Liver Disease / Pancreatitis Case Study

(Adapted from Clinical Nutrition Case Studies, by Wayne Billon)

Mr. G is a 56 yo construction worker. He is recently divorced with three children in college. He started drinking heavily when he was in high school and is currently consuming approximately 8 beers and 1-2 shots of whiskey per day. For the last several days, Mr. G has been experiencing severe epigastric pain with radiation to his back. He has also felt nauseous and has had several episodes of vomiting. His abdomen is sore to the touch and slightly distended. His temperature has been elevated. When his symptoms continued for 3 days, Mr. G went to a neighborhood treatment center to see a physician.

The physician took a medical history and did an examination that included lab values. His labs were as follows:

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Normal</th>
<th>Test</th>
<th>Result</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>144 mEq/l</td>
<td>12.0-14.4 sec</td>
<td>ALT</td>
<td>125 u/l</td>
<td>5-40 u/l</td>
</tr>
<tr>
<td>K</td>
<td>3.1 mEq/l</td>
<td>3.5-5.3 mEq/l</td>
<td>AST</td>
<td>183 u/l</td>
<td>10-35 u/l</td>
</tr>
<tr>
<td>Mg</td>
<td>1.2 mEq/l</td>
<td>1.4-2.5 mg/dl</td>
<td>Total Bili</td>
<td>3.2 mg/dl</td>
<td>0.2-1.3 mg/dl</td>
</tr>
<tr>
<td>Cl</td>
<td>103 mEq/l</td>
<td>99-112 mEq/l</td>
<td>Ser. Amylase</td>
<td>685 u/l</td>
<td>5-115 u/l</td>
</tr>
<tr>
<td>Glucose</td>
<td>145 mg/dl</td>
<td>70-110 mg/dl</td>
<td>Lipase</td>
<td>463 u/l</td>
<td>7-58 u/l</td>
</tr>
<tr>
<td>Serum Albumin</td>
<td>2.9 g/dl</td>
<td>3.5-5.0 g/dl</td>
<td>PT</td>
<td>12.2 sec</td>
<td>135-145 mEq/l</td>
</tr>
</tbody>
</table>

The physician told Mr. G that he had acute pancreatitis with an enlarged liver. Mr. G was told that his liver enzymes were elevated and that if he continued drinking, he could develop severe liver disease. The physician wanted to admit Mr. G to the hospital for bowel rest, hydration and observation, however, Mr. G refused.

The physician told Mr. G that if he stopped drinking, his liver could clear up in a matter of weeks. Mr. G was encouraged to follow a low fat, high protein diet with absolutely no alcohol, and call if the pain and discomfort did not clear up within 2-3 days.
Mr. G's height was 5'11" and he weighed 170 lbs. The physician sent Mr. G home with the following prescription:

1. Pancreatin (Pancreatin tablets)--enough for 3 days
2. Potassium chloride (Slow-K)--enough for 3 days

The physician advised Mr. G not to take Pancreatin with antacids. He also advised him to take a multivitamin and mineral supplement daily.

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**Questions**

1. Explain the reasoning behind the diet order Mr. G received: "Low fat, high protein, with absolutely no alcohol.
   The pancreas secretes enzymes that digest fat, protein, and carbohydrates into the duodenum. In pancreatitis, these enzymes get activated early, breaking down cells within the pancreas, causing inflammation. To prevent further irritation of the pancreas by requiring an increased release of lipase (the enzyme that breaks down fat), a low-fat diet is typically ordered. Eating a diet lower in fat puts less stress on the pancreas, allowing it to heal. In addition, a low-fat diet will prevent the malabsorption of fat, and therefore decrease the likelihood of steatorrhea. A patient presenting with pancreatitis has increased nutrient needs due to malabsorption of nutrients, possible N/V, diarrhea, loss of appetite, etc., and without calorically dense fat in the diet, protein is a good substitute to provide adequate calories. Initiation of protein digestion begins in the stomach, unlike fat, which is only digested in the small intestines by pancreatic enzymes. Lastly, alcohol is a typical etiology of pancreatitis and therefore can exacerbate the pancreas even more. Complete cessation from alcohol consumption is the first goal to relieving pancreatitis symptoms.¹

2. Outline the instruction you would give Mr. G concerning his diet and liver disease.

- Avoid alcohol. Alcohol → damaged liver cells → build up of fat in liver (fatty liver) → inflammation of liver (hepatitis) → scarring of the liver tissue (cirrhosis)
- Avoid high fat foods. Choose low-fat items instead and keep fat intake between 25-30% of total kcals.
- Importance of compliance; if not, possible progression from hepatitis to cirrhosis (irreversible)
- Importance of protein as an energy source
3. List the lab values in the chart above that are indicative of liver disease.
   Elevated: glucose, ALT, AST, bilirubin, PT (prothrombin time—takes longer for blood to clot) and decreased serum albumin

4. What abnormal lab values are indicative of pancreatitis?
   Serum amylase and lipase (will be elevated in pancreatitis).²

5. What was the purpose of the physician giving Mr. G the potassium chloride?
   The potassium chloride (Slow K) is used as a potassium supplementation to prevent severe hypokalemia. Liver disease corresponds with natural potassium loss³ due to poor dietary intake and vomiting. If ascites is present, the administration of diuretics also causing hypokalemia.¹

6. Give the pathophysiology of fatty liver ² alcoholism
   Continuous and prolonged exposure of alcohol damages and destroys liver cells, which interferes with the normal metabolism and storage of nutrients in/by the liver, including fat. Typically, alcohol replaces food in the diet, so malnutrition is common with people who have a fatty liver. The body sees alcohol as a toxic substance, and therefore metabolizes alcohol over whatever nutrients are obtained through food. In the process of digesting ethanol by an enzyme called alcohol dehydrogenase (ADH), NADH/NAD⁺ ratio increases. This directly promotes fatty liver by inhibiting gluconeogenesis and fatty acid oxidation. In other words, the liver increases its synthesis of fatty acids and decreases fatty acid degradation in the liver, resulting in a fat build up in liver cells. Fatty liver is reversible if alcohol consumption ceases.¹⁵ If not, fatty liver can progress to alcoholic hepatitis or further to cirrhosis.

Mr. G followed his diet and took his medication and began to feel better. He stayed off the alcohol for 3 or 4 days but then began to have delirium tremens and started to drink again. He began to have stomach pains different from the epigastric pain he had earlier. He noticed his abdomen seemed to be becoming distended and he felt like he had no energy.

Mr. G continued to drink heavily. One morning he woke up with severe epigastric pain and began vomiting blood. Mr. G was brought to the hospital and admitted with GI bleed ² esophageal varices, weight gain, ascites, steatorrhea, and alcoholic cirrhosis.
Lab values included
H/H 9.7/27
BUN 35
PT increased
BG 68
ALB 2.4
K 3.0
Na 132
AST, ALT, Alkaline Phosphatase – all increased
Chol 289
TG 310
Ammonia 60

Mr. TG's weight was now 192 lbs. The physician noted 2+ pitting edema of the lower extremities. Mr. G was alert and oriented.

Questions continued

7. Describe the functions of the liver, including from where the blood is supplied and what is produced by the liver.

The liver receives blood from two locations. Oxygenated blood is supplied to the liver by the hepatic artery from the heart. Also, venous blood draining from the digestive tract is carried to the liver by the hepatic portal vein, so that nutrients absorbed from the GI tract can pass through and be processed by the liver before reaching the heart via the hepatic vein.

The functions of the liver includes:

- Carbohydrate metabolism and storage: Glycogenesis, gluconeogenesis, glycogenolysis and glycolysis/TCA cycle occurs in the liver.
- Protein metabolism: the liver synthesizes serum proteins (albumin, fibrinogen, transferrin, prothrombin) and urea
- Lipid metabolism: Synthesizes TAGs, lipoproteins, and oxidizes FAs. Lipogenesis, lipolysis, ketogenesis, cholesterol and phosphotide synthesis occurs here.
- Vitamin metabolism: of niacin, D3, pantothenic acid, folic acid, pyridoxine, thiamin, B12
- Detoxification and degradation of drugs
- Bile acid production
- Storage: Stores glycogen, FAs, fats, and fat-soluble vitamins, and minerals
- Heme Metabolism: heme → biliverdin → bilirubin

8. Calculate Mr. G's IBW and percent IBW.
IBW = 106 lb (6 x 11) = 172 lb. (78.2 kg)
%IBW = 192 lb / 172 lb x 100% = 112%

9. Calculate Mr. G’s total energy needs. (Show your calculations and weight basis).
   Using 30-35 kcal/kg and using his usual BW (edema free weight) of 170 lb (77.3 kg) and
   because his usual BW very close to his IBW.

   30 kcal x 77.3 kg = 2320 kcal
   35 kcal x 77.3 kg = 2700 kcal

10. Why is the BUN at 35 mg/dl?
    Normal BUN: 10-20 mg/dL. In advanced liver disease, BUN may be elevated due to the
    protein catabolism from malnutrition associated with reduced oral intake and increase alcohol
    intake replacing nutrients. The GI bleeding from the esophageal varices will elevate BUN, as
    well. BUN is synthesized in the liver so abnormal BUN can reflect a poorly functioning liver.

11. Why is blood sugar decreased?
    Hypoglycemia is a result of insulin resistance, glucose intolerance, increased circulation
    of glucagon, and the lack of glycogen storage in the liver (depletion of nutrition stores). With
    decreased oral intake and the inability of glycogen mobilization from the liver hypoglycemia
    occurs. It’s typical to have muscle proteolysis to compensate as an energy source, leading to
    muscle wasting and weight loss, in severe cases.

12. Explain the prolonged prothrombin time.
    PT measures the blood’s ability to clot. A normal coagulation time is 9-11 seconds. In a
    normal function liver, clotting factors are produced to keep the clotting time of blood within
    this range. During liver disease, clotting factors may be inhibited so clotting time is usually
    delayed, so PT in increased.

13. Why is the cholesterol level elevated?
    Bile acid production by the liver may be compromised when liver function is
    compromised. Cholesterol is used to make bile and without the production of bile, cholesterol
    may become elevated in the blood. Normally cholesterol is pulled from the blood to create bile
    in the liver, which lowers LDL cholesterol. Without the production of bile, malabsorption of fat
    occurs and causes steatorrhea. Also, cholesterol is metabolized in the liver and without the
    metabolism of cholesterol, it builds up in the blood.

14. Why is the serum albumin decreased?
    Albumin is synthesized in the liver, so with lack of normal liver function, albumin
    production is altered, causing a decrease in serum albumin. Being severely malnourished can
    also cause a low serum albumin. Decreased serum albumin could indicate protein energy
    malnutrition.
15. List the stages or levels of Hepatic Encephalopathy.
   Grade 0: Minimal changes in memory and undetectable changes in personality or behavior.
   Grade 1: lack of awareness, shortened attention span, mild confusion, slow ability to perform mental tasks and presence of asterisix (flapping of wrists)
   Grade 2: disorientation, slurred speech, lethargy, obvious personality changes, obvious asterixis, and inability to perform mental tasks.
   Grade 3: Unable to perform mental tasks, disorientation about time and place, lethargic and sleep, but can be aroused and confused.
   Grade 4: Coma with or without response to painful stimuli

16. What factors may be involved in precipitating Hepatic Encephalopathy?
   Hepatic encephalopathy is due to the accumulation of ammonia in the liver, which spills into the bloodstream to cross the blood brain barrier to result in mental status changes. Ammonia is typically converted to urea in the liver of healthy individuals and excreted by the kidneys. When this step cannot happen, hyperammonemia occurs, causing different stages of mental alterations leading up to a coma. Factors that increase the ammonia levels include GI bleed, infection, diarrhea, vomiting, and sedative use.

17. What can you expect the ammonia level to be (elevated or low)? How does this affect your medical nutrition therapy?
   Ammonia levels are usually elevated in individuals with liver disease. The liver loses its ability to detoxify substances like ammonia. Ammonia is produced from the breakdown of protein, and would normally enter the urea cycle where it’s converted to urea and excreted by the kidneys. In individuals with altered liver function, ammonia cannot enter the urea cycle, so ammonia builds up and can pass the blood brain barrier to cause hepatic encephalopathy.¹,²

As a dietitian in providing MNT, it’s important to ensure patient’s protein intake in adequate to prevent overall catabolism. Even though it’s thought that with increased protein in the diet, there will be an increase metabolism of protein and therefore, increased ammonia levels. However, there is not sufficient evidence to support this. There is more evidence showing detrimental effects from protein energy malnutrition in these patients. There is some research showing effectiveness of branch chain amino acids (BCAA) as a beneficial protein source due to their metabolism in skeletal muscle instead of the liver.²

18. What are the nutrition-related effects of the following medications, which are often used to treat these patients?
   Neomycin- (decrease colonic bacteria concentration that produce ammonia) may cause diarrhea, nausea, vomiting, flatulence, fatty-appearing stools, weakness, increased thirst and nephrotoxicity⁶
Lactulose- (stimulates passage of ammonia from tissue to lumen, and inhibits intestinal ammonia production) may cause dehydration, belching, cramps, diarrhea, flatulence, N/V, and borborygmi (rumbling sounds of gas in the stomach).

19. How much protein should Mr. G receive per day?
   I would use 1.2-1.5 g/kg body weight and use his usual body weight of 77.3 kg, which doesn’t include the fluid from ascites. This will give him a total protein recommendation of 92 to 116 grams.

20. Explain the relationship between alcohol and esophageal varices?
   High consumption of alcohol can eventually lead to degeneration of liver cells and build up of fibrous tissue within the liver cells (hepatocytes). When there is connective tissue overgrowth, blood cannot enter the liver from the portal vein appropriately. As a result, dilation of blood vessels within the esophageal or abdominal occurs to increase blood pressure within the portal vein. This causes portal hypertension. Varices can rupture and bleed, as a result. The more alcohol consumption, the greater likelihood of being malnourished and the more malnourished, the greater chance of seeing irreversible liver function causing cirrhosis, portal HTN, esophageal varices, or encephalopathy.

21. When Mr. G’s diet is advanced, what diet modifications would you suggest?
   I would advice Mr. G to follow a high calorie/protein diet that restricts fat to ~50 grams/day with a sodium restriction of ~2 grams per day and low fiber foods to avoid irritation the esophageal varices. This includes avoiding alcohol and stick to small frequent meals.
Resources: