Hepatorenal Syndrome</td>
<td></td>
</tr>
</tbody>
</table>
Ascites

Bulging Flanks

- Occurs when the weight of ascites is sufficient to push the flanks outwards
- Difficult to distinguish from obesity
- Sensitivity 72-93%
- Specificity 44-70%
What is a SAAG anyway???

SAAG = Serum to Ascites Albumin Gradient (Serum albumin - Ascites albumin)

**Critical concepts:**
- Ascites is either made in the liver sinusoid (hepatic lymph) or not by the liver
- The hepatic sinusoid is designed to keep albumin in the blood
- The diseased hepatic sinusoid becomes even less likely to allow albumin to escape blood into the hepatic lymph (due to capilarization and fibrosis)

**High SAAG = >1.1**
- Aka not much albumin in the fluid
- Ascites is hepatic lymph!!!
- Produced by a sinusoid with:
  - ↑ hydrostatic pressure
  - ↓ oncotic pressure
  - AKA Portal Hypertension

**Low SAAG = <1.1**
- Ascites albumin is close to plasma
- Because the ascites is plasma!
- Extrahepatic source:
  - Malignancy, Infection (TB), Trauma, Pancreatitis etc...
Algorithm for the diagnosis of ascites according to the serum-ascites albumin gradient (SAAG). IVC, inferior vena cava.

Source: Harrison's Principles of Internal Medicine (19th Ed)
MANAGEMENT OF ASCITES IN CIRRHOSIS

Management of Ascites Due to Cirrhosis

1. Treatment of underlying disorder (e.g., alcoholic liver disease, hepatitis B, autoimmune hepatitis)
2. Dietary sodium restriction (less than 2000 mg per day)
3. Diuretic therapy (maintain ratio spironolactone 100 mg: furosemide 40 mg)
4. Therapeutic paracentesis
5. Fluid restriction only if serum sodium <120 mEq/L or symptomatic hyponatremia

Alcohol cessation
Avoid NSAIDs
Sodium restriction (2 g/day)

Oral diuretics
- Starting dose: spironolactone 100 mg/furosemide 40 mg PO QD
- Progressive increase every 3 to 5 days
- Maximum dose: spironolactone 400 mg/furosemide 160 mg PO QD

Frequent large volume paracentesis with albumin infusion

Transjugular intrahepatic portosystemic shunt (TIPS)

Liver transplantation
TIPS (TRANS-JUGULAR INTRAHEPATIC PORTO SYSTEMIC SHUNT)
Peritonitis

- **Spontaneous bacterial peritonitis:**
  - Bacterial translocation across bowel
  - Low opsinization ability of ascites
  - Absolute WBC > 500 cells/mm³ or PMN count >250 cells/mm³
  - Cultures + only 50-75%, usually single organism

- **Secondary peritonitis:**
  - Free perforation, abscess, or ischemic bowel
  - Typically very high PMN count and multiple organisms on gram stain/culture

<table>
<thead>
<tr>
<th>Type</th>
<th>Ascitic Cell Count</th>
<th>Ascites Culture</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sterile</td>
<td>&lt; 250 PMNs</td>
<td>Negative</td>
<td>None</td>
</tr>
<tr>
<td>Spontaneous bacterial peritonitis</td>
<td>≥ 250 PMNs</td>
<td>Monobacterial infection</td>
<td>3rd generation Cephalosporin</td>
</tr>
<tr>
<td>Culture negative neutrocytic ascites</td>
<td>≥ 250 PMNs</td>
<td>Negative</td>
<td>3rd generation Cephalosporin</td>
</tr>
<tr>
<td>Non neutrocytic bacteriocytes</td>
<td>&lt; 250 PMNs</td>
<td>Monobacterial infection</td>
<td>Only if symptomatic or persistently positive culture</td>
</tr>
</tbody>
</table>
| Secondary                   | ≥ 250 PMNs         | Polymicrobial infection  | (1) Base on culture and sensitivities
                                                                              | (2) Identify the source of infection |

(From Ooi, C.H., Mak, S., et al. [2000].)
HEPATO RENAL SYNDROME
HEPATORENAL SYNDROME

1. Cirrhosis with ascites;
2. Serum creatinine > 133 μmol/L (1.5 mg/dl);
3. No sustained improvement of serum creatinine (decrease to a level of 133 μmol/L or less) after at least 2 days of diuretic withdrawal and volume expansion with albumin. The recommended dose of albumin is 1 g/kg of bodyweight per day to a maximum of 100 g/day;
4. Absence of shock;
5. No current or recent treatment with nephrotoxic drugs;
6. Absence of parenchymal disease as indicated by proteinuria > 500 mg/day, microhematuria (>50 red blood cells per high power field) and/or abnormal renal ultrasonography.

HEPATO RENAL SYNDROME

- **HRG Type 1**
  - Rapid progression (≤2 weeks)
  - Triggers: alcohol/drugs
  - UO doubles to >20 ml/hr
  - SCr increased to >135 μmol/L
  - Untreated, mortality ≥ 2 weeks

- **HRG Type 2**
  - Slow progression
  - Triggers: alcohol/drugs
  - UO < 20 ml/hr
  - SCr increased to >135 μmol/L
  - Better prognosis, 4-6 months
Treatment of Hepatorenal Syndrome

Vasoconstrictors and Albumin
(1 g/kg on day one followed by 20-40 g/day)

Terlipressin:
0.5 mg IV every 4 hours; can increase dose to 1 mg/4h and then up to 2 mg/4h

or

Midodrine & Octreotide:
Midodrine: 2.5-7.5 mg p.o. t.i.d with an increase to 12.5 mg t.i.d. daily if needed & octreotide: 100 ug s.c. t.i.d. with an increase to 200 ug t.i.d. if needed

or

Noradrenaline:
0.5-3 mg/hr continuous IV infusion

Duration of therapy: between 1-2 weeks

GOAL: Reduction of serum creatinine < 1.5 mg/dL
### West Haven Criteria for Semi-Quantitative Grading of Mental State

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
</tr>
</thead>
</table>
| 1     | Trivial lack of awareness  
Euphoria or anxiety  
Shortened attention span  
Impaired performance of addition |
| 2     | Lethargy or apathy  
Minimal disorientation of time or place  
Subtle personality changes  
Inappropriate behavior  
Lethargy or apathy |
| 3     | Somnolence to semi-stupor but responsive to verbal stimuli  
Confusion  
Gross disorientation |
| 4     | Coma (unresponsive to verbal or noxious stimuli) |
Asterixis

- Non-rhythmic Asymmetric lapse in voluntary sustained posture of extremities
- Arms Outstretched and Fingers separated by hyperextending the wrists
- Absent at Rest
- Rapid Flex-extension movements at the wrist joint
- Usually Bilateral
- Impaired inflow of afferent information to the brainstem resulting in lapses in posture
Treatment of Hepatic Encephalopathy

- **Lactulose**
  - Nonabsorbable Disaccharides
  - Acts like a probiotic by enhancing growth of certain bacterial strains
  - Low cost making it the preferred agent
  - Reduces pH of the colon, thereby prevents absorption of NH₃
  - Converts NH₃→NH₄⁺ so that it can be excreted

- **Rifaximin**
  - Nonabsorbable antibiotic
  - Equivalent or slightly superior to Lactulose or Neomycin
GASTROESOPHAGEAL VARICES
Treatment of Esophageal Varices

Primary Prophylaxis
- Nonselective beta blocker (nadolol, propranolol)
- Splanchnic Vasoconstriction
- Reduce portal inflow
- Endoscopic band ligation

Treatment of variceal bleeding
- Octreotide
- Endoscopic band ligation/Sclerotherapy
- TIPS

Secondary Prophylaxis
- Endoscopic band ligation
- TIPS
MANAGEMENT OF ESOPHAGEAL VARICEAL BLEEDING
ESOPHAGEAL VARICEAL BAND LIGATION
Liver Transplant: Indications

- Acute Liver Failure
- Cirrhosis
- Metabolic disorders
  - Primary oxaluria
  - Familial amyloidosis
- Hepatobiliary malignancy
  - Hepatocellular carcinoma
  - Cholangiocarcinoma
• Adult patients registered for Liver Transplant in the UNOS Database between 1/2004 and 12/2016

• NASH is the 2nd leading cause for LT overall and the 1st leading cause in women

LIVER TRANSPLANTATION