Clinical Masterclass

5th November 2025

Liver disease

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Consultant Pharmacist in Hepatology

PrescQIPP Clinical Masterclass

Housekeeping

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- If you have a microphone, please use the hands up function if you would like to speak.
- If you don’t have a microphone then feel free to type your comments into the chat.
- We will record this session and it will be available on our website, along with any supporting documents.

PrescQIPP Clinical Masterclass

Clinical Masterclass – Liver disease
Feedback - 5th November 2025

PrescQIPP
Clinical Masterclass

MASTERCLASS: LIVER DISEASE

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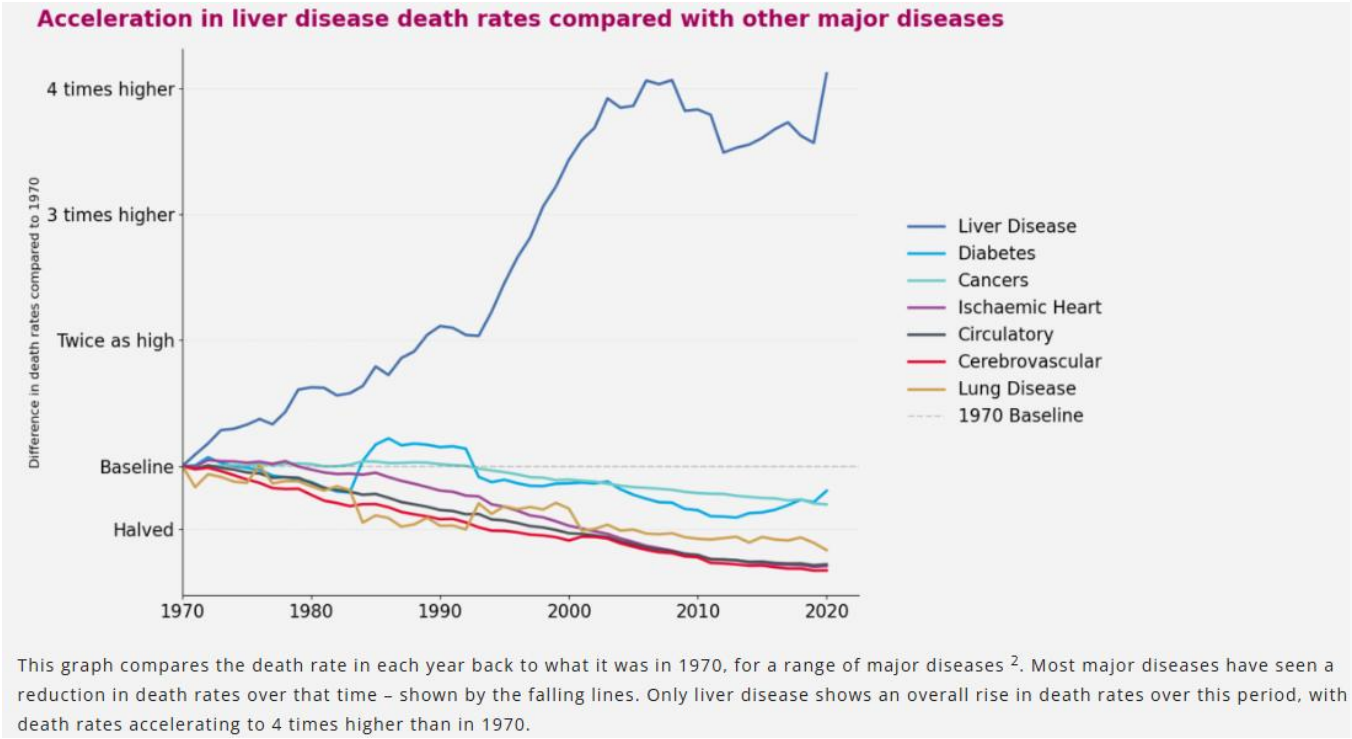
OVERVIEW

- Overview of liver disease and causes
- Function of the liver
- Classification of liver dysfunction
- Diagnosing and liver function tests (LFTs)
- Signs and symptoms
- Pharmacokinetics
- Symptoms management
- Drug-induced injury
- Deprescribing and general medicines advice

CHRONIC LIVER DISEASE AND CIRRHOSIS



This graph compares the death rate in each year back to what it was in 1970, for a range of major diseases [2]. Most major diseases have seen a reduction in death rates over that time – shown by the falling lines. Only liver disease shows an overall rise in death rates over this period, with death rates accelerating to 4 times higher than in 1970.

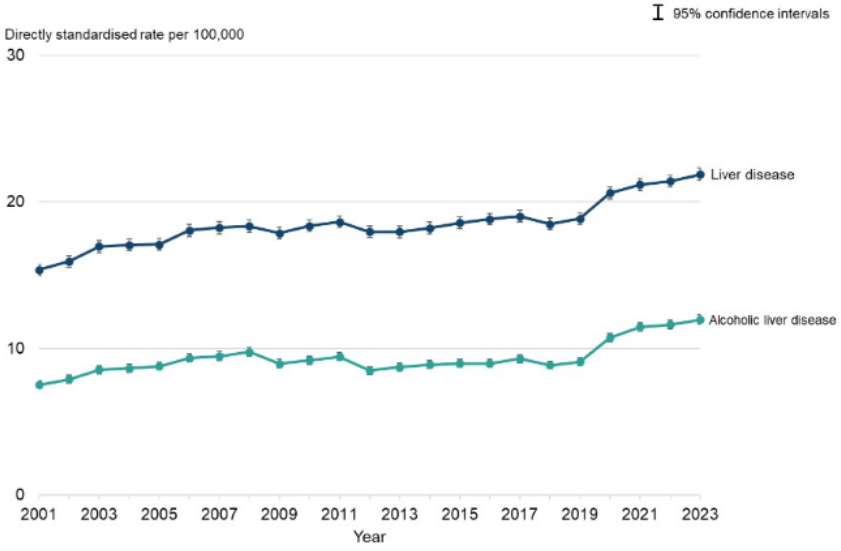
Liver disease is a greater cause of working life years lost (15-64yr) than ischaemic heart disease, colon cancer, breast cancer or strokes, but treatable if identified early.

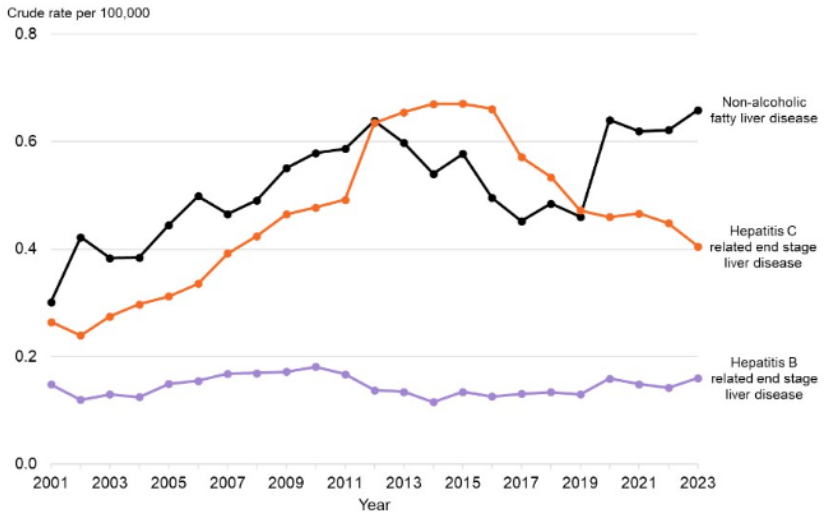
76 % of alcohol related deaths were attributed to alcohol related liver disease in 2022

Deaths from liver disease have increased by 400% since 1970 Source: OHID, local analysis 2022.

For further information on the recommendations of the Commission or to read the full report, visit http://www.thelancet.com/commissions/crisis-of-liver-disease-in-the-UK

LIVER DISEASE RISK FACTORS

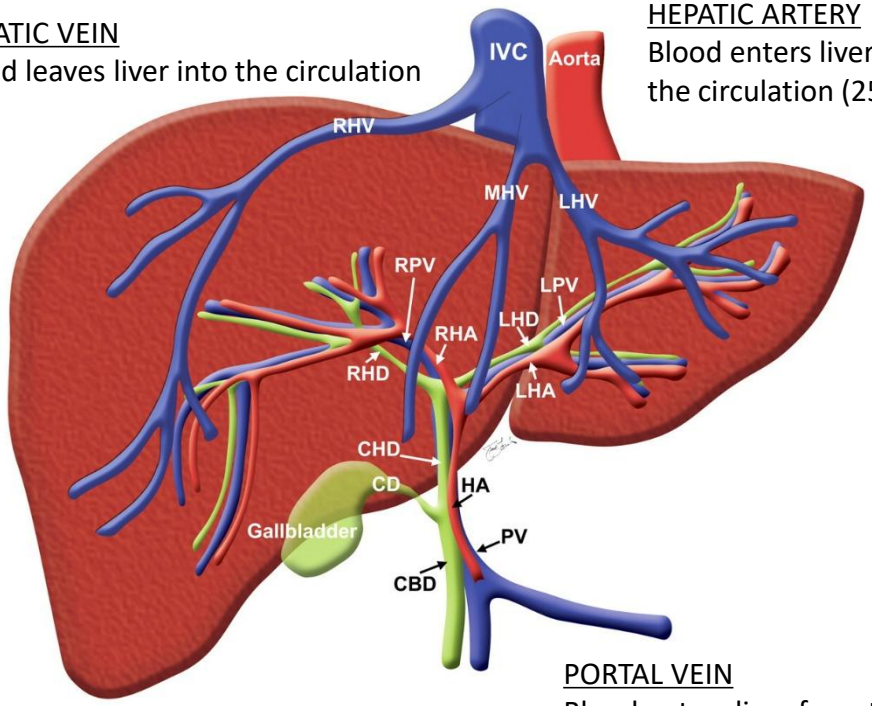




LIVER

HEPATIC VEIN
Blood leaves liver into the circulation

HEPATIC ARTERY
Blood enters liver from the circulation (25%)



PORTAL VEIN
Blood enters liver from the gut (75%)

COMMON BILE DUCT
Bile leaves liver into the gut

WHAT DOES THE LIVER DO?

Metabolism

Carbohydrate

Protein

Fat

Steroid hormones

Insulin

Aldosterone

Bilirubin

Drugs

Synthesis

Proteins (e.g. albumin)

Clotting factors

Fibrinogen

Cholesterol

25-OH of vitamin D

Gluconeogenesis
(glucose from fat and protein)

Immunological

Kupffer cells

Storage

Fat soluble vitamin

A, D, E,K, B_{12} and

Folic acid

Homeostasis

Glucose

Production of Bile

Secretion bile salts

Enterohepatic circulation

Clearance

Bilirubin

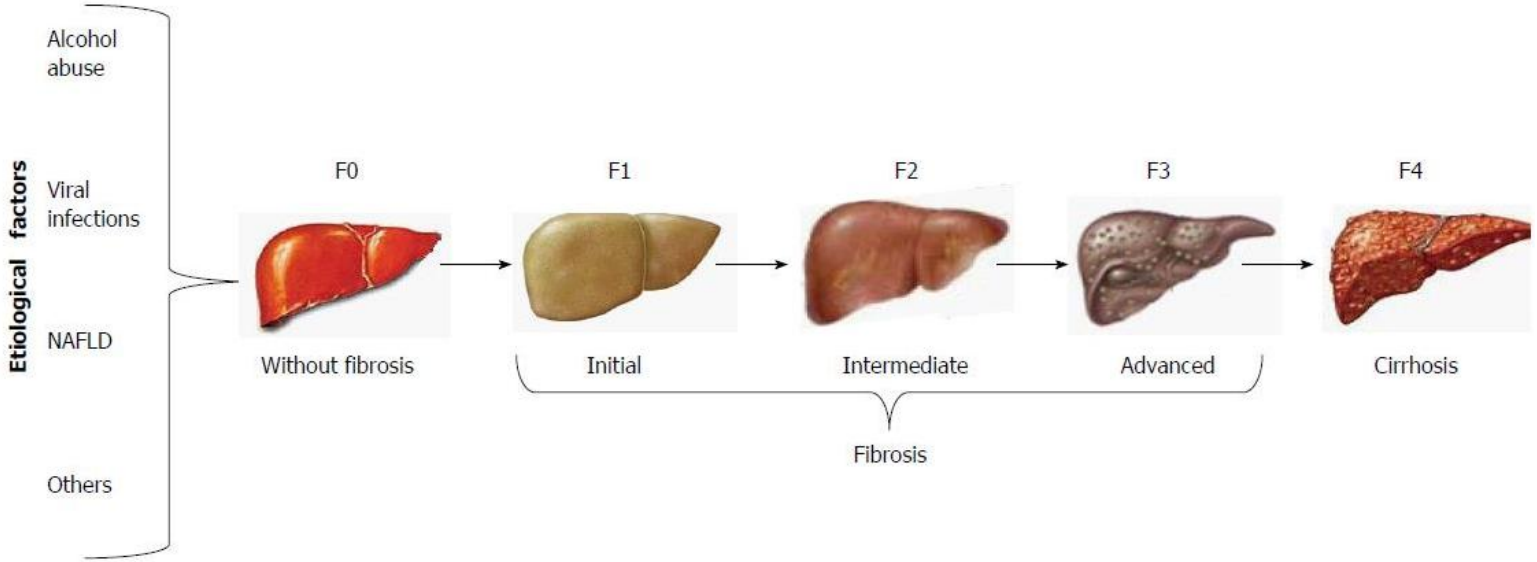
Drugs

Toxins

WHAT CAUSES ACUTE DAMAGE TO THE LIVER?

- Acute hepatitis
  - Toxic
    - Paracetamol, Amanita phylloides, iron overdose
  - Viral
    - Hepatitis A, Hepatitis B, Hepatitis E, EBV, CMV, HSV
  - Poor diet am dlifestyle → MASLD
  - Autoimmune
  - Ischaemic
    - Right heart failure
    - Budd chiari
    - Cocaine, amphetamines
    - Portal vein thrombosis Other drugs (DILI)
  - …Pregnancy, Wilsons, malignancy

CLASSIFICATION OF LIVER DISEASE - CHRONIC



COLLATERAL

- History
  - Alcohol
  - Smoking
  - Drug history
  - Family history
  - Risk factors for viral hepatitis – prisoners, PWID, high prevalence countries, sex worker, MSM
  - Medication history
  - RUQ – gallstones vs malignancy

LIVER FUNCTION TESTS

- Do not measure actual function of the liver
- Hepatocellular damage
  - ALT/AST – hepatitis (usually viral/autoimmune/DILI)
- Cholestatic – markers of bile duct injury
  - ALP/GGT – biliary issue
- Liver synthetic function
  - Bilirubin ↑
  - Albumin ↓
  - INR ↑
- Low platelets

Trends not isolation

Abnormal liver blood tests do not always indicate the extent of liver damage

Results can be normal even in advanced liver disease

Some liver blood tests can be abnormal in the absence of liver disease or affected by conditions not related to the liver

Some changes to liver blood tests are transient

Assessing liver function and interpreting liver blood tests – SPS - Specialist Pharmacy Service – The first stop for professional medicines advice

IMAGING AND OTHER INVESTIGATIONS

- LUSS
  - Normal vs nodular
  - Enlarged spleen
- CT, MRI
- ERCP, MRCP
- Biopsies

- Fibroscan
  - Measures the stiffness of the liver + fatty (CAP > 250dB/m)
  - Cut offs vary, HBV eg:
    - If >7kPa – F2
    - >11kPa – F4

FIB -4

AST, ALT, PLT

ELF (Enhanced Liver Fibrosis)

MASLD

LIVER SCREEN

- Viral Hepatitis
- Metabolic
  - Iron
  - Copper
  - Fatty liver
- Immune
  - ANA
  - AMA
  - Immunoglobulins

SIGNS AND SYMPTOMS

- Jaundice
- Ascites
- Pruritus
- Unexplained bruising and bleeding
- Varices
- Encephalopathy
- Abdominal pain
- Pale stools and dark urine
- Gynaecomastia
- Fatty stools
- Spider naevi
- Finger clubbing

MODEL FOR END-STAGE LIVER DISEASE (MELD) AND CHILD-PUGH SCORING

- Stratification for patients who are suffering with hepatic dysfunction.
- The Child-Pugh score is only validated for patients with chronic liver disease, who have cirrhosis.
- The MELD score was initially developed to predict survival in cirrhosis patients, but has now been validated for alcoholic hepatitis, acute liver failure, and acute hepatitis as well.

In simple terms both scores offer a measure of how 'sick' the patient is.

GENERAL PRESCRIBING POINTS

- Unlike renal dysfunction little information available to help guide drug dosing
- Child-Pugh Scoring System
  – often used to make recommendations in drug SPCs
  BUT
  not developed to predict drug handling but disease severity/outcome
- Are we overly cautious?
  - *Capacity of the liver is so great that liver disease must usually be extensive before effects on drug handling become important*
- BNF/SPC recommendations
  – vast majority of drugs are cautioned or contraindicated

Start low and go slow

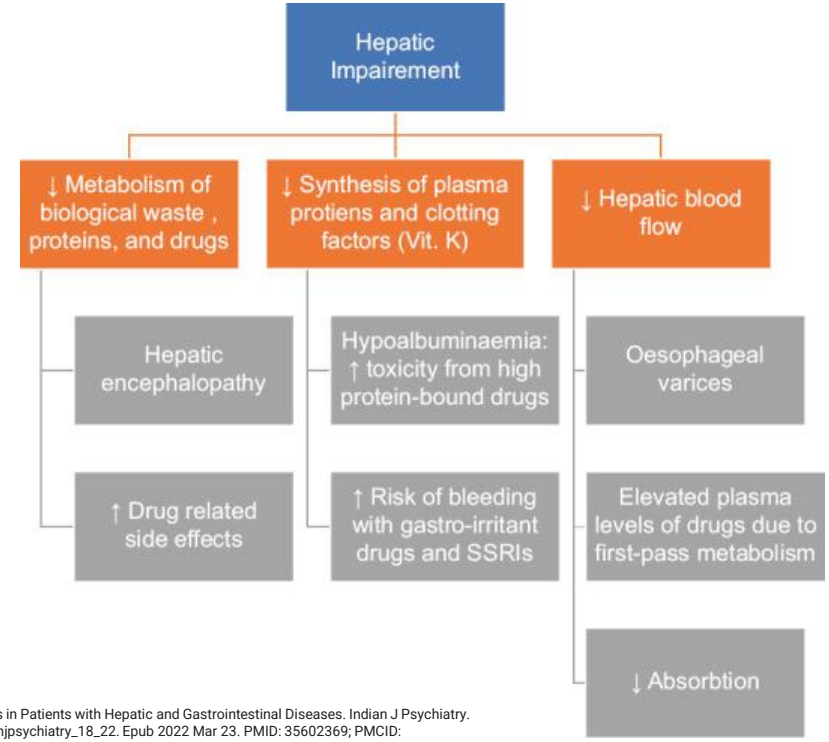
DRUG HANDLING IN LIVER DISEASE

- Patient factors
  - Diagnosis/underlying cause
  - Signs and symptoms of liver disease
- Drug factors
  - Pharmacokinetics
  - Pharmacodynamics
  - Side effect profile
  - Rout of administration

PHARMACOKINETICS

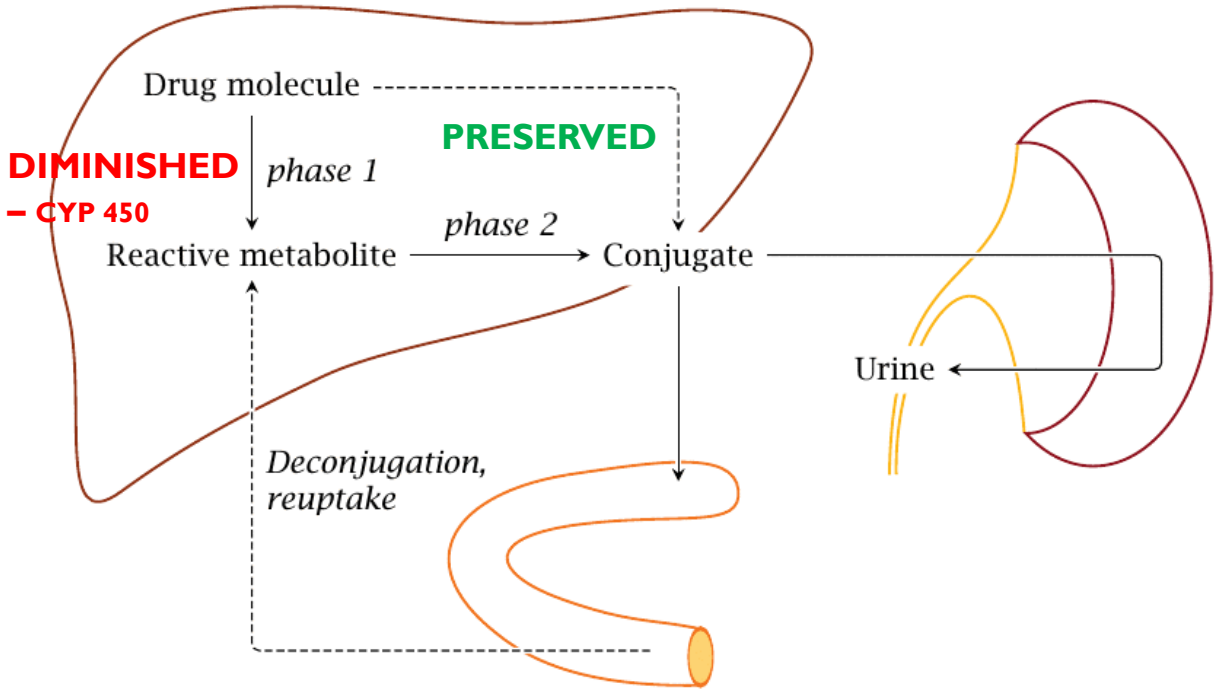
- Factors which affect hepatic metabolism are liver blood flow and functioning cell mass – difficult to assess in practice
- Drug metabolism very complex and not fully understood.
- Liver will carry out drug metabolism even in cirrhotic patients
- Difficult to predict PK changes in individual patients
- Consider route elimination – renal, biliary etc

PHARMACOKINETIC CHANGES IN HEPATIC DISEASE



Menon V, Ransing R, Praharaj SK. Management of Psychiatric Disorders in Patients with Hepatic and Gastrointestinal Diseases. Indian J Psychiatry. 2022 Mar;64(Suppl 2):S379-S393. doi: 10.4103/indianjpsychiatry.indianjpsychiatry_18_22. Epub 2022 Mar 23. PMID: 35602369; PMCID: PMC9122174.

METABOLISM


Drug molecule
PRESERVED
DIMINISHED
– CYP 450
phase 1
phase 2
Reactive metabolite
Conjugate
Urine
Deconjugation,
reuptake

METABOLISM

Hepatic cell mass

= functional capacity of the liver

<u>Liver damage :</u>

- ↓ no. of functioning liver cells
- +/- portal shunts
- ↓ metabolism, ↑ bioavailability
- prolong half-life of drugs
- ↑ toxicity with repeated dosing

BIOAVAILABILITY

High extraction ratio drugs

= drugs that are highly first-pass metabolised

- Hepatic clearance depends on blood flow to liver
- Hepatic blood flow ↓, bioavailability ↑

Low extraction ratio drugs

- Hepatic blood flow ↓, bioavailability not affected except for drugs that are highly protein bound

Remember:

- Drugs undergo liver metabolism → formation of water-soluble inactive compounds for elimination
- For some drugs pharmacologically active compounds may be formed in process

ELIMINATION

Cholestasis

= bile fails to flow from hepatocytes to duodenum and is diverted to blood

- Bile salts competes for protein binding sites
- ↓ protein binding – increased ‘free drug’
- ↓ absorption of lipid soluble drugs
- Drugs eliminated by biliary route may accumulate

FLUID STATUS

- Water soluble drugs – generally safe to use

HOWEVER:

- Changes in fluid status that can occur in liver disease patients.
- Abnormal renal haemodynamics
- Sudden change in fluid status that can occur due to some therapeutic procedures
  - paracentesis
  - extreme diuresis
  - diarrhoea induced in the treatment of liver encephalopathy
- If the total volume of body fluid is suddenly reduced, the regular therapeutic drug level can become critically toxic

Therefore, when using these types of drugs (such as lithium) in patients with cirrhosis, a strict coordination is mandatory between the different medical specialists that assist the patient

PHARMACODYNAMIC PROPERTIES

- Patients may be at risk of
  - Increased toxicity
  - Exaggerated response
  - Reduced response

  Caution with drugs which:

  - Cause sedation (cerebral depressant activity) - can cause/worsen encephalopathy
  - Cause GI ulceration - because of risk of bleeding in coagulopathy
  - Cause constipation - can cause/worsen encephalopathy
  - Interfere or have an adverse effects on clotting
  - Cause fluid retention and electrolyte imbalance (endocrine/metabolic effects)
  - Have narrow therapeutic index/ hepatotoxic /nephrotoxic

ROUTES OF ADMINISTRATION

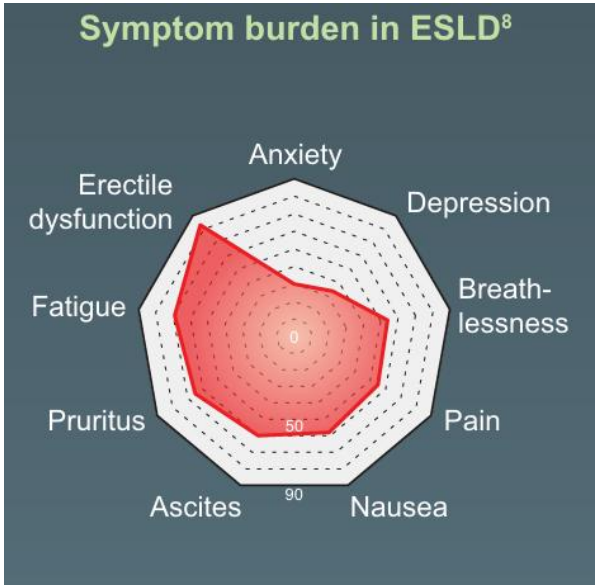
- Oral – generally preferred
- Avoid modified release and long-acting preparations
- Avoid IM if coagulopathy
- Topical preparations – consider transdermal absorption
- Topical preparations – may cause irritation
- PR – consider presence varices/bleeding

SUMMARY

- Is drug highly metabolised in liver?
- What is the route of elimination?
- What is it side-effect profile?
- Consider hepatic blood flow and synthetic function
- Be aware of potential renal impairment
- Where possible – TDM
- Think about route of administration
- Titrate to patient response

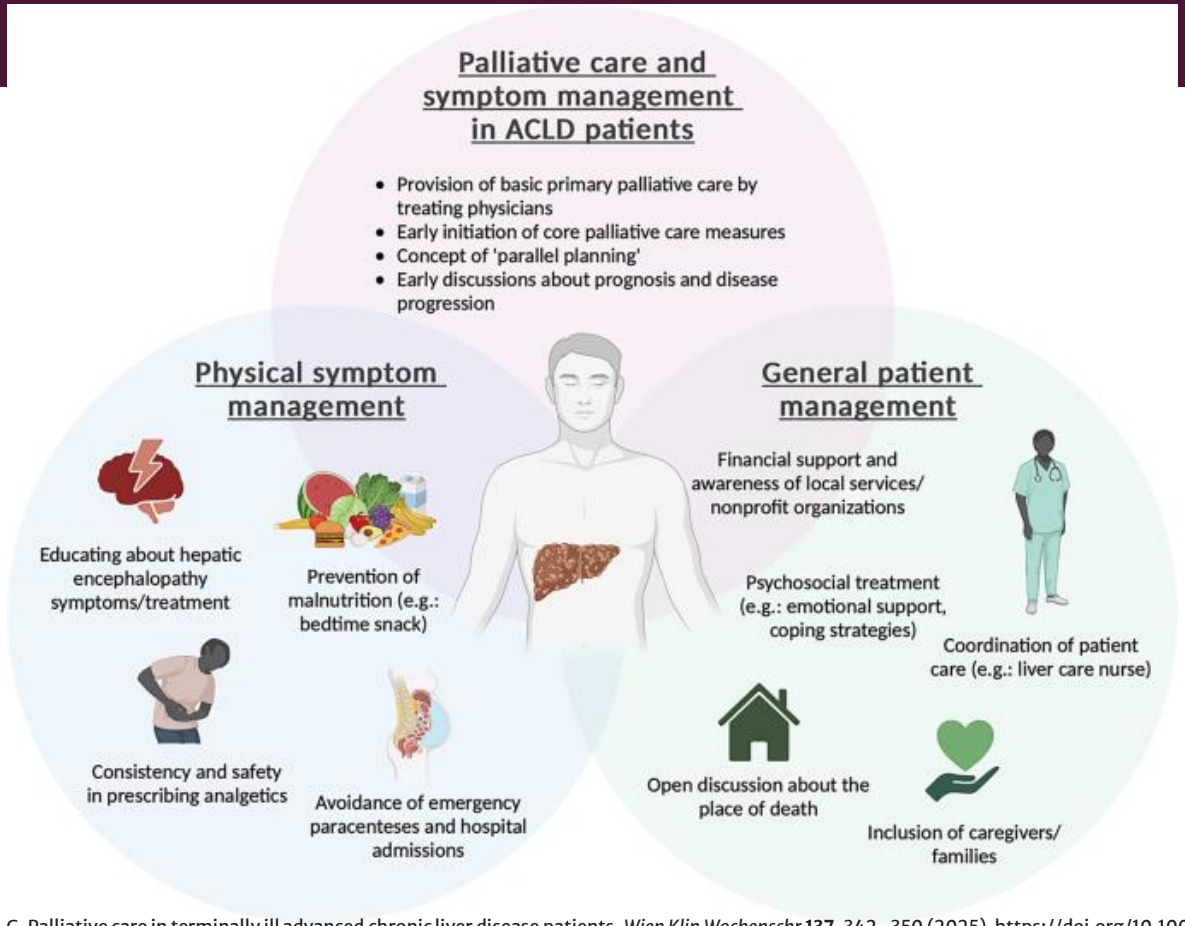
SYMPTOM MANAGEMENT IN END STAGE LIVER DISEASE

- High symptom burden
  - Pain, nausea, vomiting
  - Ascites, hepatic encephalopathy, itch
- Child Pugh B and C
- Co-existing renal impairment



Symptom control in adults with advanced liver disease - BASL Final.pdf

Improving end of life care in liver disease, P Berry et all. Journal of Hepatology, 2022 vol.76, 1225-1226



Pomej, K., Masel, E.K. & Kreye, G. Palliative care in terminally ill advanced chronic liver disease patients. *Wien Klin Wochenschr* **137**, 342–350 (2025). https://doi.org/10.1007/s00508-024-02436-z

PAIN

	Weight * ≤ 40kg	Weight * 41kg to 49kg	Weight * > 50kg
Oral dosing (PO)	500mg Four times daily	500mg - 1g Three times daily	500mg - 1g four times daily
Maximum daily dose	2 g	3 g	4 g

*Dry weight should be used
If over 50kg (dry weight), 1g QDS PO is safe for short periods (≤ 7 days)
If needed regularly long-term (> 7 days), reduce dose

Irrespective of weight where the patients eGFR is less than 30ml/min/1.73m2, the interval between dosing must be a minimum of 6 hours

Maximum 3g / 24hrs IV (even short-term)	**Avoid IV preparation whenever possible and always dose reduce when prescribing**

https://www.basl.org.uk/uploads/BHPG/Paracetamol%20Position%20Statement_Mar%202022.pdf?UNLID=

NSAIDs		Avoid (risk of bleeding and renal toxicity)
Tramadol		Avoid (half-life more than doubles and lowers seizure threshold)
Codeine	**15-30mg PO TDS** (short course only)	Avoid if possible – preferably use oral morphine If oral morphine not an option trial with caution as has unpredictable effect Monitor closely for constipation and worsening encephalopathy
Morphine sulphate	**2.5mg 4-6hrly PO PRN**	1st choice oral opioid for pain if eGFR ≥ 30 Use short-acting preparations unless pain and liver function are stable Titrate up dose as required Monitor closely for constipation and worsening encephalopathy
Hydromorphone	**1.3mg 8hrly PO PRN** (~ 10 times as potent as oral morphine)	1st choice oral opioid if eGFR < 30 Note longer than usual dose interval Monitor closely for constipation and worsening encephalopathy
Oxycodone	**1.25mg 6-8hrly PO PRN** (Twice as potent as oral morphine)	Ideally avoid (half-life more than triples) Consider as second line strong opioid if patient cannot tolerate oral morphine, particularly if there is co-existing renal impairment i.e. eGFR 30-60 Monitor closely for constipation and worsening encephalopathy
Buprenorphine transdermal patch	*Dose according to oral opioid requirements*	*Can be used if pain and liver function are stable* *Monitor closely for constipation and worsening encephalopathy* *Only initiate on advice of palliative care and/or specialist pain team*
Gabapentin	**100mg PO BD** and titrate up as normal	Probably safe but can have sedative effect
Pregabalin	**50mg PO BD** and titrate up as normal	Probably safe but can have sedative effect
Amitriptyline		Avoid

Due to the structural liver changes in cirrhosis, patients do not tend to experience liver capsule pain. Hepatocellular carcinoma or liver metastases

- Dexamethasone 4-8mg PO OD with gastric protection (e.g. ranitidine 150mg BD or omeprazole 10-20mg OD) can be used with review after 5 days.

https://www.basl.org.uk/uploads/End%20of%20Life%20SIG/Symptom%20control%20in%20adults%20with%20advanced%20liver%20disease%20-%20BASL%20Final.pdf

NAUSEA AND VOMITING

Drug	Recommended Dose	Notes
Metoclopramide	**5mg PO/IV/SC TDS** Titrate to maximum 10mg TDS	First line option if gastrointestinal (GI) cause, acts as prokinetic May increase fluid retention Consider QT interval prolongation
Domperidone	**5mg PO BD** Titrate to maximum 10mg TDS	Alternative first line option, acts as prokinetic Consider QT interval prolongation
Haloperidol	**0.5-1mg PO BD** Titrate to maximum 5mg / 24hrs in divided doses	First line option if opioid or centrally induced
	0.25-0.5mg SC TDS	
Ondansetron	**4mg PO/IV BD** Maximum dose 8mg/24 hours	Second line option Monitor closely for constipation
Levomepromazine	**3mg PO NOCTE** Titrate to maximum 12.5mg BD	Second line option Causes drowsiness and can lower seizure threshold Use only if sedating effects acceptable Note: unlicensed formulation, tablets are 6mg and can be halved
	2.5mg SC TDS	
Cyclizine	**50mg PO BD**	Third line option Monitor closely for constipation and worsening encephalopathy
	25mg IV/SC BD	

DEPRESSION

Mirtazapine	**Start at 15mg PO ON** and titrate slowly to maximum dose 30mg ON	Avoid if patient has renal impairment May help to stimulate appetite Can have sedating effect
Citalopram	**Start at 10mg PO OD** (morning), titrate slowly to maximum dose 20mg	Half-life nearly doubles Can lower seizure threshold and increase gastrointestinal bleeding risk

Hepatic encephalopathy is a brain dysfunction caused by liver insufficiency and/or PSS; it manifests as a wide spectrum of neurological or psychiatric abnormalities ranging from subclinical alterations to coma

- Reduced ammonia secretion
- Electrolyte imbalance/disturbance
- Infections
- Dehydration
- Drugs

Ntuli Y, Mangini C, Tranah TH, et al Hepatic encephalopathy: Part 1, a diagnostic approachFrontline Gastroenterology Published Online First: 19 June 2025. doi: 10.1136/flgastro-2025-103135

HEPATIC ENCEPHALOPATHY

1. Lactulose (non-absorbable disaccharide)

- Aim for 2-3 soft stools per day
- Promotes growth of beneficial micro-organisms
- Reduces gut protein load
- Lower colonic pH which discourages ammonia producing bacteria

(Enemas can be used when patient is constipated)

2. Rifaximin

- 550mg twice daily
- Semi-synthetic rifamycin derivative, poorly absorbed (reduced systemic side effects)
- Broad-spectrum with activity against aerobic and anaerobic, gram positive and negative organisms
- Binds to the β-subunit of bacterial DNA-dependent RNA polymerase resulting in inhibition of bacterial RNA synthesis.
- Reduces gut bacteria that would produce ammonia, reduces absorption of ammonia from intestinal lumen

3. LOLA

PRURITUS

- Pruritus secondary to cholestatic liver diseases can be debilitating.
- It is characterised by a diurnal rhythm, with the highest intensity at night and often in the limbs.
- No primary rash and affects palms and soles
- Reported among 80-100% of patients with diseases such as PBC, PSC, and intrahepatic cholestasis of pregnancy.
- Management is challenging due to poorly understood pathogenic mechanisms.
- It is thought that there are several pruritogens involved in mediating cholestatic itch.
- These include bile acids, lysophosphatidic acid (LPA), endogenous opioids, and progesterone derivatives.
- Exacerbated by heat, pregnancy, premenstrual, HRT therapy

NON PHARMACOLOGICAL

Environmental conditions and clothing options	• Low room temperature where possible and avoid hot environments and dry conditions • Wear light clothes made from natural fibers, such as cotton • Avoid clothing that is woolen or tight, and avoid the use of overly scented detergents
Lifestyle and relaxation	• Record daily activities to identify any precipitants • Avoid consuming any food or drink that the patient feels may exacerbate their pruritus (spicy or acidic food and drink, stimulants (e.g. coffee, tobacco and alcohol), chocolate and nuts • Cold, wet compress or ice packs • Distraction techniques, for example using a stress ball or gently putting pressure on an area of skin not affected by pruritus
Hygiene	• Shorten their fingernails • Hydrate their skin immediately after a bath • Cold showers or baths • Bathe in warm water for less than 20 minutes • Avoid cleaning products that may irritate the skin, e.g. alcohol-based soaps
Physiological intervention	• Behavioral therapy for addictive scratching/ scratch dependence • Psychological support due to stress, anxiety or feelings of depression as a result of pruritus • Avoidance of unnecessary stress (e.g. overworking), practicing relaxation techniques such as meditation and yoga, and activities that provide the patient with a sense of well-being (e.g. a scenic walk)

COLESTYRAMINE

- The **only** licensed medication for the treatment of pruritus.
- 4-12g once daily (can go to 16g rarely)
- Patients should take other drugs 2 hours before or 4-6 hours after colestyramine to minimise possible interference with absorption.
- Can be discontinued if not tolerated.
- GI symptoms- constipation most commonly
- If patients struggle with colestyramine can be switched to colesevelam tablets (other drugs should be taken 4 hours before or 4 hour after this).
- If no improvement after 4 weeks at maximum tolerated dose, stop and move to second line.

RIFAMPICIN

- 150mg-450mg once daily; maximum 600mg daily
- EASL/BSG: Second line agent
- **LOTS of drug-drug interactions!** Due to enzyme inducing effects (CYP450, also PGP, UGTs, GSTs)
- Beware of effect on: oral contraceptives, anti-epileptics, thyroid hormone replacement, anti-diabetics (sulfonylureas), statins
- Note: enzyme inducing effect can continue for up to 4 weeks after stopping rifampicin
- Side effects include: hepatotoxicity, haemolysis, increased INR due to induction of vitamin K metabolism, turns secretions orange
- Important to monitor liver function tests 2-4 weeks after starting treatment and for any signs of hepatotoxicity (nausea, jaundice, malaise etc)
- Try 8 weeks at the maximum tolerated dose

BEZAFIBRATE

- 400mg modified release tablets once daily or IR 200mg twice daily taken after a meal either morning or night.
- Must check renal function, if CrCl <60ml/min switch to immediate release formulation and reduce the dose.
- Monitor for myopathy if using in combination with a statin/or patient has risk factors for myopathy.
- Also used off license as a second line therapy in patients with PBC who are non-responders to UDCA.
- Small risk of hepatoxicity

Fibrates for Itch (FITCH) in Fibrosing Cholangiopathies: A Double-Blind, Randomized, Placebo-Controlled Trial - PubMed (nih.gov)

NALTREXONE

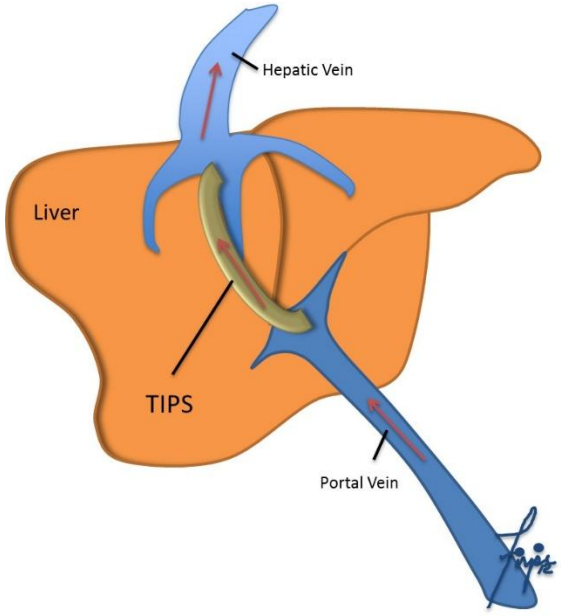
- Initial dose of 12.5mg daily which can be up-titrated to a maximum dose of 50mg daily
- Cautioned in hepatic impairment as extensively metabolised by the liver.
- Cautioned in renal impairment as naltrexone predominantly excreted in the urine.
- Cannot use in patients dependent on opioids (risk of acute withdrawal syndrome)
- Common side effects include anxiety, insomnia, headache, abdominal pain

SERTRALINE

- 100mg daily, can start low and up-titrate the dose.
- Thought to have an antipruritic effect due to changes in serotonin and histamine levels.
- Should not be initiated for cholestatic pruritus without hepatology input
- Should not be used in severe hepatic impairment as no clinical data available
- Avoid abrupt discontinuation

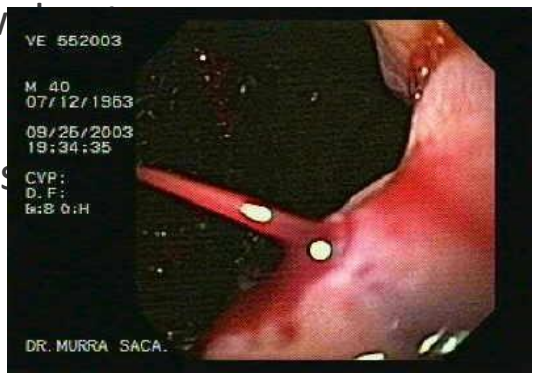
ASCITES

- Diuretics
  - Spironolactone, furosemide
  - Resistance/concomitant renal failure
  - Electrolyte disturbance
- Paracentesis
  - Day case
- Long term drains / alfapumps
- TIPSS
- Reduce salt intake



PORTAL HYPERTENSION

- Portal hypertension is caused by increased resistance to flow
  - disruption of hepatic architecture
  - compression of hepatic venules by regenerating nodules



- Collateral vessels form – enable blood to bypass the liver

- Variceal bleed is a serious complication of pHTN(>12mmHg) with ↓ clotting factors/vitamin K absorption

- Bleeding varices – mortality 50% index bleed and 30% for subsequent bleeds.

PRIMARY/SECONDARY PROPHYLAXIS

- Propranolol
- Carvedilol
  - More superior Carvedilol is a **non-selective beta blocker (NSBB)** with **additional α_1-blocking activity**, which reduces both cardiac output (β_1 effect) and intrahepatic vascular resistance (α_1 effect).
    → This leads to a **greater reduction in portal pressure** compared with propranolol
  - Start low, go slow — 6.25 mg → 12.5 mg/day
  - Target HR: 55–60 bpm
  - Target SBP: >90 mmHg
  - Avoid in refractory ascites with low MAP or AKI.
  - Restart cautiously after acute events.

DRUG INDUCED LIVER INJURY

- Incidence of DILI appears to be increasing
- Hepatocellular/ cholestatic / mixed
- ALT values >3×ULN or ALP values >2×ULN are indicative of DILI
- Liver Tox helpful - https://www.ncbi.nlm.nih.gov/books/NBK547852/
- A number of drugs have been withdrawn due to hepatotoxicity concerns

Generally accepted patients with pre-existing liver disease do not have an increased susceptibility to developing hepatotoxicity

BUT

effects hepatotoxicity may be more severe due to reduced hepatic reserve

INTRINSIC VS IDIOSYNCRATIC REACTIONS

Intrinsic

- Predictable
- Reproducible
- Dose dependent
- Tend to occur rapidly e.g. within hours
- Tend to cause necrosis, acute liver failure
- E.g. paracetamol overdose

Idiosyncratic

- Not predictable
- Not reproducible
- Not dose dependent
- Tend to take longer to occur – weeks to months
- Can result from metabolic idiosyncrasy or immunoallergic reaction
- Can cause any type of liver injury e.g. increased LFTs, jaundice, fever, rash, eosinophilia
- E.g. NSAIDS (metabolic), carbamazepine (immunoallegic)

RISK FACTORS

- Gender (tends to be more common in females)
- Age
- Genetics
- Concurrent diseases e.g. obesity, diabetes, co-infection with HIV
- Polypharmacy

STATINS

- Statins are not recommended in patients with:
- **unexplained** active liver disease or,
- **unexplained** elevations of alanine aminotransferase (ALT) or aspartate aminotransferase (AST) that are greater than 3 x the upper limit of normal (ULN).
- Chronic liver disease patients without cirrhosis or with compensated cirrhosis, requiring statins as per the National Lipid Management Pathway **should not be** routinely excluded from statin therapy.
- For patients with baseline elevated serum transaminases (2-3xULN) or compensated cirrhosis, closer monitoring is required, within 2-4 weeks after starting or adjusting statin doses.
- Rosuvastatin is the preferred choice of high intensity statin therapy in compensated cirrhosis for **primary prevention** of cardiovascular disease.
- Consider initiating at a lower dose (such as rosuvastatin 5 mg -10 mg daily) and titrating dose gradually.
- Minor increases in liver enzymes may occur in the first few months; if levels exceed 3xULN, discontinue the statin and reassess.
- Consider a rechallenge with a lower intensity statin with a lower risk of drug induced liver injury like pravastatin.
- In decompensated cirrhosis, statin use should be carefully considered and discussed with a hepatologist.

Using statins in liver impairment – SPS - Specialist Pharmacy Service – The first stop for professional medicines advice
Position-statement-Initiation-of-statins-for-primary-prevention-of-cardiovascular-disease-in-patients-with-Liver-disease.pdf

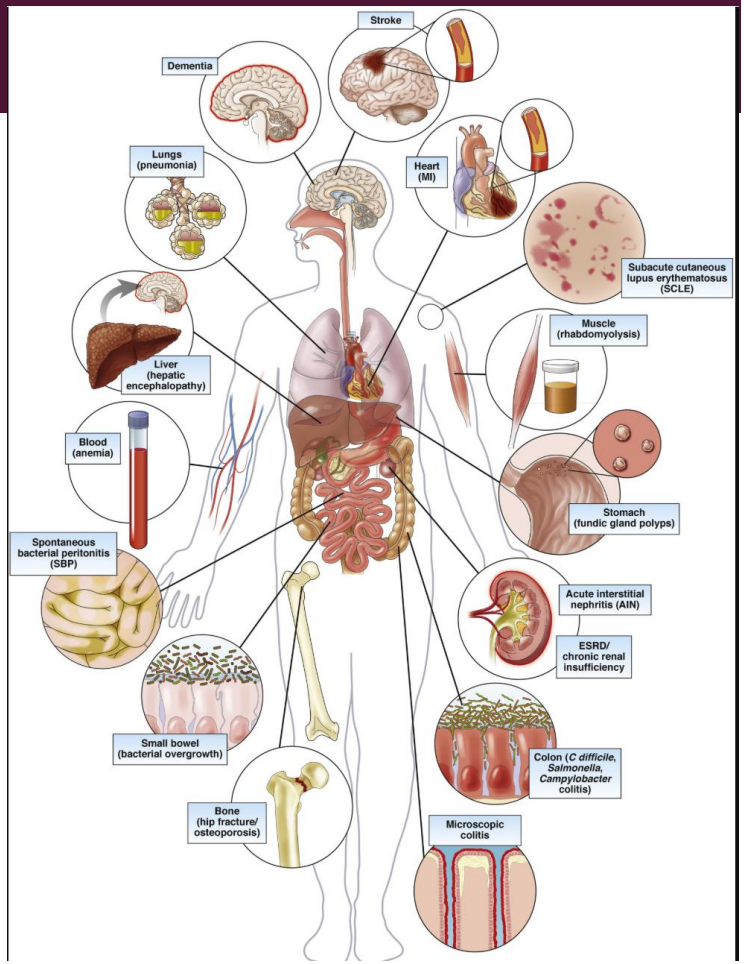
DEPRESCRIBING

PPIs

- ↑ Infection risk: Associated with Spontaneous Bacterial Peritonitis (SBP), Clostridioides difficile, and pneumonia due to gastric acid suppression altering gut microbiota.
- ↑ Hepatic Encephalopathy: Bacterial overgrowth and translocation may increase ammonia production.
- ↑ Mortality: Several cohort studies show higher all-cause and infection-related mortality in cirrhotic patients using PPIs.
- Renal and electrolyte effects: Risk of acute interstitial nephritis and hypomagnesemia can compound multi-organ dysfunction in liver disease.

Vitamin B co-strong – No evidence in ArLD

Thiamine – Stop once > 6 weeks abstinent



MEDICINES ADVICE

- Ask about the patient
- Ask about liver disease
- Ask about liver impairment (bloods/scans/signs/symptoms)
- Full Dhx (OTC/Herbal/Weight loss injections)
- Look at information sources
  - BNF (very poor in liver disease)
  - SmPC – good for understanding PK/PD, side effects
  - Liver Tox
  - Societies – BSG, BASL, BHPG, EASL
- Make a decision
  - Weigh up risk vs benefit
- Seek further advice
  - Contact your friendly local liver pharmacist

Questions to ask when giving medicines advice in liver impairment – SPS - Specialist Pharmacy Service – The first stop for professional medicines advice

QUESTIONS

- sitalshah@nhs.net
- British Hepatology Pharmacy Group
  - BASL
- RPS e learning module