What is it?

any condition that damages the liver and affects its ability to function

Progression: Healthy liver → Fatty Liver → Inflammation (hepatitis) → fibrosis of liver → cirrhosis liver (irreversible)

AKA: Healthy liver → NAFLD → NASH → cirrhosis

Causes

Hep A & C	autoimmune disease
liver cancer	toxins (alcohol, smoking)
metabolic conditions	obesity
DM	hyperinsulinemia

Biochem

Marker	Expected in LD	Explanation
LFT (ALP, GGT & bilirubin)	↑	Bile flow is blocked (biliary obstruction, intrahepatic extrahepatic). They can also rise with liver tumours or obesity
AST & ALT	↑	↑ ALT only = mild hepatic damage. ↑ ALT and AST = hepatic damage. Markers of recent (hours/days) liver injury (though AST can also rise with muscle damage. Damage to liver cells = release of AST and ALT (found in hepatocytes).

Biochem (cont)

CRP	↑	Marker of inflammation
Albumin & prealbumin	↓	Indicate cirrhosis and end stage liver disease. Albumin is synthesised in the liver. Poor indicator of nutritional status in liver disease patients. Correlate with the progression and severity of LD.
Vit A, D, E, K	↓	Fat malabsorption (↓ bile)
Zinc	↓	Develops from low intake, poor absorption (↓ bile, ↓ albumin), and diuretic (treat ascites) loss; linked to taste changes, glucose issues, encephalopathy, poor healing, and weakened immunity.
Thiamine	↓	Stores & activates @ liver. ↓ hepatic reserves and alcohol intake preventing the conversion into its active form.
B12	↓ (tissue), ↑ (serum)	Reduced hepatic stores. Liver cells release stored B12 when damages.

Biochem (cont)

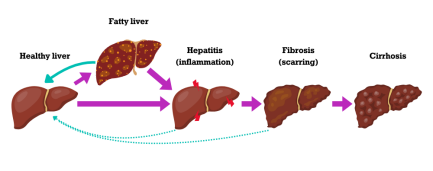
Folic Acid	↓	Liver stores & metabolises folate into its active form. Reduced hepatic reserves
Calcium	↓	Vitamin D deficiencies
Selenium	↓	↓ absorption and intake

Functions of the liver

P, CHO, F metabolism	Drug metabolism
Stores: fat sol-vits, zinc, iron, copper and magnesium	**Secretes bile – fat absorption**
Synthesis: albumin, prealbumin, retinol-binding protein & clotting factors	**Stores glycogen**

+ many more!

Stages



Child Pugh Score

5-6 points	**Grade A** = 85-100% survival rate. Well compensated.
7-9 points	**Grade B** = Significant functional compromise. 60-80% survival
10-15 points	**Grade C** = decompensated disease. 35-45% survival rate

NIS

Liver stores glycogen = **unable to regulate blood sugar.** Needing small regular meals

Maldigestion & malabsorption: reduced bile (fat malabsorption), pancreatic insufficiency

Altered taste, nausea

NIS (cont)

Portal hypertension: in advanced liver disease. Vessels in liver blocked (eg scaring) = Blood merges into portal vein in liver = ↑ pressure = backflow of blood

Oesophageal varices - dilated Abnormal veins – may bleed = reduced oral intake, dysphagia, text-mod

Ascites - Abdominal swelling caused by accumulation of fluid. Malnutrition ↑s risk. Can't use a PEG. early satiety, fluid & NA restrictions

Encephalopathy (HE)– alters brain function. Confusion, memory, shaky, trouble talk/walk. inability to self-feed, dysphagia, tiredness, malaise (general feeling unwell). Stage 0 (no abnormalities)-4 (coma)

Jaundice: liver cirrhosis or liver cancer. Serum bilirubin >2.5-3 mg/dL. Loss of liver function to metabolise bilirubin (damaged hepatocytes) = bilirubin builds up

Nut Reqs

USE DRY WEIGHT

Compensated liver disease: 100-14-5kJ/kg/day (CQHHS)

NASH, cirrhosis, transplantation, hepatic encephalopathy: 145 – 165 kJ/kg/day (CQHHS), ESPN = 30-35kcal/kg

Protein: 1.2 -1.5 g/P/kg/d (CQHHS). 1.5 for cirrhosis

Fat: Restrict if: signs of fat malabsorption. Don't remove as it's a source of fat-sol V & concentrated energy

Na: 2g/day (CQHHS)

Risk of deficiency (alcoholic liver disease): folate, Vit C, B group

Prevent toxicity: Copper & magnesium

Thiamine: 100mg OD-TDS

Vitamin D: 400-800IU/day

Nut Reqs (cont)

Vitamin K: 10mg every 4 weeks K: 10mg every 4 weeks

Calcium: 1200-1500mg/day

Zinc: multivitamin can be used

Intervention

NAFLD: Weight loss – similar to T2DM – **intensive lifestyle.**	↓ weight, lifestyle behaviours, euglycemia/normal lipids/normal BP = reduce portal hypertension

CLD (NASH, Cirrhosis): HPHE. **Malnutrition** strategies. Maintain muscle mass.

	Screen malnutrition (RFH-NPT) and Sarcopenia (SARC-F) - Prevent sarcopenia, severe fatty liver, infections, LOS, mortality, HE
	Varices: softer foods without sharp edges to avoid bleeds
	Ascites – Na 60 mmol/day (ESPEN)
	EN (NGT) – intake is <70-80% of reqs (CQHHS) eg oesophageal varies. 1.5-2 kcal feed.
	PN – liver failure - 2-in-1 bag (no fat)
	Thiamine supplementation before PN to prevent Wernicke-Korsakoff syndrome
	Minimise fasting
	BCAA Supps (12-14g/day delivered in the evening before sleep)

Intervention (cont)

For alcoholic liver disease (not w/ cirrhosis): initial remove alcohol & treatment withdrawal S/S. Correct deficiencies.	V&M affected by alcohol: ↓ folic acid, thiamine, B6, niacin, Vit K, Vit C, Vit D, Vit A, iron, K, Mg
Refer	EP. Speechie
Consider	DO THEY HAVE ASCITIES? Swallowing function, ability to self-feed, texture-mod, refeeding risk

Strategies

Malnutrition strategies - ONS, EN/PN

6-8 meals/day. Max. 6-7 hours without any intake (CQHHS)

Monitoring

tolerance to feeds	wt
N/V/D	nut deficiencies
re-asses reqs	restriction changes

Example PESS

NAFLD: Excessive oral intake, Food and nutrition knowledge deficit

NASH/Severe CLD: malnutrition, Inadequate protein-energy intake, Inadequate oral intake

Guidelines & References

ESPEN guideline on clinical nutrition in liver disease (2019)

ESPEN practical guideline: clinical nutrition in liver disease (2020)

FEEDS Gastroenterology and liver disease

Central Queensland Nutrition guidelines for liver disease management 2019