Nutrition management in liver diseases

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The main functions of liver

- **Synthesis of blood protein: albumin, transferrin, prealbumin, prothrombin**
- **Excretion of bile: required for digestion and absorption of fat**
- **Metabolism of toxin: drugs, bilirubin, ammonia**
- **Control for traffic of nutrients during fasting and fed states: glycogen storage, gluconeogenesis and glycogenolysis**
- **80-90% of liver cell have to be injured before these functions are impaired**
- **Nutritional deficiency and protein calorie malnutrition (PCM): often present in chronic liver diseases, but can be seen in acute liver diseases especially when associated with hepatic insufficiency**

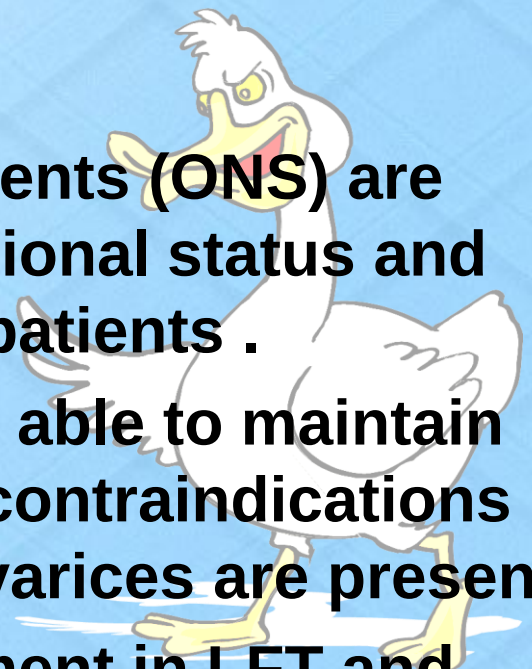
Nutritional status of patients with alcoholism

- **Moderate alcohol intake (16% of total calories): associated with slightly increase total energy intake**
- **These levels or more (up to 23%) of alcohol intake: associated with a substitution of alcohol for CHO in diet**
- **In those consume >30% of total calories from alcohol**
  - **significant decrease in protein and fat intake**
  - **intake of vitamin A, C and thiamine may below RDA**
  - **calcium, iron and fiber intake are lowered**

Effects of ethanol on digestion and absorption

- **Alcohol consumption is associated with motility changes in GI tract, including diarrhea, that affect digestion and absorption of nutrients**
- **Ethanol effects may be direct or indirect, acute or chronic**
- **Intestinal malabsorption, one of the most striking changes, secondary to folic acid deficiency (results from diminished folic acid intake and utilization accompanying alcoholism)**

Nutrition therapy in alcoholic hepatitis



- **In general, oral nutritional supplements (ONS) are recommended as it improves nutritional status and survival in severely malnourished patients .**
- **Use tube feeding if patients are not able to maintain adequate oral intake and when no contraindications like ileus. (even when esophageal varices are present)**
- **Benefit of short-term EN: improvement in LFT and decrease in length of hospital stay but no decrease in mortality**
- **Use simple bedside methods such as Subjective Global Assessment (SGA) or anthropometry to identify patients at risk of undernutrition.**

Plauth M. Clinical Nutrition (2006) 25, 285–294

Subjective Global Assessment of Nutritional Status

A. History

1. Weight change

Overall loss in past 6 months: _____ kg Percent loss _____

Change in past 2 weeks: _____ increase _____ no change _____decrease

2. Dietary intake change relative to normal

No change _____

Change: duration _____ weeks ____ months

Type: sub-optimal solid diet _____ full liquid diet _____ hypocaloric liquid diet _____ starvation _____

3. Gastrointestinal symptoms (persisting more than 2 weeks)

None _____ Nausea _____ Vomiting _____ Diarrhea _____ Anorexia _____

4. Functional capacity

No dysfunction _____

Dysfunction: duration _____ weeks _____ months

Type: working sub-optimally _____ ambulatory _____ bedridden _____

5. Disease and its relationship to nutritional requirements

Primary diagnosis: _____

Metabolic demand / Stress: none _____ low _____ moderate _____ high _____

B. Physical Examination

(for each specify: 0 = normal, 1+ = mild, 2+ = moderate, 3+ = severe)

Loss of subcutaneous fat (triceps, chest) _____

Muscle wasting (quadriceps, deltoids) _____

Ankle edema _____ Sacral edema _____ Ascites _____

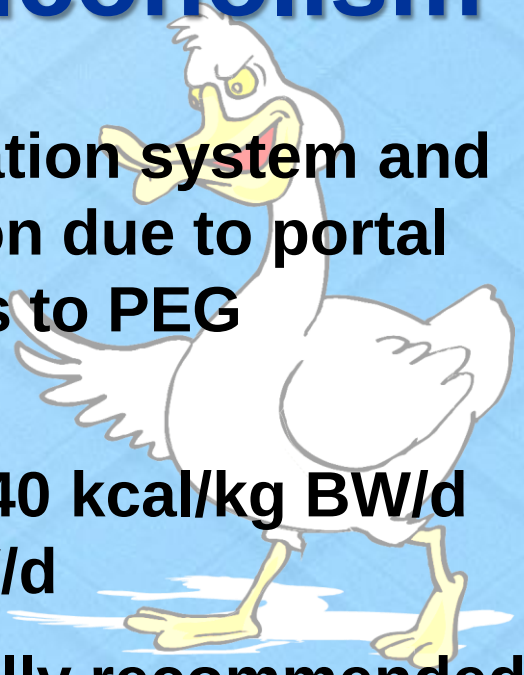
C. Subjective Global Assessment Rating

Well nourished A _____

Moderately (or suspected) of being) malnourished B _____

Severely malnourished C _____

Enteral nutrition in alcoholism



- **Ascites, impairment of the coagulation system and porto-systemic collateral circulation due to portal hypertension are contraindications to PEG placement.**
- **Recommended energy intake: 35–40 kcal/kg BW/d and protein intake 1.2–1.5 g/kgBW/d**
- **Whole protein formulae are generally recommended.**
- **Consider using more concentrated high-energy formula in patients with ascites.**
- **Use BCAA-enriched formulae in patients with hepatic encephalopathy arising during enteral nutrition.**

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Parenteral nutrition (PN) in alcoholic hepatitis (AH)

- **Start PN immediately in moderately or severely malnourished pts, who can't be fed sufficiently either orally or enterally.**
- **Give i.v. glucose (2–3 g/kg/d) when patients have to abstain from food for more than 12 h.**
- **Give PN when fasting period lasts ≥ 72 h.**
- **Energy: provide energy to cover 1.3 X REE**
- **In cirrhotics without ascites: use actual body weight for calculation of basal metabolic rate using formulae (eg. Harris Benedict equation).**
- **In patients with ascites: use ideal weight**
- **Give glucose to cover 50–60 % of non-protein energy requirements.**

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Parenteral nutrition (PN) in alcoholic hepatitis (AH)

- **Use lipid emulsions with a content of n-6 PUFA lower than in traditional pure soybean oil emulsions and should cover 40–50% of non-protein energy requirements.**
- **Compared to traditional soy bean based LCT emulsions (n-6:n-3=8:1), new fat emulsions have a lower content in n-6 PUFA due to admixture of MCT, olive oil and/or fish oil rendering them less suppressive to leukocyte and immune function and less stimulant of pro-inflammatory modulators.**
- **Amino acids: 1.2 g/kg/d in pts who are not or only moderately malnourished, and 1.5 g/kg/d in severely malnourished.**
- **Water and fat soluble vitamins, minerals and trace elements must be administered daily from D1 of PN to cover daily requirements**

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Parenteral nutrition (PN) in alcoholic hepatitis (AH)

- **Administer vitamin B1 prior to starting glucose infusion to reduce risk of Wernicke's encephalopathy.**
  - **For prophylaxis: thiamine 250 mg i.m. daily x 3-5 d**
  - **For treatment: thiamine 500 mg i.v. tid x 2–3 d**
- **In jaundiced pts: vitamin K def due to cholestasis-induced fat malabsorption may require i.v. Vit K for correction.**
- **Routine administration of twice normal daily requirement of zinc (10 mg/d) is recommended.**
- **Monitoring blood sugar determinations to detect hypoglycemia and to avoid PN related hyperglycemia.**
- **Monitor P, K and Mg levels and additional P, K and Mg will be required when refeeding malnourished patients.**

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Fulminant liver failure

- **Fulminant liver failure without treatment results in death within days.**
- **Stabilization of metabolism is mandatory, it is more important than nutritional therapy aimed at meeting daily requirements.**
- **Hypoglycemia is a frequent metabolic disturbance and merits particular attention and therapy, such as (par)enteral glucose administration.**
- **Patients with acute liver failure should receive EN via nasoduodenal tube.**
- **No recommendations concerning a disease specific composition of enteral formula can currently be given.**
- **A direct comparison between standard formula and BCAA enriched formula has not yet been made.**
- **Monitor glucose, lactate, triglycerides and ammonia plasma levels closely and used as surrogate markers of substrate utilization.**

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Parenteral nutrition in fulminant liver failure

- **Start artificial nutrition when patient is unlikely to resume normal oral nutrition within 5–7 days.**
- **Use PN when patients cannot be fed adequately by EN.**
- **Energy: 1.3 X REE.**
- **Consider using indirect calorimetry to measure individual energy expenditure.**
- **Give i.v. glucose 2–3 g/kg/d for prophylaxis or treatment of hypoglycemia.**
- **Xylitol or sorbitol in exchange for glucose is no benefit. (both have to be metabolized by liver before they can be utilized)**
- **In case of hyperglycemia, reduce glucose infusion rate to 2–3 g/kg/d and consider i.v. insulin.**

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Parenteral nutrition in fulminant liver failure

- **Consider using lipid 0.8 – 1.2 g/kg/d together with glucose to cover energy needs in insulin resistance.**
- **Amino acid is not mandatory in hyperacute LF.**
- **In acute or subacute LF: use AA 0.8–1.2 g g/kg/d in PN or protein 0.8–1.2 g/kg/d in enteral nutrition**
- **No clinical trial has shown a benefit of BCAA solution in comparison to standard solutions.**
- **Hypoglycemia is a clinically relevant and common problem resulting from loss of hepatic gluconeogenetic capacity, lack of glycogen and hyperinsulinism.**
- **Monitoring blood sugar determinations to detect hypoglycemia and to avoid PN related hyperglycemia.**
- **Repeat blood ammonia measurement to adjust amino acid provision.**

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Causes of malnutrition in cirrhosis

1. Decrease in oral intake

anorexia, nausea, vomiting, early satiety, taste abnormalities (Zn and Mg def), alcohol abuse, iatrogenic due to restrict diets or NPO status, medications

2. Maldigestion and malabsorption

fat malabsorption due to cholestasis or chronic pancreatitis

water-soluble vitamin malabsorption due to alcohol abuse

calcium and lipid-soluble vitamin malabsorption due to cholestasis

3. Metabolic abnormalities

glucose intolerance, increased protein and lipid catabolism similar to sepsis, trauma or other catabolic states

Liver cirrhosis

- **Both prevalence and severity of malnutrition are independent of the etiology of liver disease but do correlate positively with severity of the illness.**
- **The prevalence of protein energy malnutrition increases from 20% in Child-Pugh class A to over 60% in class C.**
- **Poor oral food intake is a predictor of an increased mortality.**
- **Cirrhotics with the lowest spontaneous energy intake showed the highest mortality.**
- **In cirrhotics, after an overnight fast glycogen stores are depleted and metabolic conditions are similar to prolonged starvation in healthy individuals.**

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Nutrition therapy in cirrhosis

- **Use simple bedside methods such as SGA or anthropometry to identify patients at risk of undernutrition.**
- **Use body cell mass measured by bioelectric impedance analysis (BIA) to quantitate undernutrition, despite some limitations in patients with ascites.**
- **Recommended energy intake 35–40 kcal/kgBW/d and protein 1.2–1.5 g/kgBW/d**
- **Use supplemental enteral nutrition when patients cannot meet their caloric requirements through oral food despite adequate individualized nutritional advise.**
- **Late evening CHO snack is associated with improved protein metabolism in cirrhotic patients.**

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Nutrition therapy in cirrhosis

- **Whole protein formula are generally recommended**
- **Consider using more concentrated high-energy formula in patients with ascites.**
- **Use BCAA-enriched formula in patients with hepatic encephalopathy arising during enteral nutrition.**
- **The use of oral BCAA supplementation can improve clinical outcome in advanced cirrhosis.**
- **Enteral nutrition improves nutritional status and liver function, reduces complications and prolongs survival in cirrhotics and is recommended.**

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Parenteral nutrition in liver cirrhosis

- **Start PN immediately in moderately or severely malnourished pts, who can't be fed sufficiently either orally or enterally.**
- **Give i.v. glucose 2–3 g/kg/d when pts have to abstain from food > 12 h.**
- **Give PN when the fasting period lasts ≥ 72 h.**
- **Due to somnolence and psychomotor dysfunction oral nutrition is often insufficient even in mild encephalopathy (I–II) → tube feeding may be required.**
- **Consider PN in pts with unprotected airways and encephalopathy when cough and swallow reflexes are compromised.**

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Parenteral nutrition in liver cirrhosis

- **Use early postoperative PN if pts cannot be nourished sufficiently by either oral or enteral route.**
- **After liver transplantation, use early postoperative nutrition; PN is second choice to EN.**
- **Energy: 1.3 x REE and amino acids 1.2–1.5 g/kg/d**
- **Give glucose 50-60 % of non-protein energy requirements.**
- **Reduce glucose infusion rate to 2–3 g/kg/d in case of hyperglycemia and consider i.v. insulin.**
- **Use lipid emulsions with a content of n-6 PUFA lower than in traditional pure soybean oil emulsions.**

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Parenteral nutrition in liver cirrhosis

- **Micronutrients: give water soluble vitamins and trace elements daily from D1 of PN.**
- **Monitoring blood sugar determinations to avoid PN related hyperglycemia.**
- **Monitor phosphate, potassium and magnesium levels when refeeding malnourished patients.**
- **Currently, no recommendations can be made regarding donor or organ conditioning by use of i.v. glutamine or arginine to minimizing ischemia/ reperfusion damage.**

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Amino acid solution

- **1.2 g/kg/d in compensated cirrhosis without malnutrition**
- **1.5 g/kg/d in decompensated cirrhosis with severe malnutrition**
- **Use a standard solution in mild encephalopathy (≤II°)**
- **Use a liver-adapted complete amino acid solution in more severe encephalopathy (III° – IV°).**
- **Such solutions contain an increased amount of BCAA (35-45%) and lower content of aromatic AA, methionine and tryptophan.**
- **A meta-analysis: improvement in mental state by BCAA-enriched solutions, but no definite benefit in survival.**
- **Cochrane analysis of 7 RCTs studying 397 pts with acute HE, parenteral BCAA had a significant, positive effect on course of HE, but not on survival.**

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Transplantation and surgery

- **In malnourished cirrhosis patients, risk of postoperative morbidity and mortality is increased after abdominal surgery.**
- **After visceral surgery in cirrhotics, lower complication rate when gives postoperative PN instead of just fluid and electrolytes.**
- **After liver transplantation postoperative nutrition confers advantage of shorter periods on mechanical ventilation and shorter ICU stay (compared to fluid and electrolyte infusion.**
- **In comparison between PN and early enteral nutrition, both strategies proved to be equally effective with regard to maintenance of nutritional state.**
- **Fewer viral infections and improved nitrogen retention in patients on enteral nutrition started as early as 12 h after transplantation.**

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Transplantation and surgery

- **Preoperative:**
  - **Follow recommendations for cirrhosis.**
  - **For children awaiting transplantation consider BCAA administration.**
- **Postoperative:**
  - **Initiate normal food/enteral nutrition within 12–24 h postoperatively to minimize perioperative—in particular infectious complications.**
  - **Recommended energy intake: 35–40 kcal/kgBW/d, protein intake: 1.2–1.5 g/kgBW/d**
  - **No difference between standard or high BCAA solution**
  - **Use NG tubes or catheter jejunostomy for early enteral nutrition.**

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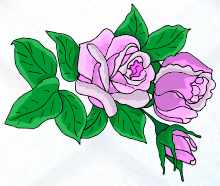
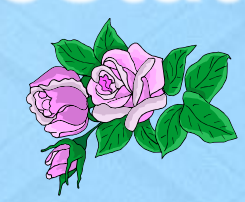
Nutrition therapy in cholestatic liver diseases





- Fat-soluble vitamin A, D, E, K may be depleted (decreased absorption and intake).
- Monitor serum values routinely and supplement when necessary
- Increased prothrombin time: result of vit k def
- Parenteral lipid-soluble vitamin supplementation (A, D, E, K) if steatorrhea > 10 g/d
- Limit fat intake while provide adequate energy and protein
- Improvement of fat intolerance with small amount of fat throughout the day
- Oral calcium supplement (1500 mg/d adequate amount for absorption and oxalate binding) taken between meals
- If weight loss, consider oral supplements with MCT oil to provide additional energy

Nutrition therapy in fatty liver

- **Weight loss of 10% of body weight if obesity**
- **Control of hyperglycemia and hyperinsulinemia with low-CHO diets and medications**
- **Control of hyperlipidemia with low-fat diets or medications**
- **Abstinence from alcohol**

Thank you for your attention