Cirrhosis

By: Kirsten Dyck, Emily Horn, Kim Jenkins, and Natalie White
Liver Physiology

By: Kim Jenkins
Liver Structure

- Largest gland in the body:
Liver Structure

- There are two main lobes:
  - The right and left lobe
Liver Structure

- The liver gets blood from the hepatic artery and the portal vein.
- The liver has bile ducts. Bile is formed in the liver cells, exits the liver through a series of bile ducts that increase in size as they approach the common bile duct.
Liver Functions

- Integral to most metabolic functions of the body and performs more than 500 tasks.
- The main functions of the liver include:
  - metabolism of CHO, protein, and fat
  - storage and activation of vitamins and minerals
  - formation and excretion of bile
  - conversion of ammonia to urea
  - metabolism of steroids
  - actions as a filter and flood chamber
Functions-Carbohydrate Metabolism

- The liver stores glucose as glycogen (glycogenesis) and then returns it to the blood when glucose levels become low (glycogenolysis).
- The liver also produces “new” glucose (gluconeogenesis) from precursors such as lactic acid, glycogenic amino acids, and intermediates of the tricarboxylic acid cycle.
Functions-Protein Metabolism

- Protein metabolic pathways:
  - Transamination and oxidative deamination are pathways that convert amino acids to substrates that are used in energy and glucose production as well as in the synthesis of nonessential amino acids.
Functions- Fat Metabolism

- Fatty acids from the diet and adipose tissue are converted in the liver to acetyl-coenzyme A by the process of beta-oxidation to produce energy. Ketones are also produced.
- The liver synthesizes and hydrolyzes triglycerides, phospholipids, cholesterol, and lipoproteins.
Functions- Vitamins and Minerals

Storage:
- Storage of all the fat-soluble vitamins in addition to vitamin B\textsubscript{12} and the minerals zinc, iron, copper, and magnesium.

Transportation:
- Hepatically synthesized proteins transport vitamin A, iron, zinc, and copper in the bloodstream.

Activation:
- Carotene is converted to vitamin A, folate to 5-methyl tetrahydrofolic acid, and vitamin D to an active form (25 hydroxycholecalciferol).
Functions - Bile

- The liver forms and excretes bile.
- Bile salts are metabolized and used for the digestion and absorption of fats and fat-soluble vitamins.
- Bilirubin is a metabolic end produce from red blood cell destruction; it is conjugated and excreted in the bile.
Hepatocytes detoxify ammonia by converting to the urea, 75% which is excreted by the kidneys. The remaining goes back to the GI tract.

The liver metabolizes steroids: it inactivates and excretes aldosterone, glucocorticoids, estrogen, progesterone, and testosterone.

It is responsible for the detoxification of drugs and alcohol and other substances.

Acts as a filter and flood chamber by removing bacteria and debris from blood through the phagocytic action of kupffer cells located in the sinusoids and by storing blood backed up from the vena cava as in right heart failure.
Cirrhosis

- Definition: Cirrhosis is a chronic degenerative liver disease in which normal liver cells are damaged and are then replaced by scar tissue.
Incidence of Cirrhosis:

- Hepatitis B: Cirrhosis occurs in 20–30% of chronically infected patients.
- Hepatitis C: Up to 85% of infected patients develop a chronic infection, with 10–20% progressing to cirrhosis.
<table>
<thead>
<tr>
<th>Country</th>
<th>Author, [Ref.]</th>
<th>Study years and population</th>
<th>Diagnosis</th>
<th>Prevalence or incidence rates (95% CI)</th>
<th>Trends in prevalence or incidence rates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denmark</td>
<td>Jepsen et al., [115]</td>
<td>1988-2005; alcoholic cirrhosis: a nationwide population-based, hospital registry study</td>
<td>Histology</td>
<td>In 2001-2005, the alcoholic cirrhosis incidence rates were 22.5 (25.7-17.4) and 11.8 (11.2-12.4) per 100,000 per year for men and women, respectively, and the prevalence rates were 132.6 (130.7-134.5) and 70.1 (68.8-71.5) per 100,000</td>
<td>The alcoholic cirrhosis prevalence and incidence rates for men and women of any age did not change significantly from 1996 to 2005</td>
</tr>
<tr>
<td>France</td>
<td>Poynard et al., [5]</td>
<td>2006-2008; 7463 consecutive subjects aged 40 years or older, seen for a free voluntary screening program in two French Social Security health examination centres, 95% male</td>
<td>Histology</td>
<td>The estimated prevalence of fibrosis was 1.3% (1.1-1.7%) and of cirrhosis was 0.3% (0.2-0.5%)</td>
<td></td>
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<tr>
<td>Sweden</td>
<td>Gunnarsdottir et al., [116]</td>
<td>1994-2003; all patients diagnosed with liver cirrhosis in Gothenburg (600,000 inhabitants)</td>
<td>Histology</td>
<td>The mean annual incidence rate per 100,000 inhabitants in Sweden was 15.3 (± 2.4)</td>
<td>The incidence rate and the proportion of alcohol aetiology were fairly constant over the study period</td>
</tr>
<tr>
<td>UK</td>
<td>Fleming et al., [117]</td>
<td>1992-2001; the UK General Practice Research Database (GPRD), persons aged 25 and over, 58% male</td>
<td>Any diagnostic or therapeutic code for cirrhosis, oesophageal varices or portal hypertension</td>
<td>Crude incidence was 14.55 per 100,000 person years. Prevalence of cirrhosis was an estimated 76.3 per 100,000 population aged over 25 in mid-2001</td>
<td>The cirrhosis incidence increased from 12.05 to 16.99 per 100,000 person years from 1992 to 2001</td>
</tr>
<tr>
<td>UK</td>
<td>Liu et al., [118]</td>
<td>1996-2005; the Million Women Study (an on-going prospective study of 1.3 million United Kingdom women aged 50 and over</td>
<td>ICD-10 diagnosis code K70, K73, or K74</td>
<td>After a mean follow-up of 6.1 years (1996-2005), incidence rate of cirrhosis was 26 per 100,000 per person-years in women</td>
<td></td>
</tr>
</tbody>
</table>
Prevalence of Cirrhosis

- 0.15% or 400,000 people have cirrhosis in the US.
- Approximately 29 million people in Europe have cirrhosis.
Prevalence of Cirrhosis

- Chronic hepatitis B affects 0.5-0.7% of the European population.
- Chronic hepatitis C prevalence was 0.13-3.26% in the last decade.
Prevalence of Cirrhosis

- NAFLD in Europe is 20-30%.
  - 36–44% of NAFLD was found in obese children.
  - 42.6-69.5% in people with type 2 diabetes.
- NAFLD in US is 20%.
  - 74% of NAFLD was found in obese people.
- NASH in US is 3%
Etiology of Cirrhosis

- Hepatitis B
- Hepatitis C
- Nonalcoholic Fatty Liver Disease
- Alcoholic Liver Disease
- Biliary Cirrhosis
- Hemochromatosis
- Wilson’s Disease
- Alpha-1-antitrypsin Deficiency
Hepatitis B and C

- Can lead to chronic and carrier states. Chronic active hepatitis can also develop, which leads to cirrhosis and liver failure.
Nonalcoholic Fatty Liver Disease

- NAFLD is defined as the accumulation of liver fat exceeding 5% of hepatocytes in the absence of significant alcohol intake. (20 g per day for men and 10 g per day for women).
- NAFLD can lead to fibrosis, cirrhosis, and even hepatocellular carcinoma.
Nonalcoholic Fatty Liver Disease

Ranges from steatosis to steatohepatitis.

- **Steatosis**: Simple accumulation of fat within the liver.
  - causes: drugs, inborn errors of metabolism, acquired metabolic disorders.

- **Steatohepatitis (NASH)**: Accumulation of fibrous tissue in the liver.
Alcoholic Liver Disease

- Most common liver disease in the U.S.
- Acetaldehyde is a toxic byproduct of alcohol metabolism that causes damage to mitochondrial membrane structure and function.
- Predisposition:
  - genetic polymorphisms of alcohol-metabolic enzymes
  - gender (women)
  - simultaneous exposure to other drugs
  - infections with hepatotropic viruses
  - immunologic factors
  - poor nutrition status
Alcoholic Liver Disease

Three stages:
1. Hepatic Steatosis
2. Alcoholic Hepatitis
3. Alcoholic Cirrhosis
FIGURE 30-2 Complications of excessive alcohol consumption stem largely from excess hydrogen and from acetaldehyde. Hydrogen produces fatty liver and hyperlipemia, high blood lactic acid, and low blood sugar. The accumulation of fat, the effect of acetaldehyde on liver cells, and other factors as yet unknown lead to alcoholic hepatitis. The next step is cirrhosis. The consequent impairment of liver function disturbs blood chemistry, notably causing a high ammonia level that can lead to coma and death. Cirrhosis also distorts liver structure, inhibiting blood flow. High pressure in vessels supplying the liver may cause ruptured varices and accumulation of fluid in the abdominal cavity. Response to alcohol differs among individuals; in particular, not all heavy drinkers develop hepatitis and cirrhosis.
Biliary Cirrhosis

- Chronic cholestatic disease caused by progressive destruction of small and intermediate-size intrahepatic bile ducts.
  - (not extrahepatic or larger intrahepatic bile ducts)
- Results in Cirrhosis, disease progresses slowly, eventually resulting in cirrhosis, portal hypertension, liver transplantation, or death.
Hemochromatosis

- Inherited disease of iron overload associated with the gene HFE.
- Patients absorb excessive iron from the gut and may store 20-40g compared to the normal 0.3-0.8g.
- Life expectancy is normal if phlebotomy is initiated before the development of cirrhosis or diabetes mellitus.
Wilson’s Disease

- Autosomal-recessive disorder associated with impaired biliary copper excretion.
- Copper accumulated in the liver, brain, cornea, and kidneys.
- Copper chelation improves survival but does not prevent cirrhosis, liver transplantation will correct the metabolic defect.
Alpha₁–Antitrypsin Deficiency

- Inherited disorder.
- Alpha₁-Antitrypsin is a glycoprotein found in serum and body fluids, it inhibits neutrophil proteinases.
- Cholestasis or cirrhosis is caused by this deficiency.
Risk Factors

- Obesity
- Overweight
- Type 2 diabetes
- Hemochromatosis
- Hepatitis B or C
Pathophysiology of Cirrhosis

- Development of scar tissue → blocked blood flow → disturbs normal function
- Precise cellular injury depends on the cause of cirrhosis.
Alcoholic Cirrhosis

- Characterized by:
  - Inflammation
  - Degeneration and necrosis of hepatocytes
  - Infiltration of leukocytes and lymphocytes
  - Immunologic alterations
  - Lipid peroxidation
  - Fibrosis

- Serum IgA is often elevated.
Alcoholic Cirrhosis

- Begins with hepatic steatosis:
  - Alcohol → acetaldehyde → lipid peroxidation → disruption in membrane function.
  - Mitochondrial function is impaired.
  - Enzyme and protein synthesis may be depressed or altered.
  - Hormone and ammonia degradation is diminished.
Alcoholic Cirrhosis cont.

- Acetaldehyde causes:
  - Altered metabolism of vitamins and minerals.
  - Liver fibrosis
  - Malnutrition
  - Hepatic specific autoantibodies
- TNF, IL-6, IL-8 and IL-18 are associated with ALD
- Inflammation and necrosis $\rightarrow$ collagen formation.
Metabolism of Alcohol

Ethanol → Acetaldehyde

ADH/CYP2E1

Altered redox potential

Oxidative stress

Steatosis

Lipid peroxidation

Adduct formation

Apoptosis/necrosis

Acetate → CO₂ + H₂O

Peripheral tissues

TRENDS in Molecular Medicine
S/S of Alcoholic Cirrhosis

- Enlarged liver
- Anorexia
- Nausea
- Jaundice
- Edema
- Fatigue
- Weight loss
- Fever
- Abdominal pain
Comorbidities of Alcoholic Cirrhosis

- Hepatomegaly
- Splenomegaly
- Ascites
- GI hemorrhage
- Portal hypertension
- Hepatic encephalopathy
- Esophageal varices
- Anemia
- Increased risk for infection
Ascites
Esophageal Varices
Biliary Cirrhosis

- Damage begins in the bile canaliculi and bile ducts, not hepatocytes.
Primary Biliary Cirrhosis

- Autoimmune
- Mitochondrial autoantibodies present
- Characterized by
  - Inflammation and destruction of small intrahepatic bile ducts
  - Fibrosis
S/S of Primary Biliary Cirrhosis

- Elevated alkaline phosphatase levels
- Pruritus
- Fatigue
- Abdominal pain
- Jaundice
- Light colored stools
- Steatorrhea
- Fat soluble vitamin deficiencies
- Hyperbilirubinemia
- Hyperlipidemia
Comorbidities of Primary Biliary Cirrhosis

- Portal hypertension
- Hepatic encephalopathy
- Liver failure
- Osteomalacia
- Osteoporosis
Secondary Biliary Cirrhosis

- Obstruction of the common bile duct or its branches
- Obstruction $\rightarrow$ increased hepatic pressure $\rightarrow$ accumulation of bile
- Necrosis $\rightarrow$ proliferation and inflammation $\rightarrow$ edema and fibrosis
S/S of Secondary Biliary Cirrhosis

- Similar to primary, especially jaundice and pruritus.
- Elevated conjugated bilirubin and alkaline phosphatase levels.
Postnecrotic Cirrhosis

- Consequence of many, chronic, severe liver diseases.
- 25% of people with hepatitis C develop this.
- Drugs or toxins, inherited metabolic disorders, and alpha antitrypsin deficiency can also lead to this.
- Fibrosis separates islands of liver cells → nodular appearance
Normal liver

Enlarged, pale yellow fatty liver

Macronodular cirrhosis

Micronodular cirrhosis
S/S of Postnecrotic Cirrhosis

- Portal hypertension
- Ascites
- Varices
- Hypersplenism
- Hepatic encephalopathy
Nonalcoholic Fatty Liver Disease (NAFLD)

- Accumulation of fat droplets in the hepatocytes
- Can lead to
  - Fibrosis
  - Cirrhosis
  - Hepatocellular carcinoma
- Caused by:
  - Drugs
  - Inborn errors of metabolism
  - Acquired metabolic disorders
- Associated with:
  - Obesity
  - Diabetes mellitus
  - Dyslipidemia
  - Insulin resistance
Nonalcoholic Steatohepatitis (NASH)

- More severe form of NAFLD
- Accumulation of fibrous tissue
- Patients may experience:
  - Malaise
  - Weakness
  - Hepatomegaly
- Progression to cirrhosis is variable.
Acute Liver Failure

- Severe complications rapidly after the first signs of liver disease (such as jaundice).
- Indicates severe liver damage (80-90% of liver cells do not function)
Acute Liver Failure

- **S/S:**
  - Cerebral edema
  - Hepatic encephalopathy
  - Coma
  - Brain herniation
Medical Diagnosis

- Liver Biopsy
  - Though unnecessary if clinical, laboratory and radiologic data indicates cirrhosis.
  - Biopsy poses a small but significant risk and cirrhosis increases risk for complications with biopsy.

- Imaging
  - CT
  - MRI
  - Ultrasound

- Endoscopy
- Labs
Important Lab Values

- Alanine Aminotransferase (ALT)
- Alkaline Phosphatase
- Ammonia
- Amylase
- Aspartate aminotransferase
- Gamma Glutamyl Transpeptidase
- Lactic Dehydrogenase
- Prothrombin Time
- BUN
- Bilirubin
- Glucose
MELD (model for end stage liver disease)

- Based on three blood tests
  - International normalized ratio (INR)
  - Bilirubin
  - Creatinine
- MELD scores usually range between 6 and 40, with a score of 6 indicating the best likelihood of 90-day survival.
Medical Therapies

By: Natalie White
Current Medical Therapies

- Management of **portal hypertensive bleeding**
  - Pharmacologic therapy
    - Adrenergic blockers to decrease heart rate
    - Somatostatin analog to decrease bleeding
  - Endoscopic banding or variceal ligation
  - Shunts (surgically placed)

- Medications used for **biliary cirrhosis**
  - Actigall
  - Chenix
Current Medical Therapies

- Medication for **encephalopathy**
  - **Rifaximin**: nonabsorbable antibiotic which kills ammonia-producing bacteria in the colon
  - **Neomycin**: nonabsorbable antibiotic which kills ammonia-producing bacteria in the colon
  - **Lactulose**: nonabsorbable disaccharide which acidifies the colon and keeps ammonia as ammonium ion; also acts as an osmotic laxative to help remove the ammonia
Current Medical Therapies

- **Treatment of ascites**
  - Large-volume paracentesis
    - Fluid drained from the abdomen through a needle
  - Diuretic therapy
    - **Lasix (furosemide):** loop diuretic; limits Na\(^+\) reabsorption
      - Possible side effects include hyponatremia, hypokalemia, hypomagnesemia, hypocalcemia, hypochloremic acidosis
    - **Spironolactone:** K\(^+\)-sparing diuretic; limits Na\(^+\) reabsorption
      - Serum levels of K\(^+\) must be monitored closely
    - Must monitor weight, abdominal girth, urinary Na\(^+\), serum nitrogen, creatinine, albumin, uric acid, & electrolytes during use of diuretics
Paracentesis
Current Medical Therapies

- **Liver transplant**
  - An established treatment for ESLD
  - The liver can regenerate itself, so a transplant of a partial liver can grow to full size
  - Complicated surgery
  - Post-transplant medications have nutritional implications
Nutritional Assessment and MNT for Cirrhosis and ESLD

“The pancreas and liver are essential to digestion and metabolism....When they are diseased, the necessary medical nutrition therapy (MNT) is complex” (Krause, 645).
Nutritional Assessment

- Moderate to severe **malnutrition** is common among cirrhosis and ESLD patients
  - Inadequate oral intake
    - Anorexia, dysgeusia, early satiety, nausea, vomiting
  - Maldigestion and malabsorption
  - Abnormal macronutrient metabolism
  - Increased energy expenditure
  - Protein loss from paracentesis
- Appropriate therapy can reverse malnutrition and improve outcomes
Nutritional Assessment

- Performed to determine the cause and the extent of malnutrition
- **Subjective global assessment** (SGA) is used
  - Why SGA?
    1. Traditional markers of nutrition status can be affected by liver disease, making interpretation difficult:
       - Body weight is affected by edema, ascites, and diuretic use
       - Visceral protein levels are affected by decreased protein synthesis
    2. Based on both anthropometrics and dietary intake
Nutritional Assessment: SGA

- Subjective Global Assessment (Box 30-1, pg 657)
  - History
  - Physical Findings
  - Existing Conditions
  - Nutritional Rating Based on Results
SGA—History

- Weight change
  - Consider edema and ascites
- Taste changes and early satiety
- Dietary intake
  - kcal, protein, Na⁺
- Persistent GI problems
  - N/V/D, constipation, difficulty chewing or swallowing
SGA—Physical Findings

- Muscle wasting
- Fat stores
- Ascites or edema
SGA—Existing Conditions

- Disease state
- Problems that could influence nutrition status
  - Hepatic encephalopathy
  - GI bleeding
  - Renal insufficiency
  - Infection
SGA—Nutritional Rating

- Well nourished
- Moderately (or suspected) malnourished
- Severely malnourished
Nutritional Assessment

- SGA gives a broad perspective, but is not sensitive to changes in nutrition status
- Lab tests for nutritional deficiencies such as vitamins, Mg$^{2+}$, and iron may also be helpful in identifying needed intervention
Nutrient Requirements

- Energy
  - REE varies among patients
    - Could have normal, hypo-, or hyper-metabolism

- Lipids
  - Preferred source of energy \(\Rightarrow\) increased lipolysis
  - Despite increased use, lipid storage not impaired
  - 25-40% kcal recommended
Nutrient Requirements

- Carbohydrates
  - Difficult to determine because of liver’s role in carbohydrate metabolism
  - Decrease in gluconeogenesis
  - Increased use of lipids and amino acids as fuel
  - Alterations in insulin, glucagon, cortisol, and epinephrine
  - Insulin resistance can also be a factor
Nutrient Requirements

- **Protein**
  - Most controversial and most complex to manage
  - Nitrogen losses with fulminant hepatic failure or decompensated disease, **but not** with stable cirrhosis
  - Uncomplicated cirrhosis **w/o** encephalopathy
    - 0.8-1.0 g/kg dry body weight
  - To promote positive nitrogen balance
    - 1.2-1.3 g/kg dry body weight
  - Alcoholic hepatitis or decompensated disease (sepsis, infection, GI bleeding, severe ascites)
    - ≥ 1.5 g/kg dry body weight
Nutrient Requirements

- Vitamins and minerals
  - Supplementation necessary for all ESLD patients
  - Liver plays key role in nutrient transport, storage, and metabolism
  - Deficiencies can contribute to complications
  - Both fat- and water-soluble vitamins needed
  - Copper and manganese should not be given in supplements
    - Primarily excreted with bile, which is often impaired
  - Zinc, magnesium, and calcium should be given
Medical Nutrition Therapy

- General recommendations:
  - Increased energy intake via small, frequent meals
  - Restriction of Na⁺ for fluid retention
  - Carbohydrate-controlled diets for hyperglycemia
  - Vitamin and mineral supplements
  - Oral liquid supplements or enteral (tube) feeding
MNT—Special Considerations

- Complications with nutrition implications:
  - Portal hypertension
    - EN cannot be used during acute bleeding episodes
    - PN is used if patient will be taking nothing orally for 5 days
  - Ascites
    - Na\(^+\) restriction
    - Adequate protein intake to replace losses from paracentesis
  - Hyponatremia
    - Fluid restriction
MNT—Special Considerations

- **Glucose alterations**
  - Patients with diabetes: standard MNT to control blood sugar
  - Patients with hypoglycemia: eat more frequently

- **Fat malabsorption**
  - Increase intake of medium-chain triglycerides (MCTs)
  - Don't require bile salts or micelle formation for absorption

- **Hepatorenal syndrome**
  - Alteration in fluid, Na⁺, K⁺, and P

- **Osteopenia**
  - Maintain weight and eat a well-balanced diet
  - Adequate protein to avoid wasting of muscle mass
  - Adequate vitamin D
MNT—Hepatic Encephalopathy

- Controversial, and varies somewhat by patient
- Avoid unnecessary protein restriction
  - Most patients can tolerate up to 1.5 g/kg
- Vegetable proteins and casein may be more beneficial than meat protein
  - Higher BCAAs and fiber
- Probiotics and synbiotics may also be beneficial
  - May reduce ammonia in portal blood or prevent uptake of lipopolysaccharides
  - Decreases inflammation and oxidative stress in liver cells
MNT—NAFLD & NASH

- Gradual weight loss if overweight or obese
  - Losing weight too fast accelerates development of cirrhosis and increases risk of gallstones
- Eat a heart healthy diet
  - Rich in fruits and vegetables
  - Low in saturated fat and cholesterol
    - Control dyslipidemia
  - Include whole grains
- Control blood sugar (if diabetic)
- Exercise and be physically active
MNT—Liver Transplants

- Major surgery → increased protein and energy needs
- EN is vital for liver cell proliferation
  - Added probiotics and fiber may decrease post-operative infection rate
- PN is not used unless gut is not functioning
  - Can adversely affect liver function
- Dietary modification based on nutritional side effects of medications
Enteral Nutrition--NutriHep

- **kcal/mL**: 1.5
- **Protein**: 11%
- **Carbohydrate**: 77%
- **Fat**: 12%
- **Protein Source**: crystalline L-amino acids, whey protein concentrate
- **NPC:N Ratio**: 209:1
- **MCT:LCT Ratio**: 70:30
- **n6:n3 Ratio**: 4:1
- **Osmolality**: 790
- **Water**: 76%
- **Meets 100% RDI for 19 key micronutrients**: 1000 mL

- High ratio of branched-chain amino acids to aromatic amino acids
- Calorically dense for fluid management
- High MCT to LCT ratio to facilitate absorption
- May be used for tube feeding or for oral supplementation
Prognosis

By: Emily Horn
Prognosis

- Fibrosis/scarring are irreversible
- Life expectancy depends on underlying disease, extent of liver damage, number of complications
- Alcoholic cirrhosis
  - Continued drinking worsens prognosis
- Solid Line – pts who abstained from alcohol
- Dotted Line – pts who decreased alcohol consumption
Prognosis, cont.

- **Primary Biliary Cirrhosis**
  - Patients usually live 8-10 years after first symptoms appear \(^{(1)}\)
  - Medications can prolong life
    - Liver transplant
- **Secondary Biliary Cirrhosis**
  - Surgery can be performed to reopen bile channels – prolongs survival and relieves symptoms
Prognosis, cont.

- **End-Stage Liver Disease**
  - Ascites – 25% of people “who develop ascites caused by cirrhosis die within 1 year” (1)
  - Hepatorenal syndrome – prognosis worse depending on severity of liver dysfunction
  - Hepatic encephalopathy – if not treated, can lead to coma and death
  - Liver transplants usually necessary
Liver Transplants

- Demand is higher than supply
- Model for End-Stage Liver Disease (MELD)
  - Prognostic tool to choose liver recipients
  - Post-op survival predicted by prothrombin time, serum bilirubin and creatinine
  - High score = bad
  - Patients with decompensated cirrhosis and MELD score <20 get livers
- 7-year survival rate of 60% (2)
Living Donor Liver Transplants

- Transplant often taken from right lobe
- “Almost 60% of the liver mass of the donor must be transplanted”
- Worth the risk?
  - Lower survival rate in recipients of LDLT vs. deceased donor liver transplant (DDLT)
  - Puts donors at risk
  - However, reduces waiting list mortality from low availability of DDLT
Herbal Supplements

- **Milk Thistle**
  - Contains silymarin
  - Supposedly reduces oxidation

- **S-adenosyl-L-methionine (SAMe)**
  - Supposedly involved with glutathione production
  - Small studies – helpful for cholestasis?

- **Astragalus**
  - Not well understood; possibly dangerous side effects

- **Licorice Root**
  - May help treat hepatitis C
  - Dangerous side effects

- None are overwhelmingly effective
Case Study
Teresa Wilcox – Grad Student and Teacher

- 26 years old
- C/O - Fatigue, anorexia, N/V, weakness, bruising unrelated to injury
Nutrition Assessment

- **Anthropometric**
  - 86% of IBW, with 7% wt loss in last 6 mo.
  - BMI = 18.49

- **Biochemical**
  - alb – severely depleted
  - prealb – mildly depleted
  - bilirubin – high
  - ALT, AST – both high
  - TG - high
Nutrition Assessment, cont.

- Clinical
  - Diagnosed w/ hepatitis C 3 years ago
  - Dx: probable cirrhosis secondary to chronic hepatitis C

- Dietary
  - Protein: 1-1.2g/kg = 57 – 68.4g
  - Energy: (USE INDIRECT CALORIMETRY!)
    - 1704-1988 kcals (30-35 kcal/kg)
PES Statement

- Inadequate oral intake related to loss of appetite secondary to cirrhosis as evidenced by 7% weight loss in last 6 months.
Sample Diet – 1805 kcals, 68.6 g protein

- **Breakfast**: Carnation instant breakfast mixed in 8 oz. low fat milk and medium banana
- **Snack**: 8 oz. OJ, 1 Tbsp. peanut butter, 6 Ritz crackers
- **Lunch**: 1 c. tomato basil soup w/1oz. shredded cheese, 1 wheat dinner roll, 8 oz. diet coke
- **Snack**: ¾ c. potato salad
- **Dinner**: 1 serving lasagna, ½ c. steamed zucchini, 8oz. OJ