Nutrition in acute and chronic liver disease

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Acute **L**iver **F**ailure

Definition

- rapid development of hepatocellular dysfunction, specifically coagulopathy and mental status changes (encephalopathy) in a patient without known prior liver disease

- Viral infections
- Toxins
  - Alcohol
  - Drugs
  - α-amanitin

Treatment

- Supportive treatment
- Treatment of the underlying cause
- Liver transplantation

- Nutritional support
  - Aim
    - Assist liver regeneration
    - Support the patient before transplantation

Nutritional support

- Usually good nutritional status
- Risk of hepatic come and fatal brain oedema

- No RCT available

Metabolic response

- Liver insufficiency
- Stress metabolism

- Glucose metabolism
- Lipids metabolism
- Protein metabolism

Glucose metabolism

- ↑ plasma insulin
- ↓ glucose elimination rate (~50%)
- ↓ insulin sensitivity (~15%)
- ↑ glucagon concentration

- Glucose intolerance

- Monitor blood glucose level

Lipids metabolism

- Absent hepatic ketogenesis
  - ↓ ketone bodies
  - ↓ FFA
- Intravenous lipid emulsions are usually well tolerated

Protein metabolism

- ↑ AA plasma level
- ↓ **B**ranched **C**hained **A**mino **A**cids (Val, Leu, Ile)
- ↑ Trp, aromatic and sulphur AA

- Hiperammonemia

Requirements

- ↑ REE ~ 20-25%
- Protein – BCAA?
- Carbohydrate/glucose
- Fat/lipid emulsion

Route

- No specific recommendations
- EN
- PN

Chronic **L**iver **F**ailure

Definition

- disease process of the liver that involves a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis

- Hepatic encefalopathy (HE)

- Malnutrition
- Macro-/ micronutrients deficiencies

CLD

- Viral infections
- Toxins
  - Alcohol
  - Drugs
- Metabolic
  - NAFLD
  - Wilson’s disease
  - Hemochromatosis
- Autoimmune

Hepatic encephalopathy

- severe metabolic disorder with neurologic manifestations (flapping tremors, coma) in patients with liver failure

- Liver insufficiency
  - ↓ urea cycle of liver
  - hyperammonemia

- Skeletal muscles
  - Ammonia + **BCAA** → Gln

Nutritional assessment

- NRS
- SGA
- Antropometrics (MAC)
- Bioimpedance
  - ascites
- BMI
  - ascites

- Indirect calorimetry

Malnutrition

- Loss of appetite
  - Reduced clearance of satiation mediators
  - Splachnic cytokines
  - Ascites, intestinal oedema
- Malabsorption
  - Bile obstruction
- Increased requirements

Increased requirements

- 34% REE >120%

- Protein
  - ↑1,2g/kg/d
  - BCAA

- No indication that an adequate dietetary intake aggravated HE
- Protein restriction aggravates malnutrition

Requirements

- Energy: 35-40kcal/kg/d
- 1,3x REE

- Glucose
  - 20-30% intolerance
  - 50-60% of non-protein energy

Requirements

- Protein
  - 1,2g/kg/d for maintenance
  - 1,5-1,8g/kg/d for increase demands

- BCAA
  - Protein intolerance
  - Worsening of existing HE

Vitamins

- Thiamine
- Fat-soluble vitamins (steatorhea)
- Normal supplementation

Route of feeding

- No specific recommendation

- Normal food
- ONS
- TF
  - NGT – oesophageal varices?
  - Whole protein formula
  - PEG not recommended
    - ↑ Complications rate
- PN

Dietary counseling

- 4-6 meals (improve nitrogen balance)
- Reduce length of the evening and night period of starvation
  - „Evening meal”

Monitoring – ALD/CLD

- Standard monitoring
- Plasma glucose
- Plasma TG
- Plasma ammonium
- Mental status

Take home message

- Patients with ALF have glucose intolerance
- Patients with ALF have severe hyperammonemia leading to brain oedema
- Patients with ALF have increased REE

Take home message

- ALD - good nutritional condition / CLD - malnourished
- Nutrition improves survival in CLD
- Hepatic encephalopathy is not contraindication to NS
- Worsening encephalopathy caused by protein intolerance - BCAA

- European Society for Clinical Nutrition and Metabolism
- www.espen.org

- ESPEN The Life Long Learning (LLL) programme in Clinical Nutrition and Metabolism
- www.lllnutrition.com