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Cirrhosis of the Liver Secondary to Alcoholism

The liver regulates most chemical levels in the blood and excretes a product called bile. The bile helps carry away waste products from the liver for excretion. There are more than 500 vital functions that have been identified with the liver. It is the largest solid organ in the body weighing about three pounds. It performs many important functions such as, manufacturing blood proteins that aid in clotting, oxygen transport, and immune system function. The liver also stores excess nutrients and returns some of the nutrients to the bloodstream for delivery to other organs. The list of functions does not end there. The liver also stores glycogen, breaks down saturated fat and produces cholesterol. Without the liver we would not be able to perform various functions or rid the body of harmful toxins (7).

When the liver becomes cirrhotic secondary to alcohol there is a disruption in the blood flow through the liver. The chronic disease causes cell destruction and fibrosis of hepatic tissue. The tissue is slowly dying at each stage causing death of the hepatocytes leading to malfunction of many processes (7). Alcohol is a small molecule that is water and lipid soluble so it can permeate all organs and their vital function (1). This is why people who have a damaged liver due to alcohol also have many other organs that are damaged. The cell destruction and fibrosis of the hepatic tissue happens in three different stages. At the first stage, alcohol is the preferred fuel source in the TCA cycle, so fat becomes displaced. The hydrogen from the alcohol replaces up to 90% of the fat. The fat then accumulates in the blood and body leading to fatty liver. At the same time as fat is

accumulating in the blood, it is causing a rise in the triglyceride levels. (6) This is where the term was obtained for “fatty” liver. Luckily, at this stage full abstinence will lead to recovery with no scar tissue or damage.

Unfortunately, if the drinking does not stop, the fatty liver will advanced leading into the second stage. Alcohol consumption increases iron stores in the liver, which contributes to the hepatitis C virus. Unbound iron building up in the liver causes progressive damage by inducing fibrosis. Fibrosis is when the death of the liver tissue begins to occur. Even with adequate nutrition, the cell death will continue to progress. Symptoms are not common with cirrhosis in the early stages, which is why it tends to go unnoticed until it’s too late. If a person does have symptoms at this stage it may include swollen liver, nausea, vomiting, and abdominal pain. Many of these symptoms can be mistaken for the flu, cold, or written off as insignificant. At this stage recovery is possible, but the scar tissue remains. (1)

In the third stage, also know as the cirrhotic stage the cell death continues to occur until it is termed a cirrhotic liver. The growth of the connective tissue becomes so great that it destroys the liver cells leading to fatty degeneration of hepatocytes. This leads to inadequate bile acid secretion, resulting in fat malabsorption. Not only is the fat being displaced in the TCA cycle, but now bile secretion has diminished so fat is no longer being absorbed. Bile is also important for converting fat soluble vitamins into their active form. At this stage the damage is irreversible, and even with strict discontinuation of the alcohol the liver will remain cirrhotic (1).

In the cirrhotic stage, as scar tissue replaces healthy tissue, symptoms become apparent. Jaundice can occur which is the yellowing of the skin. The liver can no longer

absorb an adequate amount of bilirubin, leading to a build up in the blood causing the skin and eyes to look yellow. Loss of appetite can occur which can be attributed the nausea and vomiting or from the ascites. Sometimes itching can even occur due to the bile products being deposited in the skin. There is an inadequate production and flow of the bile causing it to deposit in other areas within the body. (5) The liver also manufactures proteins that aid in blood clotting, so as the liver becomes damaged the protein production may be decreased, causing easy bruising and gastrointestinal bleeding. Another common symptom that patients experience when suffering from withdrawals from alcohol is agitation and excitement which can contribute to that increase in anxiety. Lastly, but not least, is the symptoms of disorientation, confusion, behavior change, and slurred speech. This can occur if the ammonia levels are rising in the blood known as hepatic encephalopathy. When these symptoms become more predominate and frequent, it is the body's way of saying that it is beginning to shut down. (7).

There are many complications that can arise with cirrhosis of the liver due to the extensive damage that takes place. As mentioned before, hepatic encephalopathy can occur. The liver can no longer convert ammonia to urea causing a significant rise in the blood. A failing liver cannot remove toxins from the blood, and they eventually accumulate in the brain. The buildup of toxins in the brain can decrease mental function and cause coma. Signs of decreased mental function include confusion, personality changes, memory loss, trouble concentrating, and a change in sleep habits. Ascities can also occur which is when the blood cannot leave the liver so fluid is pulled to dilute the load. The patient may have decreased appetite due to the fluid overload that can cause bloating and feeling full. With ascities it is important to decrease sodium intake and fluid

intake in order to prevent exasperation of the problem. Since there is a significant decrease in blood flow through the liver, it causes an increased pressure which can lead to portal hypertension. In return portal hypertension can cause varices as the pressure becomes greater causing damage to the vessels. Edema can also occur in other parts of the body, just like ascities. (3) The damage of cirrhosis does not end there. A cirrhotic liver becomes insulin resistance. As the pancreas try's to produce more insulin thinking the body needs more, it soon can not keep up with the demand. This will eventually lead to excess glucose in the blood. Furthermore, there is impaired blood clotting which can lead to GI bleed due to the liver slowing or stopping the production of proteins needed for blood clotting. Lastly, alcoholics with cirrhosis of the liver tend to be very malnourished due to the malabsorption of many vitamins and nutrients. Since alcohol replaces fat in the TCA cycle most fat soluble vitamins become displaced. This leads to many deficiencies.(6)

Cirrhosis of the liver is not only caused by alcohol abuse. There are many other diseases or manifestations that can affect the normal function of the liver. A more common complication of the liver is the hepatitis B and C virus. The infections cause swelling of the liver which can lead to cirrhosis. Another predominant case of cirrhosis can occur due to biliary duct disorder. This is when there is a blockage of the bile duct, which carries bile formed in the liver to the intestines. Since the bile is blocked, it builds up in the liver and causes it to become swollen and inflamed leading to cirrhosis. If the duct is blocked the liver is not able to detoxify and rid the body as toxins. A less common cause of cirrhosis is autoimmune hepatitis. This occurs when the immune mistakes livers normal cells for invaders. The body attacks the hepocytes leading to death

of the hepatic tissue. The liver can be affected in various ways. (3) Less commonly known is the nonalcoholic fatty liver disease. This disease resembles alcohol cirrhotic liver, but occurs in a person who does not drink or drinks little. Although this has been a rare cause, cases are becoming more frequent. There has been research that this disease may be linked to fructose intake. (3)

Another possible culprit for cirrhosis is obesity. As we know obesity affects a lot of organs and function in the body. The theory with cirrhosis is that the increase pressure from the excess fat and weight can lead to fatty liver. The higher a persons BMI, the greater their risk is for developing fatty liver or liver disease. The last cause of cirrhosis that will be touched on is hereditary hemochromatosis (HHC). HHC is when there are mutations in the *HFE* gene, causing an autosomal recessive disorder of iron metabolism. People with this absorb too much iron from foods they eat. The unbound iron causes free radical formation ultimately leading to organ injury. (1)

As dietitians there are many important guidelines that should be followed in order to ensure adequate care and health. The most important aspect is to make sure you are feeding the patient a healthful diet with adequate kcal to promote healing of the liver. Alcoholics displace a lot of nutrients and calories so it is important to make sure they are nourished with enough calories to help maintain body weight and promote healing of the liver. Alcoholics tend to replace alcohol for meals so it is important to assess the state they are currently in. People always talk about the “beer” gut, but alcohol can actually lead to weight loss and malnutrition. It is important to keep in mind that although the patient may be at an adequate weight, they can still be malnourished due to alcohol displacing many of the nutrients. The main goal is to improve protein calorie malnutrition

and correct nutrient deficiencies. A rule of thumb is to calculate the patients need based on 30-35 kcal/kg to prevent protein sparing. Protein: 1.0-1.5 g/kg for non-hepatic encephalopathy due to the catabolism state cirrhosis patients are in. An average range of carbohydrates recommended is between 55-65% with avoidance of simple sugars. Lipids can also be at an average range of 25-30% with essential fatty acids and a high unsaturated diet. (7) Another important aspect to note is the supplementation of B-complex. The B vitamins are used in the TCA cycle for digestion of alcohol. It is important to supplement these vitamins since they have had a history of chronic alcohol abuse. It is also important to supplement folic acid, Vitamin C, foods high in antioxidants due to the unbound iron causing free radical damage. (7).

In the advanced stages of cirrhosis it is important to supplement ADEK (fat soluble vitamins) due to the decrease bile formation which leads to failure to convert to vitamins in their active forms. Thiamin is also important to supplement because it helps bind unbound iron and helps reduce iron load in the liver causing less free radical damage. Lastly, it is important to provide a bedtime meal to decrease the increased lipid perioxidation and gluconenogensis. Enzyme called CYP2E1 has been found to be localized in regions of liver lobule that are damaged by alcohol. In the study they found that CYP enzyme has been shown to contribute to oxidative stress cause by alcohol. (9) This enzyme is relatively loosely coupled with cytochrome reduction which can therefore leak electrons to oxygen or catalyze lipid peroxidation. It is important to give the snack to prevent the increase in the catabolic state. (9)

Not only is it important to treat the cirrhosis, but we also have to treat the complications that arise due to the damaged liver. At times this can pose risks or

problems. Hepatic encephalopathy is a common issue with advanced stage cirrhotic patients. There has always been a debate on how much protein to give. Some studies suggest as low as 0.5 g/kg, while others suggest no lower than 0.8 g/kg to prevent the negative nitrogen balance. A rule of thumb is to go no lower than 40 g/day to avoid the negative balance. (3) It can sometimes be tricky because every patient is different. It is important to use your best clinical judgment. More recent debates discuss the important of BCAA in hepatic encephalopathy. The recommended amount is up to .25 g/kg day. A unique aspect of BCAA is that they are metabolized in the muscle and brain, not the liver. This is important for patients with liver damage. BCAA are metabolically very active in peripheral tissues and can be oxidized to produce energy or may act as an anticatacolic factor (lecine) by stimulating the synthesis and reducing the degradation of muscle protein. They are catabolized to L-alanine and L-glutamine in skeletal muscle. . BCAA stimulates the building for protein in muscle and possibly reduce muscle breakdown. It also can prevent faulty message transmission in brain cells. In liver disease BCAA is decreased in the body and AAA increased. Supplementation therapy of BCAA will compete with AAA for blood brain transport, which will reduce brain entry of AAA. It is important to keep a normalized ratio of BCAA:AAA. (2)

When reviewing at study titled, “Role of Branched Chain Amino Acids in the Management of Hepatic Encephalopathy”, they discussed how the increased muscle protein degradation coupled with impaired liver function induces a profound alteration of the plasma AA pattern characterized by an elevation in AAA and reduction in BCAA. Plasma and brain accumulation of AAA may in fact cause a severe impairment of brain neurotransmitter synthesis causing hepatic encephalopathy. The decrease in plasma

BCAA, which competes with AA for blood brain transport, contributes greatly to the accumulation of AA in the brain. (2) There were 48 patients in the study with decompensated liver cirrhosis who met the following criteria: HE grade II coma, or a history of hepatic encephalopathy with current liver cirrhosis. There were both males and females that were evenly distributed with each group have 17 males and 7 females. They were diagnosed based on clinical history, examination, laboratory (prothrombin time, albumin, ammonia level). The BCCA group received 28 g/day of BCAA. The groups received conventional therapy with equal calorie and protein ratios. The Long term BCAA supplementation showed an advantage compared to equal calorie and protein intake on non-BCAA group. Overall the BCAA group had lower ammonia levels, higher serum albumin, lower death rates, and lower hospital lengths of stay. (2).

Cirrhosis and the use of milk thistle of been dated back to the 1960's. Some studies are for the use of it, while some are against it. The part of the plant that is used for therapy with damaged liver is called silymarin. It is little black seeds that have been used since the 1960's in Europe for jaundice. Silymarin has been found to improve liver function, protect liver from toxins, and has antioxidant and anti-inflammatory properties. It has also been found to help the liver repair itself by growing new cells. I discovered a study with the use of silymarin in the treatment of liver diseases. (8) The study was a double blind, placebo-controlled study with silymarin in alcohol-induced liver disease. There were 106 patients in the study, which were selected based on elevated serum transaminase levels. Patients were forbidden alcohol during the trail. Only 97 patients completed the 4-week trial, with 47 in the silymarin group and 50 in the placebo. The silymarin group received 230-600 milligrams per day divided into two to three doses

based on weight. The overall outcomes showed a highly significant greater decrease in ALT and AST levels with silymarin compared with placebo. The AP and GT enzyme were significantly lower in silymarin group. When the AP and GT enzyme are raised it can be a sign of liver damage. There was no significant difference between groups in reduction in serum total and conjugated bilirubin. Overall, changes occurred significantly more often in the silymarin treated patients (11 out of 15) than in controls (4 out of 14; p = 0.022). (8) In conclusion, there are many studies and findings out there about silymarin. There needs to be more studies done until it can be used clinically. The other great aspect of silymarin is that unlike with medications taken for cirrhosis, silymarin did not show to have any adverse side effects. It is well tolerated and safe, so a trial is generally reasonable, although lack of better efficacy data prevents a stronger recommendation for its use. (4)

Once a patient is diagnosed with cirrhosis their prognosis is based on the severity of the damage at time of diagnosis. A person may not have symptoms of liver failure for 5 to 20 years. Since cirrhosis does not have symptoms until the end stage, sometimes patients don't realize they have cirrhosis until the damage is irreversible. Cirrhosis cannot be reversed once in the last stage. The scaring and damage remains even with discontinuation of alcohol. Death usually occurs within 5 years after the liver starts to fail.

My patient is a 61 year old male that is married. He lives with his daughter and wife, but there is martial conflict. He tends to eat out for most meals, skipping breakfast and eating his lunch and dinner at his favorite restaurant Whataburger. He is a barber who works long hours so he does not have time to cook or to grocery shop. He was admitted

on 9/13 and discharged on 9/26 so I did have time to meet with the man and talk to him. I visited him as often as I could to obtain adequate information. He was admitted to the hospital due to alcohol withdrawals, delirium tremors, and altered mental status.

He has a significant past medical history. He was diagnosed with cirrhosis back in 2008. At that time he was consulted to discontinue his alcohol intake and cut back on his pain pills. He also has a long history of reoccurring ascities. The patient has had hypertension, pancreatitis, gastiris-02/2005, chronic lower back pain, shortness of breath, alcohol abuse, and narcotic abuse for quite some time. The patient was originally prescribed narcotic pills for his back injury and pain. He started to abuse them, taking up to twenty pills a day. He is a regular smoker with a slow weight gain over the past five years. There is a history of insomnia that occurs when he has not drunk enough or taken enough pills.

This man came to the emergency room with an extreme altered mental status. Prior to his visit he ran out of money to support his drinking habit and narcotic abuse. He had not been drinking for three days and started to have hallucinations, agitation and confusion intermittently alternating with lethargy. He was very anxious and worried about when he was going to get his next “fix”. At this time they diagnosed his confusion with hepatic encephalopathy. He knew the doctors were weaning him from pain pills and he was very worried about not having them. He kept begging for more pills saying that he could not function normally without. This all related to his noncompliance in the past. He also is very focused on the mental dependence, rather than his health.

Once he was admitted to the floor, they diagnosed him with delirium tremors, hepatic encephalopathy due to cirrhosis, and pre-diabetics. They confirmed his diagnoses through

his high ALT and AST. ALT is the enzyme produced within the cells of the liver. The level of ALT abnormality is increased in conditions where cells of the liver have been inflamed or undergone cell death. AST It is less specific for liver disease. It may be elevated and other conditions such as a myocardial infarct (heart attack) (6). They confirmed the hepatic encephalopathy by the high ammonia levels. The pre-diabetes was confirmed by the A1C, which is plasma glucose concentrations over long period of time, know as glycated hemoglobin.

The treatment plan was Librium and Topamax, for his delirium tremors and anxiety. These medications work by decreasing the excitement in the brain. He was also prescribed lactulose to help rid his body of ammonia. He was put on an 1800 diabetic diet initially before I assessed him. This man is 68 inches and weights 115 kg. His IBW is 70 kg, with a BMI of 39. Since he is overweight I adjusted his weight to be 90kg. Although the patient is overweight, it is important to provide enough calories to promote liver healing. I felt this patient was L2-moderatly compromised, so I estimated his needs for maintenance to repair the liver and to prevent energy-protein malnutrition. I calculated his calories to be between 2025-2440 kcal (25-30 kcal/kg AdBW). Protein was calculated to be between 64-81 g (0.8-1.0 g/kg AdBW). I went lower due to the hepatic encephalopathy, but not as low as 0.5 g/kg to prevent the negative nitrogen balance.

When giving this patient a nutrition diagnosis I chose, excessive alcohol intake related to not ready for lifestyle change as evidence by intake record, DT’s, ascites, cirrhosis, and past medical history. I recommend 2,000 diabetic diet to promote liver healing, snacks three times a day between meals, low protein diet and discontinuation of alcohol. I felt the 1800 diet was too low for a cirrhotic patient. It is important to promote

healing and supplement the patient with vitamins and nutrients. I discussed with the patient cirrhosis nutrition therapy. It is important to be eating small amounts frequently making meal time a regular pattern. People with addiction have a pattern of impulses that make them repeat, so replacing that time with a meal can become a new healthy pattern. It is also important for them not to go too long without eating or getting hungry because that can create an urge of impulse. I also gave an education on diabetic diet since he was newly diagnosed. Lastly, I referred him to case manager since he came in with withdrawals due to lack of money.

With this patient it was important to monitor his access to food due to his lack of funds. Monitor alcohol intake with goal of discontinuation, narcotic abuse with goal of 2 pills per day. When looking at biochemical labs it is important to monitor his protein and glucose profile with a goal of the values being within normal limits. Monitor liver profile with a goal of trending towards normal limits. It is important to monitor his intake and labs in order to preserve lean body mass and maintain skin integrity. Lastly, to promote and monitor the nutrition quality of life that he deserves through the help that can be given to him.

My personal impression of this patient was that he has a long history of noncompliance, and from talking to him I felt he has just given up. He was more focused on how he was going to get his next drink or pill rather than the fact that he was in the hospital ill. He was very depressed. I tried to discuss with him baby steps, cutting back one day at a time or one week at a time. I think the main picture of reality was hitting him hard and it was overwhelming to him. I wanted to give him hope, but I feel he is most likely back out there drinking and abusing.

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