

Cirrhosis of the Liver Secondary to Alcoholism



Healthy



Cirrhosis

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[The Liver]

- Largest solid organ in the body.
- It performs many important functions, such as:
 - Manufacturing blood proteins that aid in clotting, oxygen transport, and immune system function.
 - Storing nutrients and returning some of the nutrients to the bloodstream.
 - Manufacturing bile
 - Glycogen, Glucoseneogenesis
 - Ridding the body of Toxins
 - Breaking down saturated fat and producing cholesterol

[Patient Profile]

- 61 y/o male. Married.
- Admit 9/13
- Discharge: 9/26
- Pt was admitted to the hospital due to ETOH withdrawal, DT's, and altered mental status.
- Occupation: Barber
- Home life: 1 daughter, but there is marital conflict.
- Eats out- usually fast food.

[PMHx]

- Cirrhosis
- Ascites
- HTN
- Pancreatitis
- Gastritis
- Chronic lower back pain
- Anxiety
- Depression



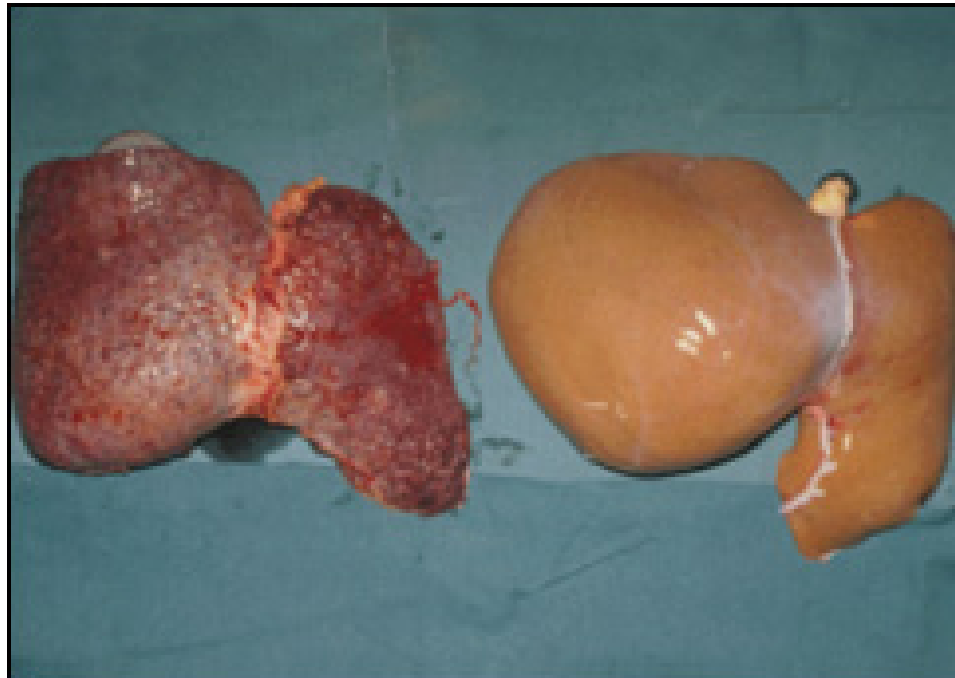
Health History

- ETOH abuse
- Narcotic abuse
- Tobacco use
- Wt gain
- Insomnia



Cirrhosis of the Liver

- Chronic disease that causes cell destruction and fibrosis of hepatic tissue, resulting in disrupted blood flow through liver.



Pathophysiology

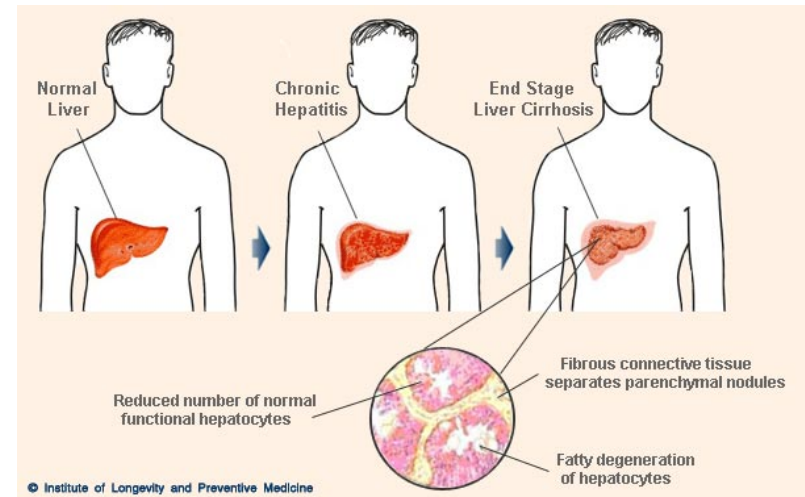
- ETOH-small water and lipid soluble molecule that can permeate all organs and their vital function.
- First Stage:
 - ETOH is preferred fuel source.
 - Hydrogen replaces 90% of fat as fuel in TCA cycle. Fat accumulates leading to fatty liver and raises TG levels in blood.
 - Strict abstinence will lead to full recovery

[Second Stage]

- ETOH consumption increases iron stores in the liver, contributes to HCV progression by inducing fibrosis.
- Can occur even when adequate nutrition is available.
- Symptoms: swollen liver, N/V, abdominal pain.
- Recovery is possible, but scar tissue remains.

Cirrhotic Stage

- Growth of connective tissue destroys liver cells.
- Fatty degeneration of hepatocytes.
- Inadequate bile acid secretion, results in fat malabsorption.
- The damage is irreversible



[Symptoms]

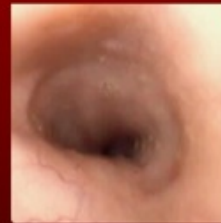
- Jaundice
- Loss of appetite
- Itching
- Easy bruising
- Agitation, excitement, or seizures (occur rarely)
- Disorientation
- Drowsiness or confusion
- Inappropriate behavior or severe personality changes
- Slurred speech

Complications

- Hepatic encephalopathy
- Ascites
- Edema
- Insulin resistance, T2DM
- Portal Hypertension
- Esophageal varices
- GI bleeding
- Malabsorption of fat

Effect of Portal Hypertension on the Esophagus

Normal



Varices



[Etiology]

- ETOH abuse
- Hepatitis C and B
- Autoimmune Hepatitis
- Biliary duct disorder
- Nonalcoholic Fatty Liver Disease
- Obesity
- Hemochromatosis

Nutritional Intervention

- Adequate kcal to promote healing of the liver.
- **Energy:** 30-35 kcal/kg to prevent protein sparing.
- **Protein:** 1.0-1.5 g/kg for non-hepatic encephalopathy
- **CHO:** 55-65% avoidance of simple sugars.
- **Lipid:** 25-30% -High in unsaturated fats, and essential fatty acids.
- **B-complex**, folic acid, Vitamin C, foods high in antioxidants.

[Tx Continued]

- Advanced stage:
 - ADEK supplementation- Due to decrease bile, failure to convert to active forms
 - Ca+ d
 - Folic acid
 - Thiamin
 - Mg
 - Bedtime meal to decrease- increased lipid peroxidation, gluconenogenesis.

[Acites Tx]

- Due to poor appetite and early satiety:
 - small frequent meals
 - aggressive oral supplementation
 - fluid restriction
 - 2 gm Na restriction

Hepatic encephalopathy Tx

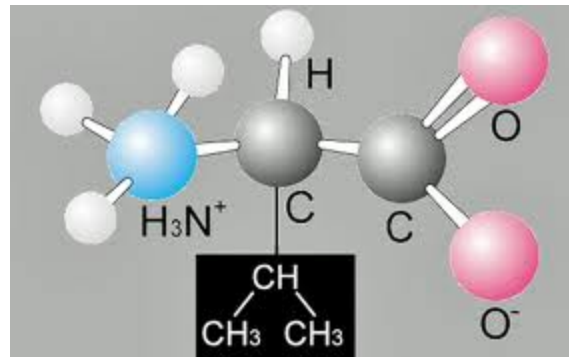
- How much protein??!
 - .5-.8 –lactulose
 - No lower then 40g /day to avoid negative nitrogen balance.
- BCAA up to .25 g/kg day
 - valine, leucine, isoleucine.
 - Metabolized in muscle and brain, not liver.
 - Promote protein synthesis, suppress protein catabolism, substrates for gluconeogenesis.
 - Catabolized to L-alanine and L-glutamine in skeletal muscle.

Role of Branched Chain Amino Acids in the Management of Hepatic Encephalopathy

Aftab Ahmed Soomro, World Journal of Medical Sciences 3 (2): 60-64, 2008

Class: A –Randomized control Study

Grade: +



Inclusion Criteria

- Patients with decompensated liver cirrhosis who met the following criteria:
 - HE grade II coma
 - Or a history of HE with current liver cirrhosis
 - Both males and females
 - Diagnosed based on clinical history, examination, laboratory (prothrombin time, albumin, ammonia level)

Criteria

- N= 48 patients
 - Group 1 (24 patients) placebo
 - 17 males, 7 females
 - Group 2 (24 patients) BCAA= 28 g/day
 - 17 males, 7 females
- Both groups received conventional therapy

Outcomes Ammonia $P < 0.001$

Ammonia levels	Initial	6 days	4 months
Group I Placebo	110.8	70.4	50.08
Group II BCAA	120.5	50.8	20.5

Recurrence of Hepatic Coma (observed during 4 months)



Recurrence	Group 1 Placebo	Group 2 BCAA
Up to 4 times	3	0
Up to 3 times	6	2
Up to 2 times	9	3
Up to 1 time	2	2
None	4 (16.7%)	17 (70.85) P=0.003

[Outcomes]

- Albumin level initially was < 2.8 in each group.
- From the placebo group the albumin rose to remain between 2.8 and 3.1 at 4 months
- However, the BCAA remained near or slightly above 3.1 at 4 months.

Mortality Outcomes



6 Months	Group I Placebo	Group II BCAA
Hospital stay > 14 days	66.7%	25% P < 0.001
Mortality	21 (67.5%)	14 (58.3%) P < 0.04

Conclusion of BCAA Study

- Long term BCAA supplementation showed an advantage compared to equal calorie and protein intake on Non-BCAA group.
- Overall lead to:
 - Lower Ammonia levels
 - Higher serum albumin
 - Lower death rates
 - Lower hospital LOS

The Use of Silymarin in the Treatment of Liver Diseases

- Double blind, placebo-controlled study with silymarin in alcohol-induced liver disease
 - Fintelmann et al
 - Class: A
 - Grade: +



[What is Silymarin?]

- Extracted from plant Milk Thistle
- Name from white veins on its leaves which exude a milky sap when broken.
- Extracts of the plant's black seeds have been used for centuries as a therapy for jaundice.
- Widely used in Europe since the 1960's.

Inclusion & Exclusion Overview

- N= 106 patients with liver disease.
- They were selected on the basis of elevated serum transaminase levels.
- Alcohol was forbidden during the trial.
- The patients were randomly allocated to silymarin or placebo.

Overview

- N= 97 patients completed the 4-week trial out of 106
- N= 47 silymarin
 - 230-600 milligrams per day divided into two to three doses
- N=50 placebo

Table V. Weighted mean values for liver function tests from the trials of DiMarzio et al.,^[91] Salmi et al.^[92] and of others indicated between treatments at admission (START) and at the end of study (END). Validity of a significance at the end of study is indicated if already significant at admission.

Test	START (±SD)	END (±SD)	Difference	No. of trials	No. of patients
AST (U/L)					
Silymarin	69.3 (±13.5)	32.5 (±10.0)	-36.8	3	79
Placebo	62.7 (±16.3)	46.5 (±12.9)	-16.2		83
ALT (U/L)					
Silymarin	105.6 (±11.6)	43.3 (±10.5)	-62.3	3	79
Placebo	78.5 (±10.3)	67.8 (±10.9)	-10.7		83
γ-GT (U/L)					
Silymarin	264.0 (±50.0)	111.0(±21.3)	-153.0	2	32
Placebo	224.0(±42.0)	170.0(±37.0)	-54.0		33
Total bilirubin (mg/dl)					
Silymarin	2.8 (±0.6)	1.5 (±0.5)	-1.3	1	15
Placebo	1.6 (±0.5)	1.5 (±0.6)	-0.1		14
AP (U/L)					
Silymarin	159.6 (±45.3)	144.6 (±36.3)	-15.0	2	32
Placebo	144.9 (±43.3)	169.6 (±44.2)	24.7		33

ALT = alkaline amino transferase; **AP** = alkaline phosphatase; **AST** = aspartate amino transferase; **γ-GT** = gamma glutamyl transferase

Outcomes

- Overall Silymarin lead to:
 - Lower AST, ALT
 - AP enzyme
 - There was no significant difference between conjugated bilirubin.
 - Changes occurred significantly more often in the silymarin treated patients (11 out of 15) than in controls (4 out of 14; $p = 0.022$)

Conclusions

- Many studies and findings
- Overall has shown a improvement in liver enzymes
- Symptoms?



[Prognosis]

- Cirrhosis cannot be reversed.
- The outlook depends on the severity of the damage at the time of diagnosis.
- A person may not have symptoms of liver failure for 5 to 20 years.
- Death usually occurs within 5 years after the liver starts to fail.

Application to Pt

- Initial Dx:
 - Altered Mental status
- Duration and intensity:
 - No drinking for 3 days
 - hallucinations, agitation and confusion intermittently alternating with lethargy.

Pts understanding of disease and Tx:

- Confused- hepatic encephalopathy.
- Weaning pain pills “He needs them to get going in the am.”
- PMHx noncompliance
- Focused on the mental dependence, rather than his health.

[Current Admission]

- Dx: DT's, hepatic encephalopathy due to cirrhosis, pre-diabetic, ascites
- Dx procedures: Pt has had cirrhosis for several yrs now.
 - High AST, ALT.
 - ALT is a more specific indicator of liver inflammation.
 - High Ammonia
 - H1C

[Current Admission]

- Tx: Librium, Topamax, Lactulose
Balanced diet, d/c of alcohol,
- Increased kcals to promote liver healing
- Decreased protein due to hepatic encephalopathy .

Nutrition Care Process

- Diet order: 1,800 diabetic diet
- Anthropometrics:
 - Ht: 5ft 8in.
 - Current Wt: 116 kg
 - IBW: 70 kg
 - % IBW: 165%
 - BMI 39
 - Adjusted BW: 81lbs

Biochemical Labs

Significant	9/13	9/18	9/22
Ammonia	32		54H
Glucose	179H	147H	123H
AST	146H	147H	133H
ALT	147H	165H	169H
A1C	6.3H		
Albumin	4.6N	4.3N	4.2N
MRSA	+		
Alcohol	<5		

History

■ Usual diet:

- Fast food two times day.
- No one at his home normally cooks.
- Grocery shopping only occurs as they need items.

■ Previous MNT:

- Instructed to quit drinking and reducing the amount of narcotics since 2008.
- Pt has tired to cut down both but feels that he can not function “normally” without it. He has a PMHx of noncompliance.

Evaluation of intake prior to admission: Fast food x2

Food Item	Kcal	Fat	Sat Fat	Protein	CHO	sodium	sugar
Whataburger	620	30g	10g	26g	58g	1262mg	13g
Medium Fries	480	27g	4.5g	5g	55g	347mg	1g
Sweet Tea	330				82g		82g
Total	1430 kcal	57g	14.5g	31g	185g	1609mg	96mg
%		37%		10%	53%		

Current intake: BMC House menu

Diet Order	Kcal	PRO	Na	K	PO4	CHO
Diabetic	1800	85g	2.5g	2.5 g	1g	225g

Estimated Needs

- L2 moderate compromise.
- Estimated needs for maintenance to repair liver and to prevent energy-pro malnutrition:
 - Kcal: 2025-2440 kcal (25-30 kcal/kg AdBW)
 - Protein: 64-81 g (0.8-1.0 g/kg AdBW)
 - Fat: 62-74 g (25-30% of 2232 kcal)
 - CHO: 306-362 g (55-65% of 2232 kcal)

Nutrition Diagnosis

- Excessive alcohol intake related to not ready for lifestyle change as evidence by intake record, DT's, ascites, cirrhosis, and PMHx.

Interventions

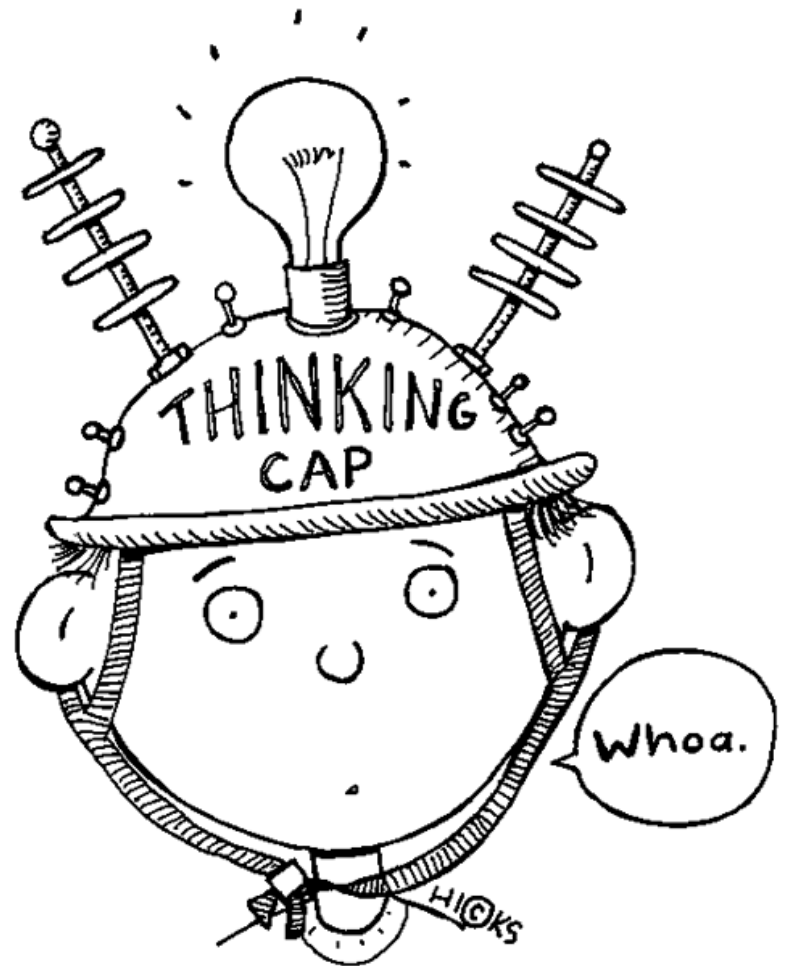
- Recommend 2,000 Diabetic diet to promote liver healing.
- Snacks TID between meals.
- Recommend low protein diet
- D/c alcohol intake.
- Education on Cirrhosis Nutrition Therapy, Diabetic diet
- Refer to case manager

Monitor/Evaluation

- Monitor access to food
- Monitor Wt
- Protein and glucose profile WNL.
- Liver profile trending towards NL
- Preserve LBM
- Maintain skin integrity
- Promote nutrition quality of life

Personal Impression

- PMHx of noncompliance
- Confused- HE
- Weaning
- Reality



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[Any Questions?]

