Consultatieve hepatologie

M. Guichelaar

Consultatieve hepatologie

Vragen over leverenzymstoornissen en aanverwante punten

-Leverenzymstoornissen?

-Vena porta trombose: behandeling nodig?

-Patient heeft ascites: komt dit door leverpathologie?

-Patient is suf en heeft leverproefstoornissen = is er een relatie?

Vragen over leverpatienten

-mag patient paracetamol?

-mag patient lorazepam?

-kan hij geopereerd worden? Wat zijn de risico's?

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Consultatieve hepatologie

Vaak vragen over leverenzymstoornissen

ORIGINAL RESEARCH

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Prevalence of Liver Disease and Utilization of a Hepatology Consultation Service at an Urban Tertiary Care Hospital

452 pts without known liver disease:
-218 (48,2%) had liver biochemistry testing
→ 192 (88.1%) had abnormal liver enzyme and / or function tests

Clin Invest Med 2015; 38 (6): E358-E361.

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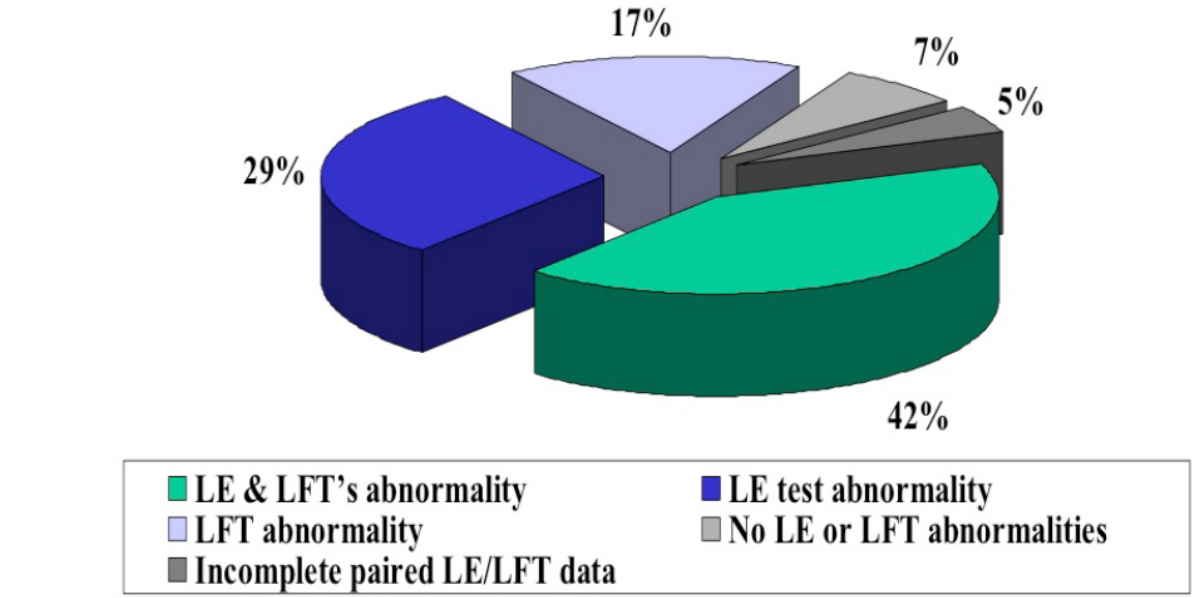

FIGURE 1. Results of liver biochemistry testing in 218 patients with no history of liver disease admitted to a general medical ward for non-hepatic disorders. LE and LFT refer to liver enzyme and function tests, respectively.

1) Uit algemene populatie: prevalentie abnormale leverproeven = 10-20%
   = associatie met BMI

2) Geassocieerd met indicatie ziekenhuisopname = sepsis, myocardinfarct, hartfalen

3) Geassocieerd met niet-lever problematiek = hemolyse, spierziekte

4) Belangrijk = toxisch component tijdens opname = DILI = drug induced liver injury

Analyse van leverenzymstoornissen in ziekenhuis

Speurwerk: anamnese / gegevens / LO

- Opname indicatie
    - Sepsis
    - Cardiaal lijden / myocardinfarct
- Toxiciteit:
    - Alcohol
    - Welke medicatie tijdens en voorafgaand aan medicatie
- Cormorbiditeit:
    - DM, cardiovasculair lijden
- Aanwijzingen pre-existente leveraandoening
    - Stigmata (spider naevi, ascites, etc)

Aspect leverenzymstoornissen

-Pre-existente leverproefstoornissen

-Trombocytopenie?

-Aanwijzingen leversynthese / excretie dysfunctie?

-Aspect leverenzymstoornissen
Cholestatisch?
Parenchymateus?
Mixed patroon?

Beeldvorming:
-leversteatose?
-aspect lever (cirrotisch?), aanwijzingen portale hypertensie
-vena porta trombose

*Fibrosccan

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-vena porta trombose

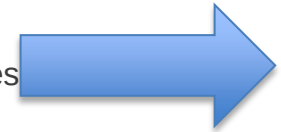
Leverenzymstoornissen

Parameters leverschade

Leversynthese parameters

1) **Patroon:**
   - -Parenchymateus (ASAT, ALAT)
   - -Cholestatisch (Bilirubine, AF, GGT)
   - -Mixed

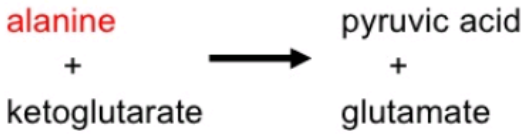
2) **Ernst van afwijkingen**: ASAT / ALAT
   - -Snelheid stijgen transaminases
   - -Hoogte afwijkingen



Parenchymal disease: AST, ALT = *Damage to hepatocytes*

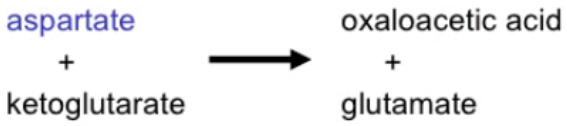
Transaminases are enzymes that catalyze the transfer amino groups from amino acids to α-keto acids.
These enzymes are important in gluconeogenesis.

ALT (alanine aminotransferase)



-In cytosol, mainly in liver (hepatocytes)
- Released in blood by injured hepatocytes
- **Half –life 47 hrs**

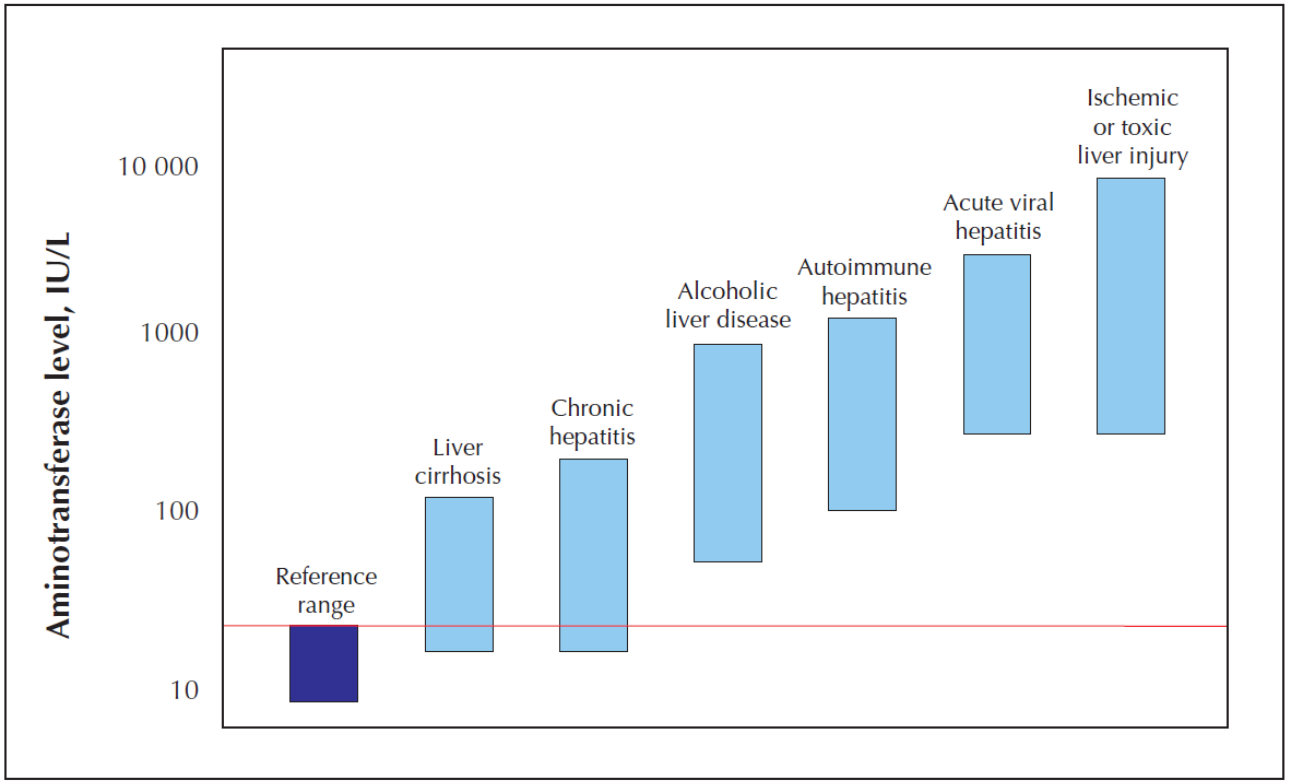
AST (aspartate aminotransferase)



-In cytosol (cAST) and mitochondria (mAST) of hepatocytes.
- But also non-hepatic (skeletal, muscle, blood)
- **Half-life: total AST 17 hrs, 87 hrs for mitochondrial AST**

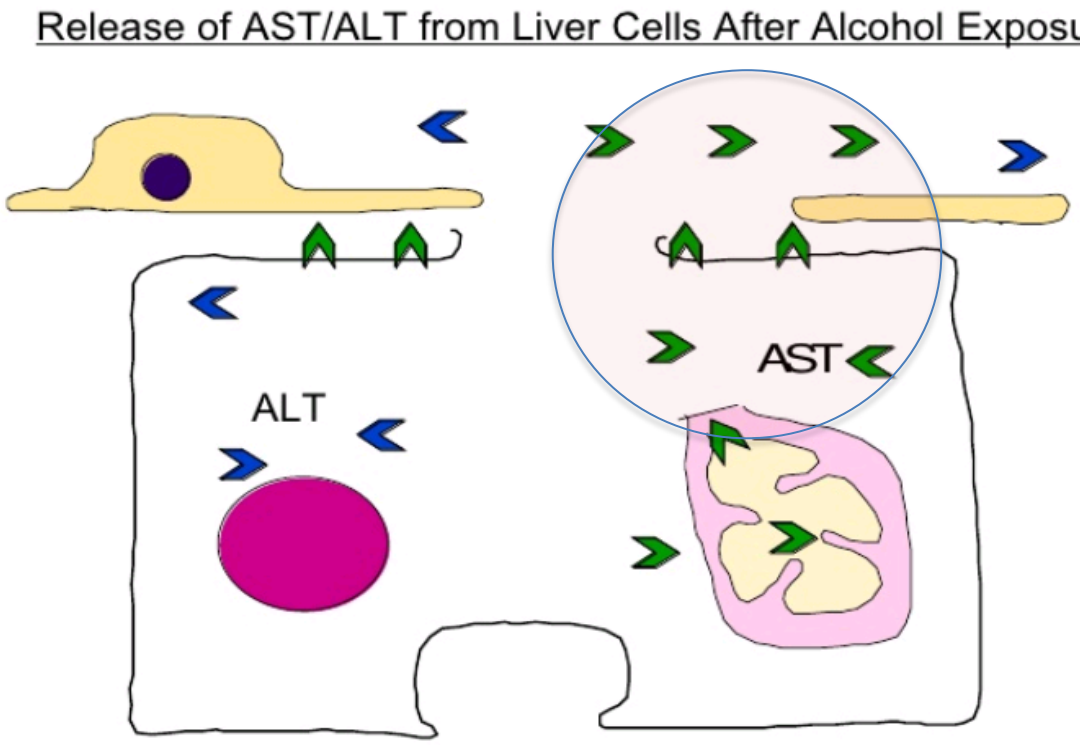
Pattern of ALT and AST

-**AST / ALT ratio > 2 =** Alcohol, ischemia, cirrhosis



Question: Why with alcoholic hepatitis AST >ALT?

Question: Why with alcohol AST higher than ALT?



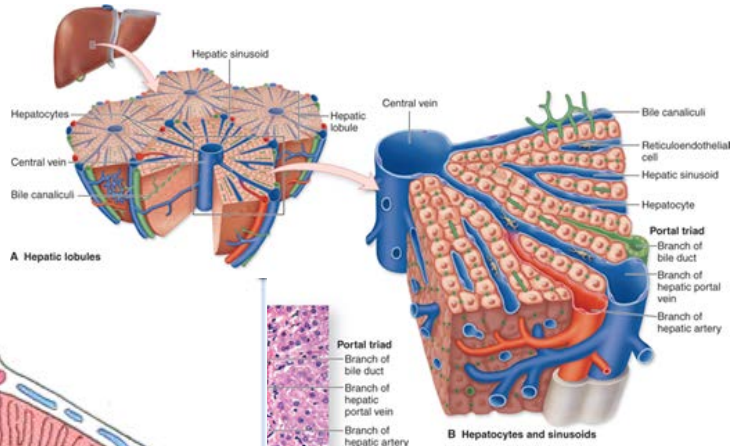
Mitochondrial AST = long half life (> 80 hrs)

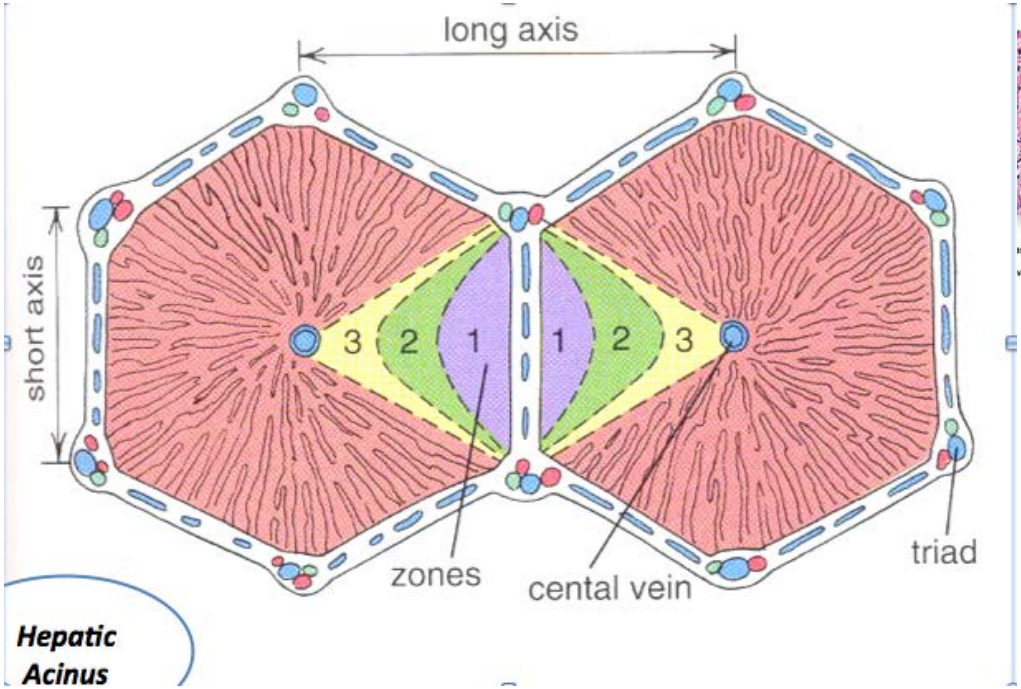
Question: Why with ischemia relative higher AST than ALT?

Question: Why with ischemia relative higher AST than ALT?

-Zone 3 of the hepatic acinus has a higher concentration of AST

-Damage to zone 3 (ischemia, toxic) = restult in greater alteration to AST





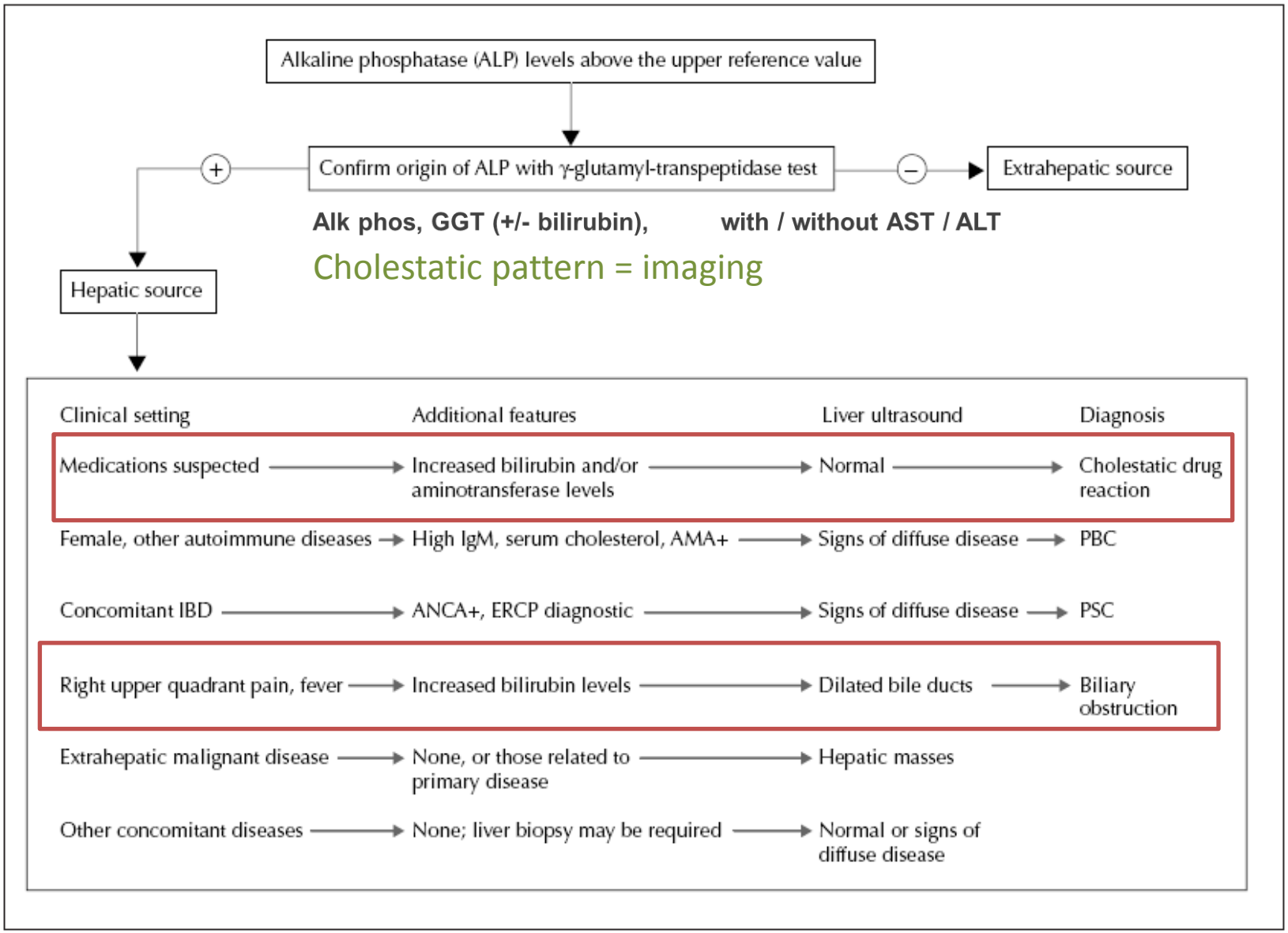


Fig. 5: Suggested diagnostic algorithm for patients presenting with increased alkaline phosphatase levels. AMA = antimitochondria antibodies, PBC = primary biliary cirrhosis, ANCA = antineutrophil cytoplasmic antibodies, ERCP = endoscopic retrograde cholangiopancreatography, PSC = primary sclerosing cholangitis.

DILI = drug induced liver injury
= *alle patronen leverenzymstoornissen*

Per medicament:
-overview
-background
-hepatotoxicity (patroon en ernst)
-links
-references

Leverenzymstoornissen

Parameters leverschade

1) **Patroon:**
   - -Parenchymateus (ASAT, ALAT)
   - -Cholestatisch (Bilirubine, AF, GGT)
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Leversynthese parameters

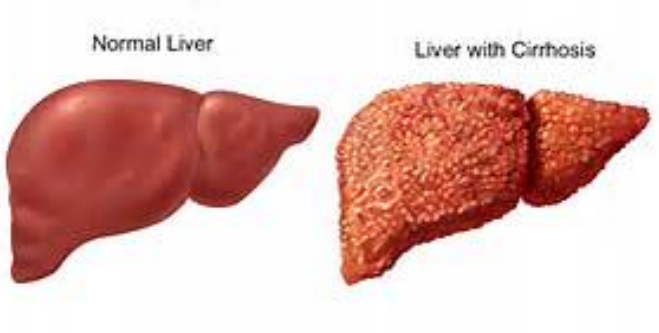
Prothrombine tijd: geeft veranderingen in dagen (alternatief: antitrombine III, factor V)

Albumine: veranderingen over weken / tot maanden. DD: malnutritie, verlies

Bilirubine: verminderde conjugatie en excretie. Snelle veranderingen zichtbaar

Hepatische encefalopathie = ernst correleert niet met ammoniak, doch ondersteunt verdenking

Verminderde functie



1) Ernstige hepatitis, zonder pre-existente leverziekte
   - -Tgv massale hepatocyten-necrose
   - -Transaminasen > 10-50 ULN, icterus
   - - DD: acute hepatitis A,B, AIH, M Wilson (alk phos N), toxines

2) Acute op chronische leverziekte
   - -Tekenen van pre-existente leverziekte
   - -Lab: lagere transaminases maar ernstig effect

3) Gedecompenseerde levercirrose met leverfalen

Casus: Leverproefstoornissen : gering gestegen transaminases, GGT en geringe hyperbilirubinemie

Speurwerk: anamnese / gegevens / LO

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  - Cardiaal lijden / myocardinfarct
- Toxiciteit:
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Aspect leverenzymstoornissen

-Pre-existente leverproefstoornissen
-Trombocytopenie

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Beeldvorming

Casus: Leverproefstoornissen

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  - **Alcohol = ja (> 2 E /dag)**
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- **Comorbiditeit = o.a. DM, cardiovasculaire ziekten**
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Aspect leverenzymstoornissen

-Pre-existente leverproefstoornissen = ja (milde transen, ASAT > ALAT, GGT)

-Trombocytopenie = ja

-Aanwijzingen leversynthese / excretie dysfunctie?

-Aspect leverenzymstoornissen

Beeldvorming:

-Slanke CBD, geen uitgezette galwegen

- aspect lever grofkorrelig, aanwijzingen portale hypertensie = splenomegalie, spoor vrij vocht

-

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-Trombocytopenie = ja

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60-jarige man: acute cholecystitis

Kenmerken chronische leverziekte

* Mogelijk levercirrose met portale hypertensie

    Spoortje vocht: gedecompenseerd DD cholecystitis

* Etiologie : (N) ASH

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* Etiologie : (N) ASH

OPTIES

1) Leversynthese en excretie functietesten zijn goed = mag PCM, morfine hebben zoals in niet – cirrotische pts, liever geen NSAID's

2) Leversynthese en excretie functietesten zijn goed = mag PCM, morfine hebben zoals in niet – cirrotische pts, en ook NSAID's want goede nierfunctie

3) Gezien levercirrose, liever geen NSAID's en (therapeutische doseringen) paracetamol. Mag wel morfine varianten hebben.

4) Ik weet het niet zeker, ik ga het opzoeken

Chronische leverziekte

Chronische leverziekte / milde hepatitis – zonder cirrose:

- Geen leverdysfunctie
- Pijnstilling = in algemene populatie

Veranderingen in drug metabolisme: in patienten met

- Ernstige acute hepatitis
- Levercirrose patient → ernst van verstoringen in drug metabolisme neemt toe met ernst van de leverziekte

Drug metabolic disturbances

Advanced liver disease and cirrhosis

- *Reduced efficacy of drug removal and transport to end-organ by*
  - Reduced hepatic flow
  - Reduced hepatic enzyme capacity
  - Reduced plasma protein binding

-Changes in pharmacokinetic behaviour
-Altered accumulation of free drug
-Change in end-organ response

Important pain medication drugs = metabolized in the liver

- NSAID's, acetaminophen, COX-2 inhibitors, anticonvulsants, antidepressents, opioids

Problem:

- No major studies

Mayo Clin Proc. • May 2010;85(5):451-458 •

Pain management in cirrhotic patient

Acetaminophen (Paracetamol)

- longer half life in patients with liver cirrhosis = **reduced clearance**

- acetaminophen toxicity = mainly if **glutathione** stores become depleted = not severely effected in liver cirrhosis patients
  - probably earlier depleted in **malnutrition and alcohol**

- Dosage with liver cirrhosis:
  short-term or 1 time dosering: 3-4 gr/d appears safe.
  long-term: 2-3 gr/d

- With alcohol / malnutrition: max 2 gr/d

Mayo Clin Proc. • *May 2010;85(5):451-458* •

Pain management in cirrhotic patient

NSAID's:

- largely metabolized by CYP's and heavily proteind bound =
  in cirrhosis = altered metabolism and bioavailability with increased serum levels

- **main concern:** hepatorenal syndrome in pts with portal hypertension
  - inhibition of prostaglandins
    (cirrhotic pts: need prostaglandins to counteract RAAS)

- other: mucosal lesions stomach / intestines

Dosages:

- Mild liver disease: may tolerate.
- No use in cirrhotic patients

MST

een santeon ziekenhuis

Mayo Clin Proc. • May 2010;85(5):451-458 •

Pain management in cirrhotic patient

Opioids:

-Liver is main site for metabolism

-Result in cirrhosis: decreased drug clearance / increased oral bioavailability = drug accumulation

- **Half life of morphine is approximately double**
- Dosage: lower dosing and / or longer intervals between doses
- Probably better tolerated: fentanyl and hydromorphone

OTHER drugs:

- Gabapentine = preferred anticonvulsants (not metabolized by the liver or bound to plasma proteins)
- Tricylische antidepressives (TCA)
  - Rely on first pass effect liver (also other drugs: midazolam)
  - If liver disease / cirrhosis : TCA starting at low doses, may lead to more sedative side effects.
  - General: nortriptuline and desipramine are less potent and appear to be less sedating

MST

een santeon ziekenhuis

Mayo Clin Proc. • *May 2010;85(5):451-458* •

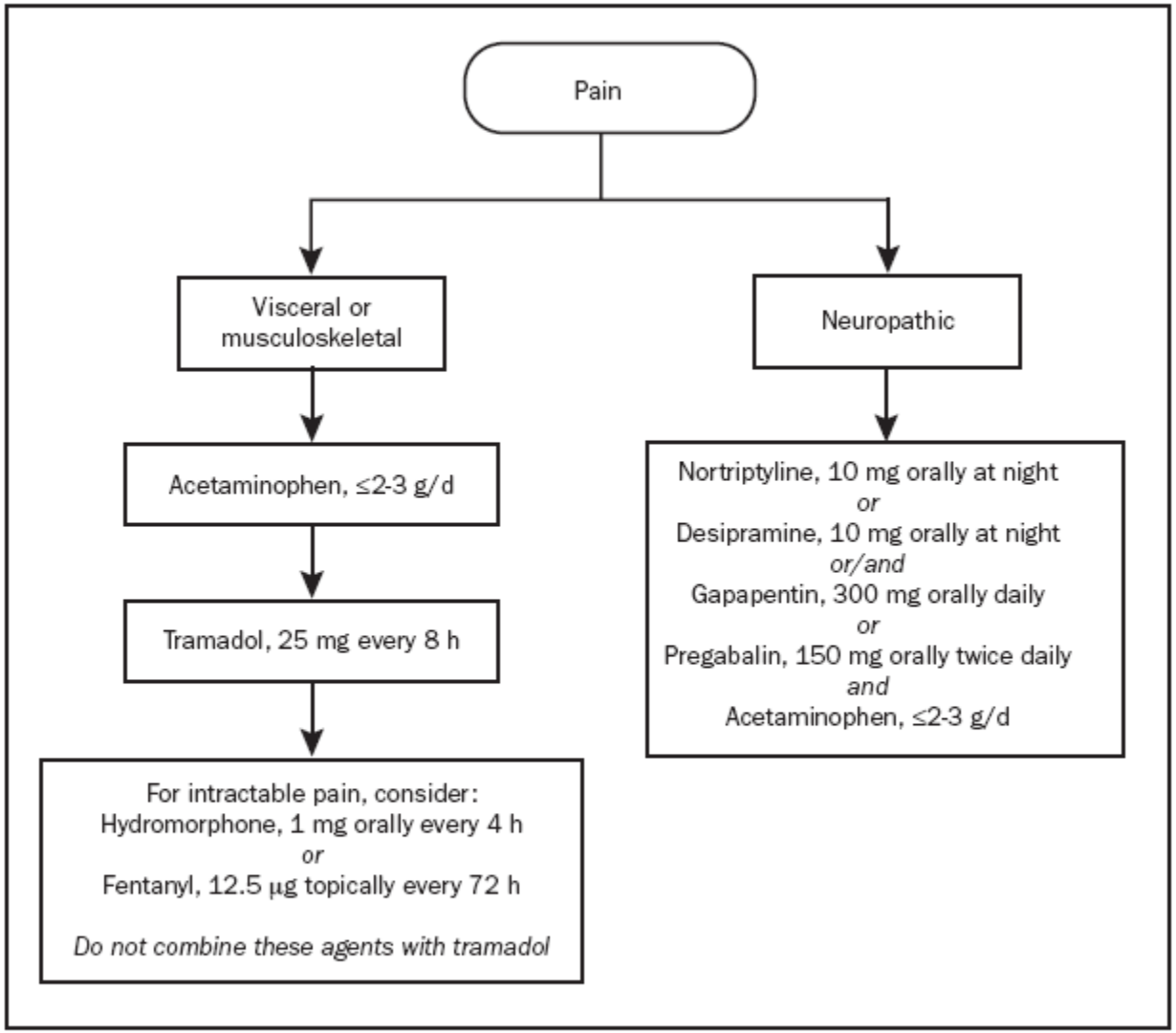
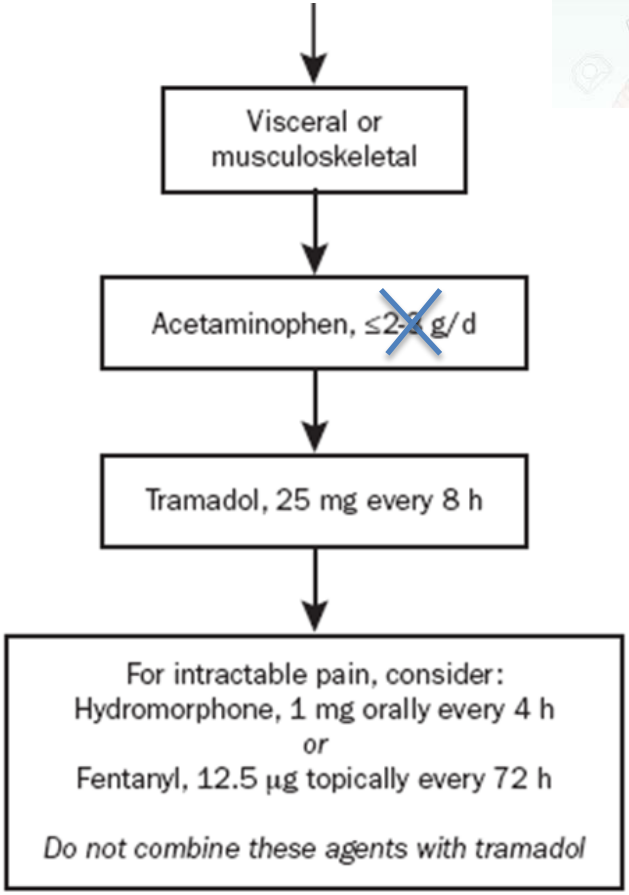


FIGURE 2. A pharmacological approach to analgesia in patients with cirrhosis who have no renal failure, active alcoholism, or active substance abuse. Starting doses are used unless otherwise indicated. Doses should be carefully titrated as tolerated. Minimize total acetaminophen to less than or equal to 2 to 3 g/d. Avoid polypharmacy and monitor for adverse drug events.

60-jarige man: acute cholecystitis

Kenmerken chronische leverziekte

* Levercirrose Child A met portale hypertensie.
* Etiologie : (N) ASH



60-jarige man: acute cholecystitis

Kenmerken chronische leverziekte

* Levercirrose Child A met portale hypertensie.
* Etiologie : (N) ASH

Benzodiazepines = effects GABA

GABA – in non-cirrhotics

GABA:

gamma –aminobutyric acid is an amino acid that acts as the principal calming neurotransmitter in the human central nervous system.

→ Inhibits nerve transmission = calming nervous activity

→ Produced in brain from glutamate acid

→ **Benzodiazepines**: imitate GABA effects : initially it increases the effectivenes of GABA

→ (caffeine does the opposite = stimulant)

GABA – in cirrhotics

Liver cirrhosis:

-**ammonia** can augment the activity of GABA-ergic receptors / neurons

-increase circulating levels of **endogenous benzodiazepines**

-"**super sensitive GABA receptor complex**" = more effect benzodiazepnies

Benzodiazepines = effects GABA

GABA – in non-cirrhotics

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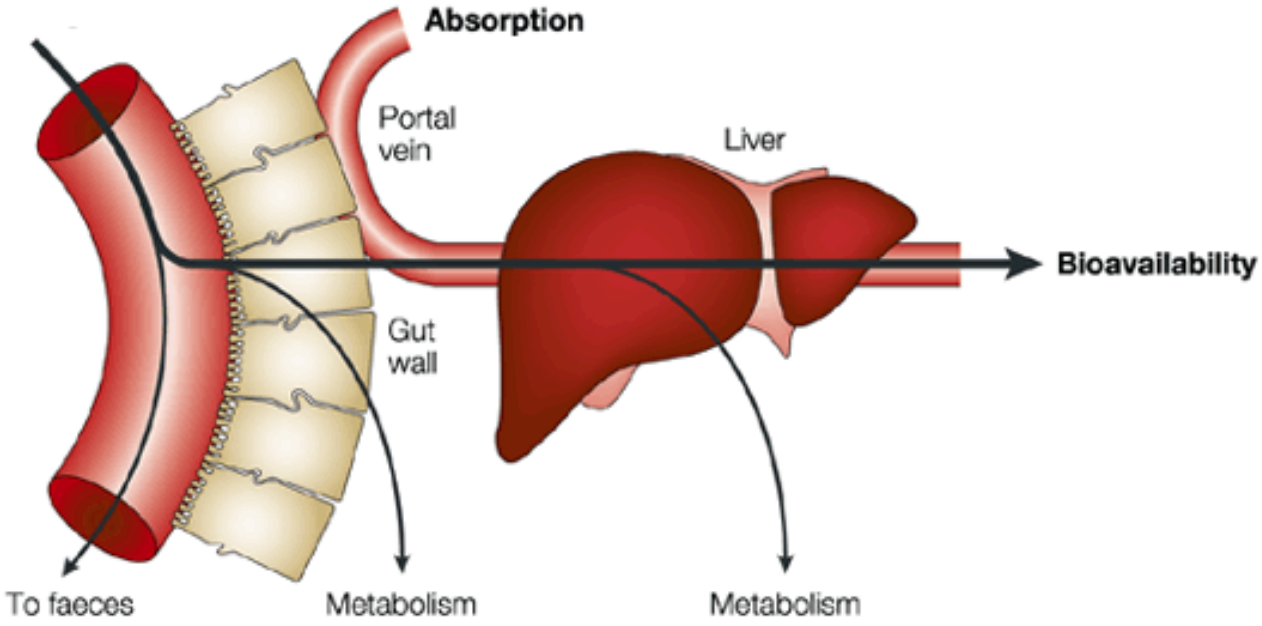

Nature Reviews | Drug Discovery

Midazolam: depends on **first pass effect**

-first pass effect is less in liver cirrhosis = more bioavailability

60-jarige man: acute cholecystitis

Kenmerken chronische leverziekte

* Levercirrose Child A met portale hypertensie.
* Etiologie : (N) ASH

Sedatieve werking verhoogd = lage dosis starten (Proefdosis), per dag evalueren

Cave: ontwikkeling sepsis, toename leverenzymstoornissen = kans op verminderde functie van de lever

60-jarige man: acute cholecystitis

Kenmerken chronische leverziekte

* Mogelijk levercirrose met portale hypertensie.
* Etiologie : (N) ASH

1) Ja kan het beste op korte termijn. Niet veel verhoogd risico: vrijwel geen ascites, geen evidente collateralen gezien

2) Nee, liever "afkoelen" en later op electief moment

3) Overig…..

Aliment Pharmacol Ther 2013; 37: 1132-1156

Table 4. Important parameters to look for and manage appropriately in patients with cirrhosis planned for non-hepatic surgery.

System	Pathology	Assessment	Management
Abdomen	Ascites Increased risk of abdominal wound dehiscence, abdominal wall herniation, respiratory compromise, spontaneous bacterial peritonitis (SBP)	Check response to diuretics, pulmonary function tests, diagnostic ascitic tap	~ Low sodium diet and diuretics with careful monitoring of creatinine and electrolyte levels ~ Large volume paracenthesis for uncontrolled ascites with albumin ~ Antibiotics for SBP
Renal	Renal insufficiency/hepatorenal syndrome (HRS) due to drugs, infections, gastrointestinal bleed	Renal function tests, creatinine clearance, DTPA scan	~ Avoid nephrotoxic drugs, contrast agents for diagnostic studies ~ Combination of terlipressin, albumin in HRS ~ Optimal fluid, electrolyte status
Central nervous system	Hepatic encephalopathy (HE)	Clinical assessment, arterial ammonia levels	~ Use of lactulose, metrogyl, branched chain amino acids ~ Treat infections, avoid diuretics, constipation, CNS depressants, azotemia
Pulmonary	Hydrothorax, hepatopulmonary syndrome (HPS), portopulmonary hypertension (PPH)	~ Chest imaging ~ Bubble ECHO/MAA scan for HPS	~ Optimize pulmonary functions ~ Intravenous epoprostenol, sildenafil has also been tried perioperatively
Cardiac	Cardiomyopathy	~ Dobutamine stress ECHO ~ ACC and AHA guidelines for non-cardiac surgery	Beta blockers in perioperative period
Homeostasis	Electrolyte disorders (esp. hyponatremia)	Regular electrolyte profile and arterial blood gases	Slow correction of serum sodium with fluid restriction, discontinuation of diuretics
Nutrition	Malnutrition, hypoalbuminemia, muscle wasting, increased need for postoperative ventilation	Methodical nutritional assessment	~ Preoperative nutritional build-up (high carbohydrate/lipid content, low in amino acid) ~ Vitamin B1 in alcoholics
Other systems	Anaemia and coagulopathy	Intraoperative thrombo-elastogram	Appropriate blood products perioperatively to maintain desired INR (<1.5), haemoglobin (>9 g%), platelet (>50,000/mm^3) levels
	Glucose intolerance	Laboratory testing	Insulin infusion
	Gastroesophageal varices	Endoscopy, portal pressure measurements	Beta blockers, variceal banding
	Concurrent infections	Screening	Antibiotic prophylaxis
	Autoimmune hepatitis patients developing stress-induced insufficiency	Serum cortisol levels	Stress-dose steroids preoperatively

Operatie risico in levercirrose patienten?

Patient met cirrose + cholecystitis = cholecystectomie risico's?

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Contents lists available at SciVerse ScienceDirect

Best Practice & Research Clinical Gastroenterology

5

Morbidity and mortality related to non-hepatic surgery in patients with liver cirrhosis; A systematic review

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64 articles were selected from 5247
-lack of evidence varied

*non-hepatic surgery

Non-hepatic surgery in cirrhosis

Surgical risk determined by

Degree of decompensation (MELD / CTP) ?	Is surgery an performed as an emergency procedure?	What is the nature / type of surgery?
Child A cirrhosis without portal hypertension = majority of non-hepatic surgery is safe	Doubles morbidity and mortality (OR 2.4)	Lowest increased risk mortality: cholecystectomy, umbilical and inguinal herna correction
Portal hypertension itself = independent risk factor	If emergent + portal hypertension = OR 5.9	Highest increased risk : pancreatic surgery, cardiovascular and trauma surgery
MELD < 8 = mortality 5.7% MELD > 20 = > 50%		
Preventive effect of portal decompression (preoperative TIPS?)		

Non-hepatic surgery in cirrhosis

Effect portal hypertension

	Liver cirrhosis without portal hypertension (mortality risk vs non-cirrhotic pts)	Liver cirrhosis with portal hypertension
-Cholecystectomy	: 3.4 fold increase mortality	12.3
-Colectomy	: 3.7	14.3
-CABG	: 8.0	22.7

Cholecystectomy:

-best outcome if laparoscopic

-however: increased risk of conversions during operation

Colectomy:

- In hospital mortality after elective surgery = 14% in cirrhotic / 29% if cirrhosis + portal hypertension (compared to 5% non-cirrhotic pts)
- However: if emergent = 20.9% for cirrhotic vs 35.8% cirrhotic + portal HT

Non-hepatic surgery in cirrhosis

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Child A cirrhosis without portal hypertension = majority of non-hepatic surgery is safe	Doubles morbidity and mortality (OR 2.4)	**Lowest increased risk mortality: cholecystectomy** (3.4-fold vs **12.3-**fold increase), umbilical and inguinal herna correction
Portal hypertension itself = independent risk factor	**If emergent + portal hypertension = OR 5.9**	Highest increased risk : pancreatic surgery, cardiovascular and trauma surgery
MELD < 8 = mortality 5.7% MELD > 20 = > 50%		
Future: Preventive effect of portal decompression (preoperative TIPS?)		

Onze casus : Cholecystectomie?

Richtijn 2016 Ned ver heelkunde

Conclusie

Niveau 1a	Directe cholecystectomie leidt tot een sneller herstel met een kortere opnameduur. Er is geen verschil in complicaties en conversies. In 23 tot 26% van de initieel conservatief behandelde patiënten dient alsnog een eerder dan geplande cholecystectomie te worden verricht (Gurusamy 2013, Gutt 2013).

Overige overwegingen

Een andere mogelijkheid is een volledige conservatieve behandeling, die met name overwogen kan worden bij patiënten met ernstige comorbiditeit (Vetrhus 2003, Schmidt 2011).

In de meeste onderzoeken wordt voor directe cholecystectomie een termijn van 1 week na het begin van de klachten gehanteerd. Het zo vroeg mogelijk opereren bij een acute cholecystitis leidt tot minder morbiditeit (de Mestral 2013, Gutt 2013, Gelbard 2014).

Opties

1) Cholecystectomie nu doen = nl. minder kans op complicaties van een gecompliceerde cholecystectomie later

2) Antibiotica (met / zonder galblaasdrainage) en wellicht dat cholecystectomie niet meer nodig is

3) Antibiotica (met / zonder galblaasdrainage) en tzt cholecystectomie

Cholecystectomie : nu of later?

Voordeel uitstellen :

- Electief moment
- Stoppen alcohol = mogelijk minder decompensatie klachten / minder portale hypertensie

Nadeel :

- Risico op geen verbetering met conservatieve benadering. Met toename verklevingen in galblaasbed
- Percutane drainage : complicaties bij portale hypertensie?

*Geen studies die dit onderzoeken

*Per patient afweging in multidisciplinair team en patient = overleg dan wel overname expertise kliniek

In summary ……

Liver enzyme evaluation = take your time ..

To take into account:

1) Anamnesis:

1) Medication use (when, what)
2) Intoxications
3) Ethnicity
4) Other symptoms, why admission

2) Physical examination

3) Other comorbidities

- DM
- Cardiovascular disease
- Cancer
- Other autoimmune diseases

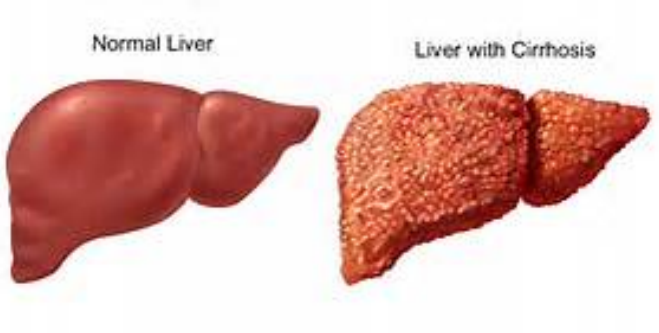
4) Liver enzymes:

- distribution / pattern
- signs of liver failure
- signs of pre-existing liver disease

Differential diagnosis

-Further evaluation:
-Radiology?
-Serology?
-Other?

Hepatology in consultation



Liver enzyme disturbances

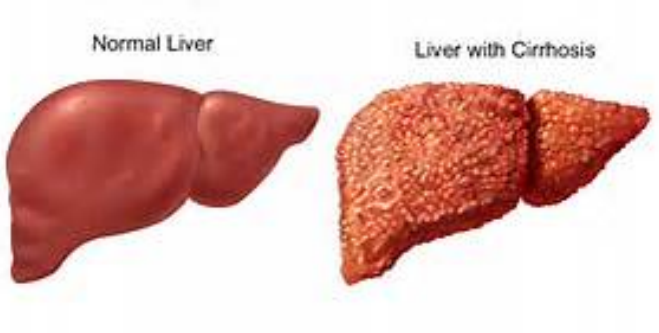
- 1**) Pattern:**
  - Parenchymal
  - Cholestatic (intra vs extrahepatic disease)
  - Mixed

2) **Typical features:** GGT, AST/ALT ratio

3**) Severity:**
- Transaminases (AST/ALT, severity)
- Acute on ***chronic liver disease***
- Liver function tests (bili, Pt, alb) and other additional signs (HRS, HE)

Chronic liver disease patient

Hepatology in consultation



Chronic liver disease patient

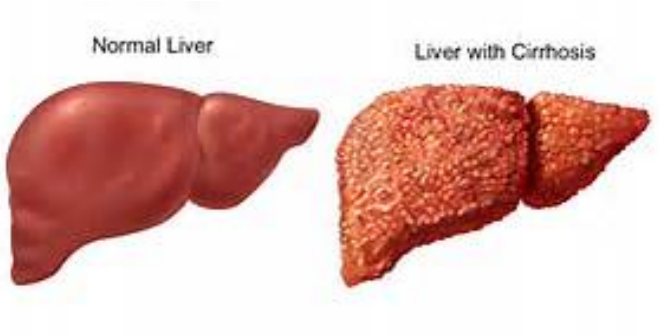
Pain medication in cirrhotic patients (with or without decompensation):
-Paracetamol: lower dosing if longer used = **max 2-3 gram per day**
(esp with malnutrition / alcohol = **max 2 gram per day**)

-**No NSAID's**

-Morphine in reduced dosing.
- Fentanyl

Benzodiazepines:
- In multiple ways = more effect and bio-availability
- Cautious use, per day evaluation

Hepatology in consultation



Chronic liver disease patient

Multidisciplinary setting / expert center

Operation risk:

- Low in child A **without** portal hypertension

Increases with :

- Portal hypertension
- Liver function failure = synthesis + excretion dysfunction

* *CTP and MELD score = predictive for morbidity and mortality*

Depends also on

- Emergency or not
- Type of surgery

HEALTH

The Liver: A 'Blob' That Runs the Body

The underrated, unloved liver performs more than 300 vital functions. No wonder the ancients believed it to be the home of the human soul.

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Basics

By NATALIE ANGIER JUNE 12, 2017

Cuyco

The liver also keeps track of time. In a recent issue of the journal Cell, Ulrich Schibler of the University of Geneva and his colleagues described their studies of the oscillating liver, and how it swells and shrinks each day, depending on an animal's normal circadian rhythms and feeding schedule.

To the Mesopotamians, the liver was the body's premier organ, the seat of the human soul and emotions. The ancient Greeks linked the liver to pleasure: The words hepatic and hedonic are thought to share the same root.