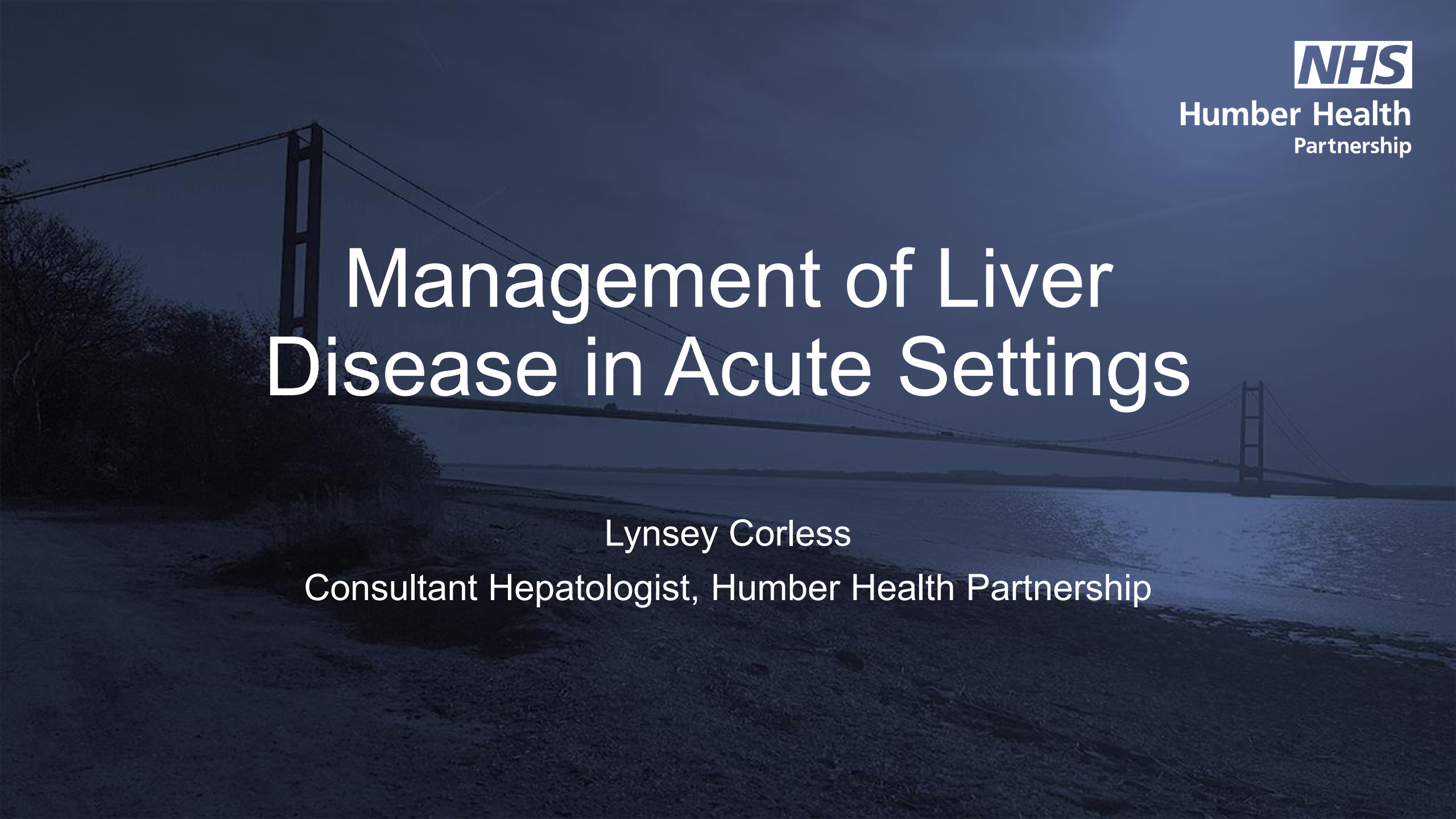

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Management of Liver Disease in Acute Settings

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Declarations of interest

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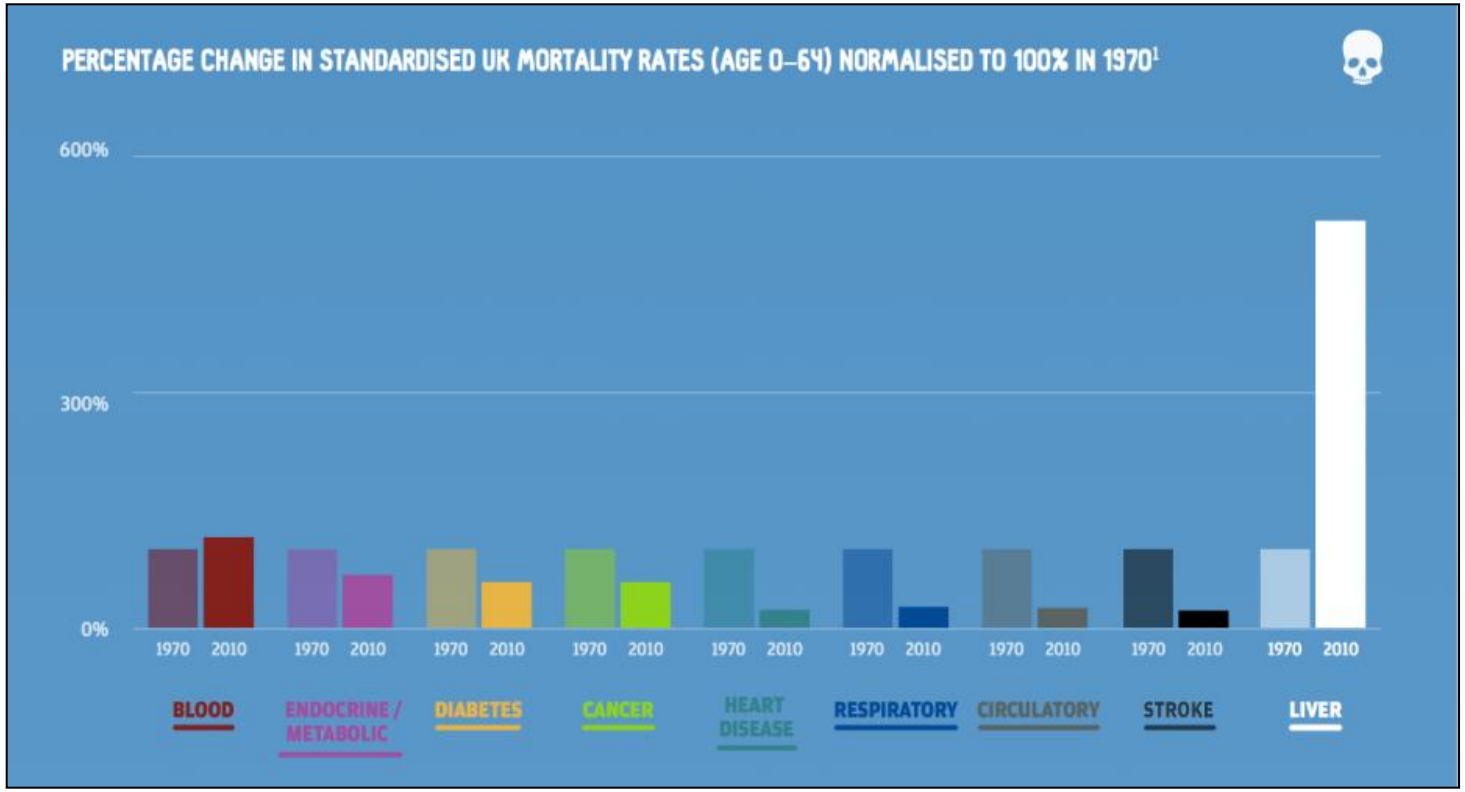
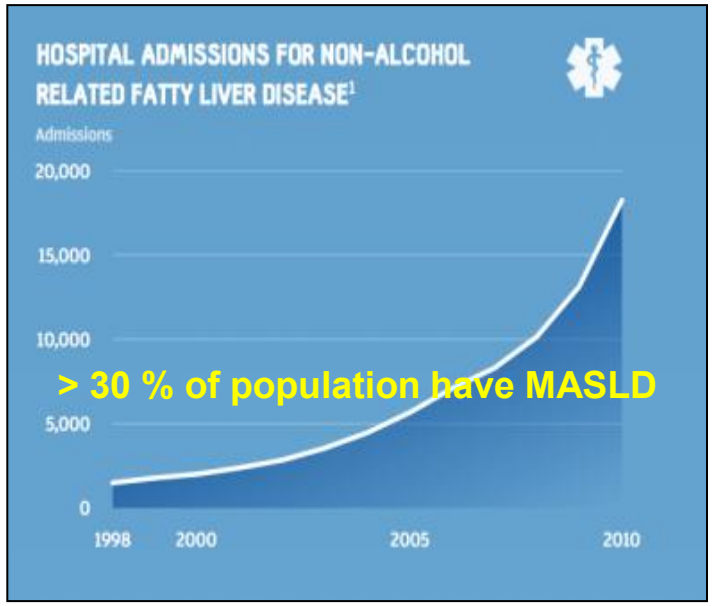
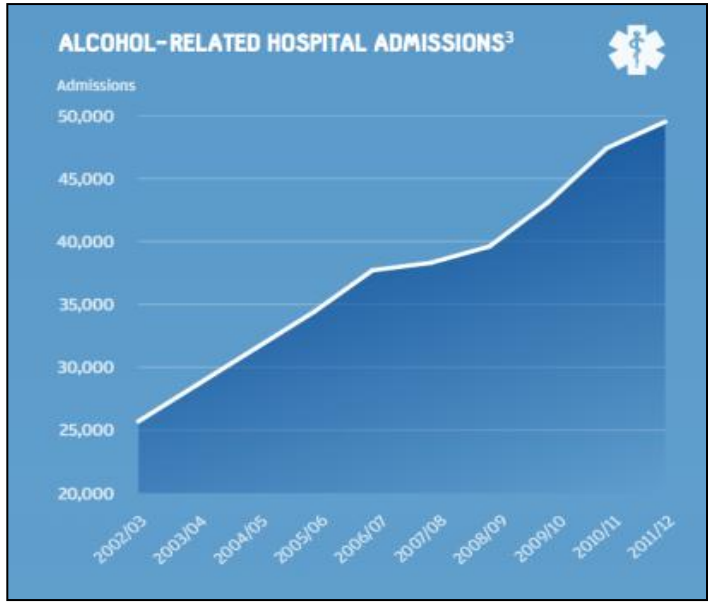
- I have no declarations or financial interests to declare.







All physicians regularly manage patients with liver disease

Williams et al., Lancet. 384:1953 - 1997

Overview

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- Good management of decompensated cirrhosis
  - Recognising those at highest risk
  - Updated guidance for acute management

- Incidental finding of liver disease – who needs liver clinic?
  - New terminology for steatotic liver disease
  - Identifying risk of significant liver disease

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Good management of decompensated cirrhosis

Early management determines outcome

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- Overall acute decompensation (AD) mortality 10-20 %
- AD admissions in those with ArLD up 57 % in 10 y
- Premature liver mortality ↑20 % since pandemic
- Good outcome = rapid identification/management of triggers
  - First 6-12 hr critical, i.e. acute settings
- Stigma
  - All alcoholics
  - All die
  - A lot going on

http://www.bsg.org.uk/

Not all decompensation is the same

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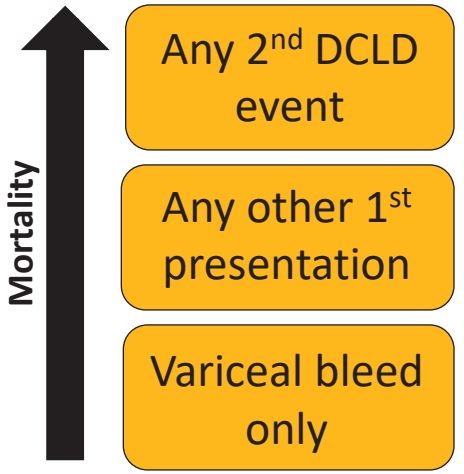
- AD can occur at any stage 2º to stressor
  - Whatever the trigger, LFTs will worsen in similar pattern
- Need to identify those at highest risk of death
  - Excellent supportive management
  - More aggressive intervention
  - Decision making
- 3 broad strata based on 2 yr survival
  - Plus the very sickest, with worst outcomes
- Most people stratify by Child's score



Gustot & Moreau J Hepatol 2018 69;1384-93.

Measure	1 point	2 points	3 points
Bilirubin	< 35	35 - 50	> 50
Albumin	> 35	28 - 35	< 28
INR	< 1.7	1.7 – 2.2	> 2.2
Ascites	None	Controlled	Uncontrolled
Encephalopathy	None	Grade I/II	Grade III/IV

Points	Class	1 yr (%)	2 yr (%)
5 - 6	A	100	85
7 - 9	B	81	57
10 - 15	C	45	35

Severity of liver disease | **Physiological reserve** | **Superadded complications** | **Timing of intervention**

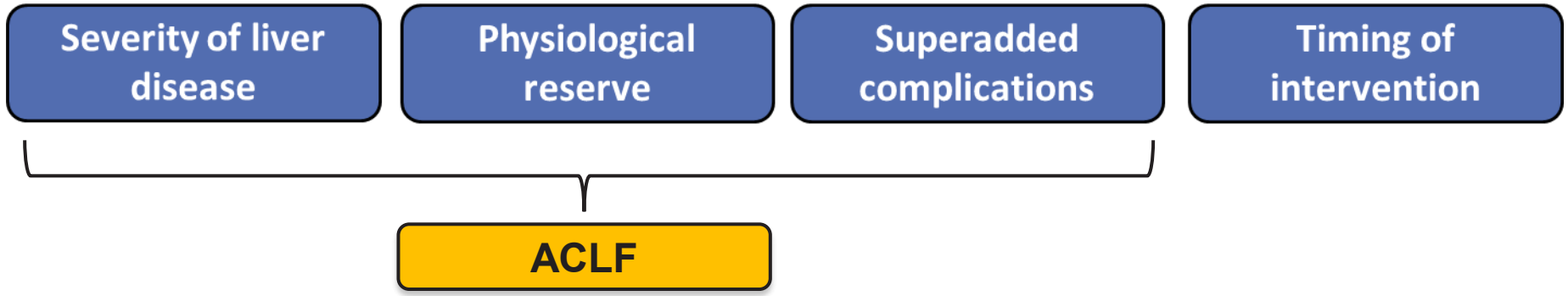
Liver status is only one piece of the puzzle

Child's score is good for long-term prognosis, but doesn't identify those at highest risk today

Acute on chronic liver failure = high short-term risk

Acute decompensation + organ failure

- AD = ascites, bleed, encephalopathy and/or bacterial infection
- Organ failure = respiratory, renal, brain, coagulation, circulatory, liver



Arroyo et al., J Hepatol 2015 62 j S131–S143

ACLF is a critical determinant of outcome

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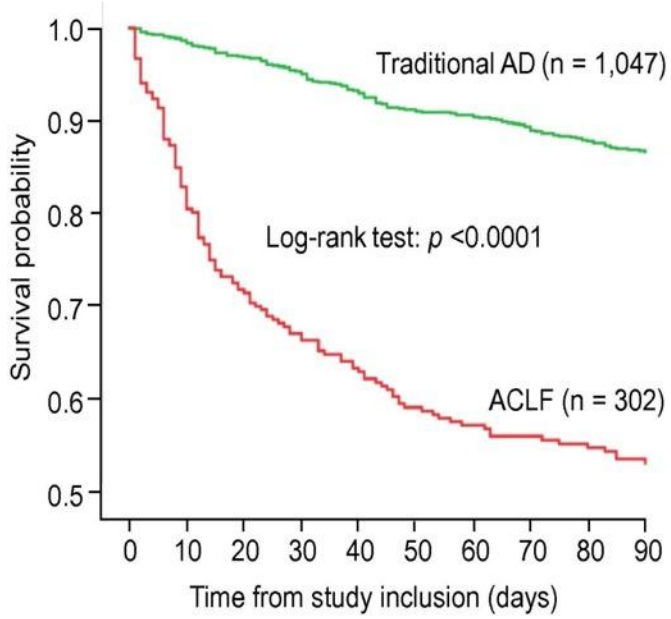
- Prevalence 30% (20% admit; 10% during)
  - Best predictor of mortality in AD
  - Overall median survival 18 vs 455 days*
- ACLF presence/absence allows stratification
  - Early involvement of Gastro/Critical Care
  - Aggressive treatment + closer monitoring

http://www.clifconsortium.com/aclf-calculator/



ACLF is reversible and preventable

Suspect ACLF:

- Renal failure (creat ≥ 175 μmol/L)
- Brain failure + renal dysfunction (> 130 μmol/L)
- Renal or brain dysfunction + other organ failure

Burke et al., J Hepatol 2017;66:S333-542.

Gustot & Moreau J Hepatol 2018 69;1384-93.

Arroyo et al., J Hepatol 2015 vol. 62 j S131–S143.

Good early care benefits everyone

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- Identification of triggers and stratification of severity enables faster treatment and decision making
  - Better care
  - Better use of resource
- Care bundles support early management of complex disease
  - BSG/BASL DC bundle 2014 improved care
  - But only used in 11.4% of patients
- Updated bundle to include new advances and improve usage, with involvement of SAM

McPherson S, Abbas N, Allison MED, et al. doi: 10.1136/flgastro-2025-103074

Decompensated cirrhosis: an update of the BSG/BASL admission care bundle

Patient details

Decompensated Cirrhosis Care Bundle - First 6 hours

ALL patients presenting with symptoms/signs of decompensated cirrhosis[1]

- *Bloods:* FBC, LFT, U/Es, clotting, Ca^{2+}, PO_4, Mg, CRP, lactate, glucose ❑
- *Septic screen:* CXR ❑ Urine dipstick ❑ Blood cultures ❑
- *Clinical ascites:* Ascitic tap ESSENTIAL[2]; cell count, MCS, protein, albumin ❑
- Request USS abdomen including Doppler of hepatic and portal vein ❑
- VTE prophylaxis, unless platelets <50 or active bleeding ❑
- Referral to dietetics made ❑
- Specialist review: refer to GI/liver team at earliest opportunity ❑

Ongoing alcohol intake? *(If NO- move to next section)*

- IV Pabrinex/Thiamine as per hospital guidelines ❑
- Commence CIWA or GMAWS according to hospital guidelines ❑
- Monitor for refeeding syndrome ❑

N/A ❑

Spontaneous Bacterial Peritonitis? *(If NO- move to next section)*

- *(Diagnosis: Ascitic neutrophils >250/mm3 or >0.25 x 10^9/L)*
- Prescribe 1.5g/kg of 20% Human Albumin Solution (HAS) ❑
- Antibiotics as per hospital guidelines ❑

N/A ❑

Acute Kidney Injury as per KDIGO criteria[3] *(If NO- move to next section)*

- Suspend all diuretics and nephrotoxic drugs ❑
- Fluid resuscitate with crystalloid in 250ml boluses ❑
- Strict urine output monitoring ❑

At 6 hours, if deteriorating despite this, obtain senior review and consider escalation to ITU/HDU.

N/A ❑

GI bleeding AND varices suspected? *(If NO- move to next section)*

- Target Hb 7-8 g/L, but if massive bleeding aim for Hb >8g/L ❑
  It is not recommended to routinely correct INR/APTT with blood products (unless on anticoagulants).
- Terlipressin: if no clear contraindications[4] prescribe 2mg stat IV followed by 2mg QDS ❑ (*If contraindication to Terlipressin, contact on-call GI bleed team and consider dose reduction/alternate agent*[4])
- Prescribe prophylactic antibiotics as per hospital guidelines ❑

N/A ❑

Symptoms/signs of Hepatic Encephalopathy?

- Lactulose 20-30mls QDS or phosphate enema ❑
- If clinical concern, for CT head to rule out a subdural haematoma ❑

N/A ❑

Name ..... Grade ..... Date ..... Time ..... ([1,2,3,4]) Please turnover for additional information

([1,2,3,4]) Important additional information

[1] Presentation of Acute Decompensation of Cirrhosis

Jaundice
Ascites
Hepatic Encephalopathy
Suspected Variceal Haemorrhage

[2] Diagnostic Ascitic Tap

Performed with a green needle, IRRESPECTIVE of clotting parameters.

Ensure ascitic fluid goes into universal container bottles for fluid albumin, MCS (with WCC differential) and blood culture bottles (minimal 5mls each bottle) to maximise yield of diagnosis of SBP.
Human Albumin Solution (HAS): 20g of albumin in 100ml of 20%.

[3] Acute Kidney Injury as per: Kidney Disease Improving Global Outcomes criteria (KDIGO)

1. Increase in serum creatinine ≥ 26 µmol/L within 48 hours *or*
2. ≥50% rise in serum creatinine over the last 7 days *or*
3: Urine output (UO) <0.5mls/kg/hr for more than 6 hours based on dry weight *or*
4: Clinically dehydrated.

[4] Variceal Haemorrhage

Contraindications to Terlipressin:
Absolute- Hypersensitivity, pregnancy, acute respiratory distress/hypoxia, septic shock, Creatinine ≥ 442µmol/l.
Relative- Age >70, peripheral arterial disease, prolonged QTc, cardiac arrhythmia, uncontrolled hypertension, acute coronary syndrome, previous myocardial infarction.

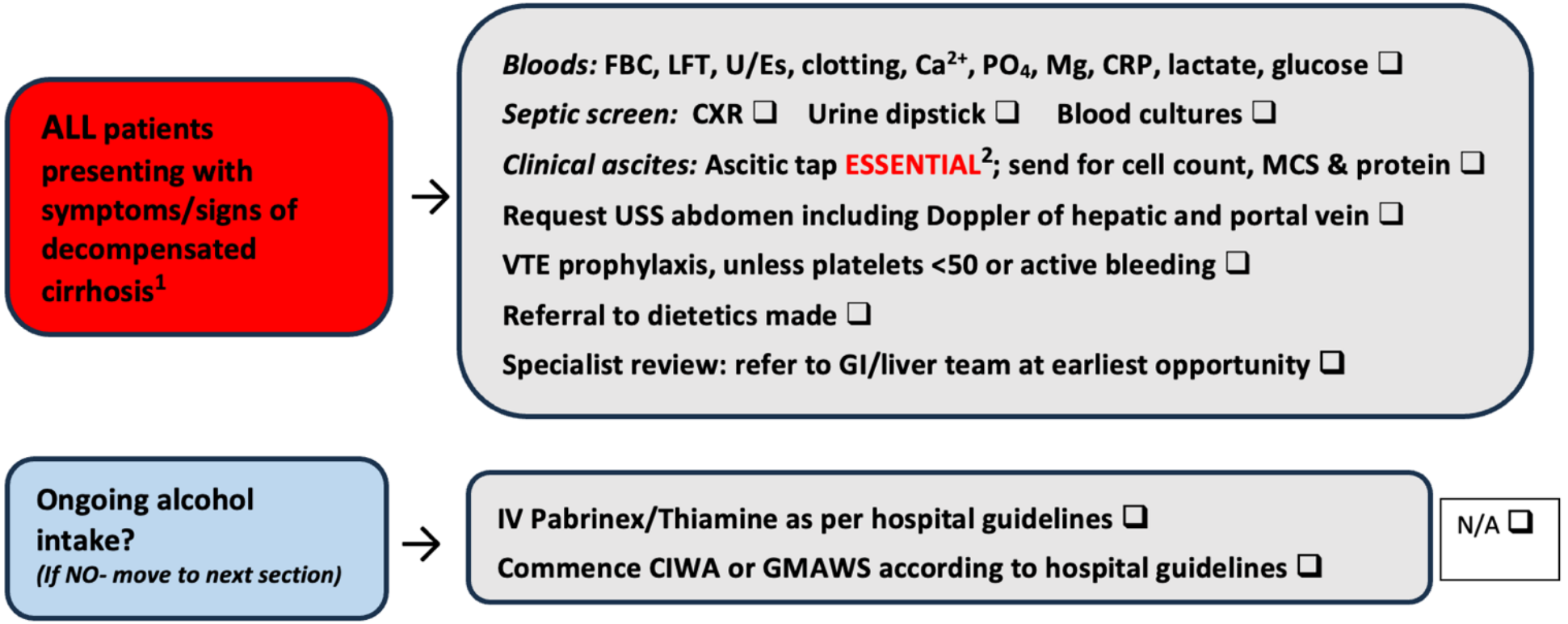
Alternative to Terlipressin:
Octreotide: 50 micrograms bolus followed by 25-50micrograms/hr infusion.

Suspend B blockers if Terlipressin/Octreotide commenced.

Stable patients: Routine administration of platelets, FFP, PCC and other products to correct haemostatic tests is ***not*** recommended outside of patients taking anticoagulants.

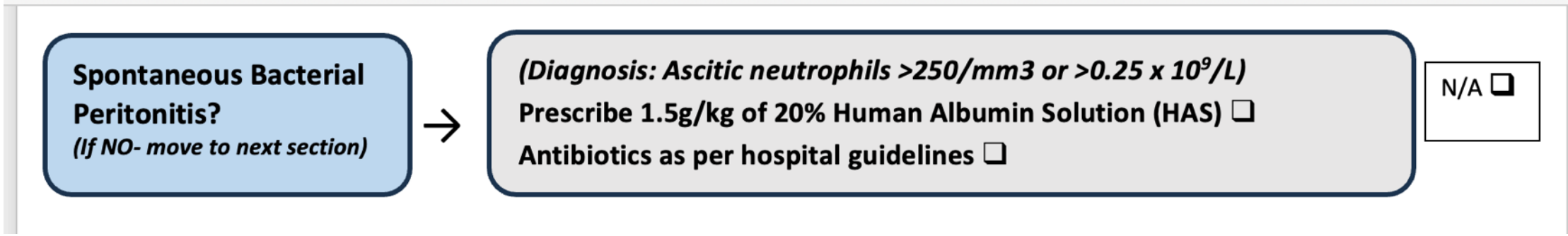
Unstable patients: Discuss with the upper GI bleed team +/- Haematologist +/- and consider major haemorrhage protocol. Avoid FFP in portal hypertension. Critical care review.

https://fg.bmj.com/content/early/2025/04/18/flgastro-2025-103074



Alcohol

- 75% have alcohol as major aetiology
- Consider lorazepam in ACLD
- Advise working towards complete alcohol abstinence
- Involve Alcohol Care Team/Community Support



- 8-10% of hospitalised patients with cirrhosis
  - Prompt treatment with antibiotics improves prognosis
- Tap for all with clinically detectable ascites
  - Each hour of delay increases mortality
  - Delay 12 hr+ = 2.7-fold ↑ mortality risk
  - Coagulopathy not contraindication - no correction required
- High risk of HRS:
  - IV HAS 20% 1.5g/kg @ dx; 1g/kg @ 72 h
  - Reduces HRS incidence from 30% to 10% and mortality from 29% to 10%

Sort P, et al. N Engl J Med 1999;341:403–9.
Kim JJ, et al. Am J Gastroenterol. 2014. doi: 10.1038/ajg.2014.212.

Importance of bacterial infection

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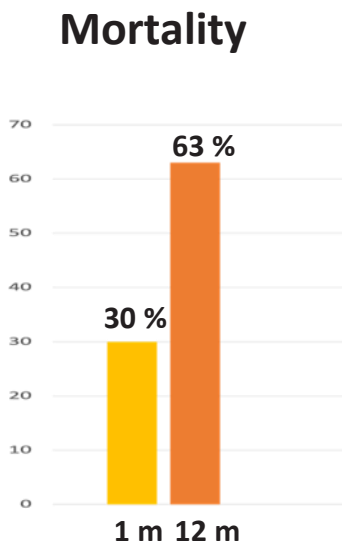
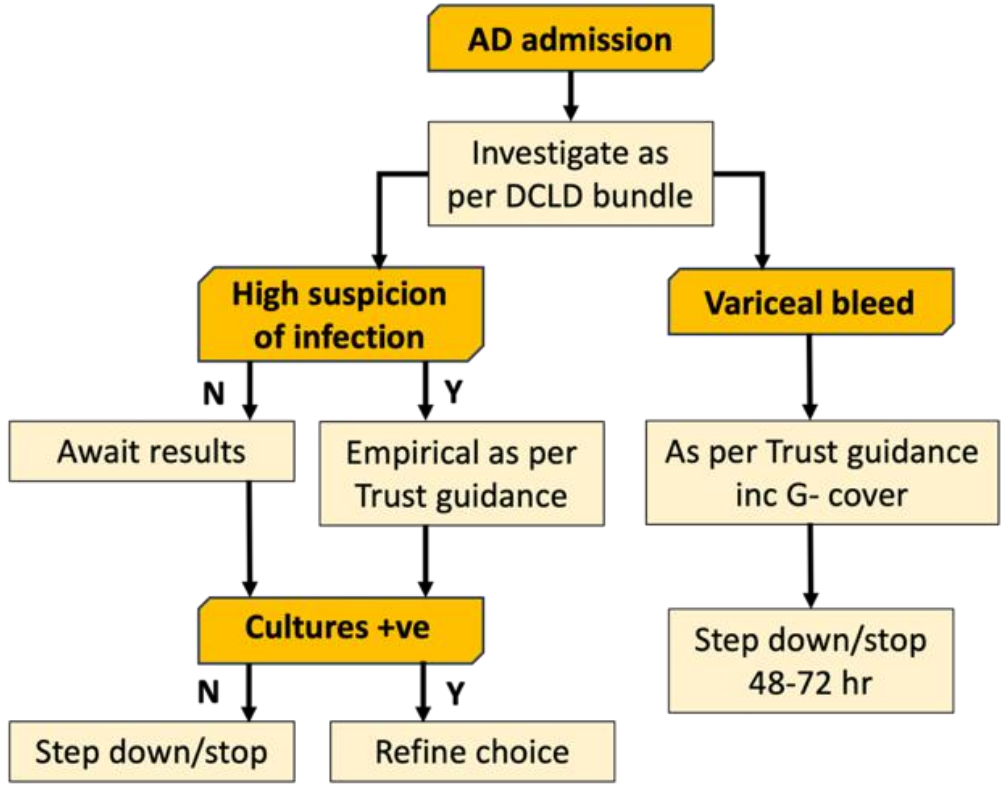


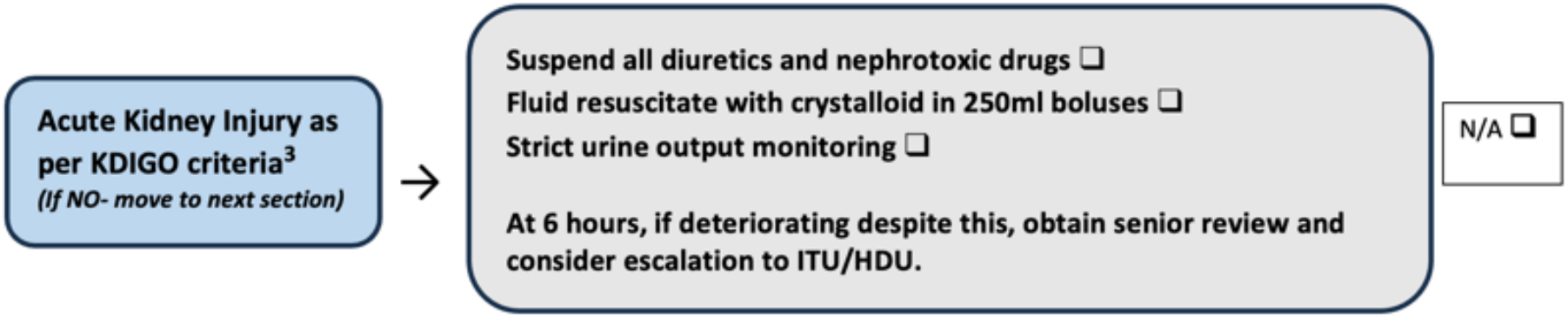
- Huge problem
- Commonest precipitant of AD
- Complicates 20-40% of AD admissions
- Frequent trigger of ACLF
- High mortality in DCLD
  - 3.75x ↑ mortality risk



Foreman MG, et al. Chest. 2003. doi: 10.1378/chest.124.3.1016.

Fricker Z, et al. Hepatol Commun. 2024. doi: 10.1097/HC9.0000000000000356.

Mücke MM, et al. Aliment Pharmacol Ther. 2024. doi: 10.1111/apt.17899.



- AKI occurs in 20%–30% of AD, and associated with poor outcome
- Staged according to % rise in creatinine from baseline
- Often multifactorial, usually not HRS
  - Prerenal commonest (45%), ATN/GN (32%), HRS (23%)

Stage 1: increase sCr ≥26.5 µmol/L or increase ≥1.5-2x baseline
Stage 2: increase sCr >2 - 3x baseline
Stage 3: increase sCr >3 x baseline or sCr ≥ 353.6 µmol/L with acute increase ≥ 26.5 µmol/L or initiation of RRT

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AKI in cirrhosis

Rise in creatinine ≥ 26 umol within 48 hours
OR rise in creatinine ≥ 1.5x from baseline

Urine output <0.5ml/kg/hr for >6 consecutive hours

Consider ITU support for RRT particularly with the presence of:

Acid-base imbalance
Significant electrolyte impairment/ refractory to replacement
Volume overload
Uraemic encephalopathy

Immediate action for all:

Suspend diuretics
Suspend B blockers
Suspend NSAIDS/nephrotoxic drugs
Treat confirmed infections with antibiotics

If volume depletion:

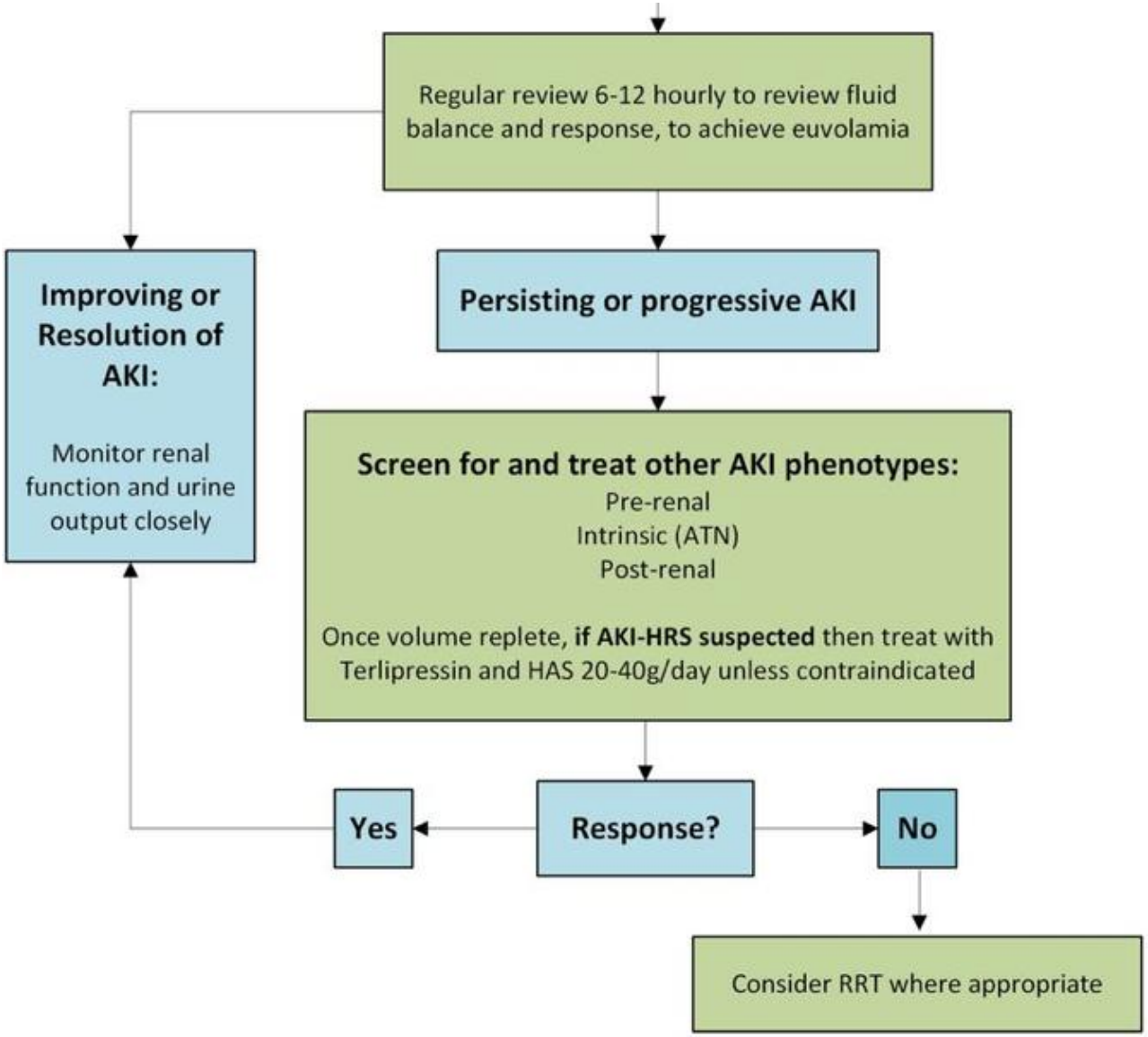
Haemorrhage: transfuse RBC (target Hb 7-8 for portal hypertension)
Vomiting/diarrhoea/diuresis: IV crystalloids with 250mls boluses (aim for urine output >0.5mls/kg/hr)
Consider 20% HAS 1g/kg (body weight) for plasma volume expansion if ≥ stage 1B AKI or confirmed infection

If tense ascites: offer limited therapeutic paracentesis with HAS cover (if no SBP)

1–2 L IV crystalloid will correct most losses and restore euvolaemia

No evidence that routine use of 5% HAS is beneficial

McPherson S, Abbas N, Allison MED, et al. doi: 10.1136/flgastro-2025-103074

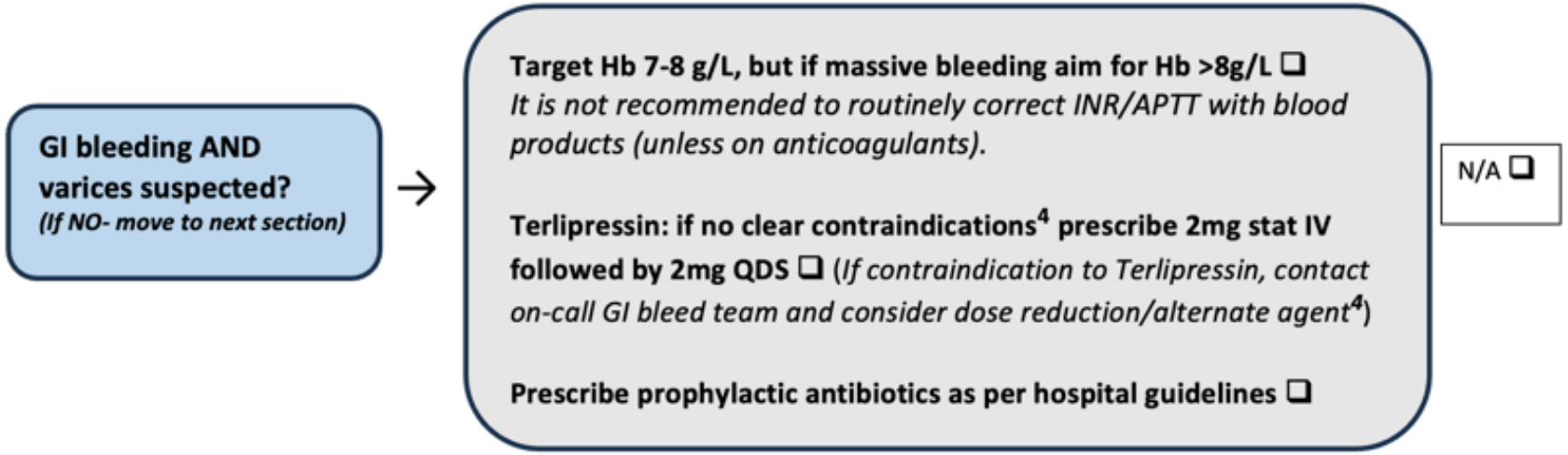


- **Consider escalation for fluid management, invasive monitoring, vasopressors or RRT**
- **RRT case-by-case (only 25% will have renal recovery)**

HRS

- Cirrhosis with ascites, and AKI
- No response to 2 days off diuretics + volume expansion with HAS (1g/kg wt/d)
- Absence of shock
- No signs of structural kidney injury

McPherson S, Abbas N, Allison MED, et al. doi: 10.1136/flgastro-2025-103074

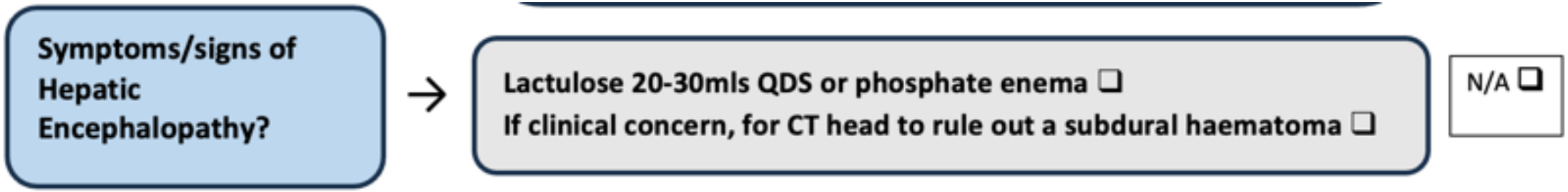


GI bleeding

- Resuscitation then OGD
- Octreotide if terlipressin contraindicated (Cr >442; ARDS etc)
- No PPI

No routine correction of clotting/platelet required if haemodynamically stable

- High volume FFP etc. can increase portal pressure + risk of rebleed
- Haemodynamically unstable = discuss with endoscopist/haematologist
- Tranexamic acid does not reduce mortality in UGIB but increases VTE risk



Encephalopathy

- Identify & Treat Precipitating Factors
  - Sepsis, GI bleeding, renal failure, electrolyte abnormalities, constipation, sedatives, over-diuresis
- Ammonia levels usually >100 in acute HE, but can be raised in other conditions
  - Normal ammonia reliably excludes HE

Admission to critical care

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- Although poor survival for cirrhosis in ICU - mortality ~ 40 %...
- ACLF outcome similar to non-CLD when matched for illness severity
- Liver disease is not a reason to discount escalation
  - Well compensated pre-event likely to do well
- Consider escalation:

Bleeding | **Sepsis** | **ACLF**

- Set clear parameters and review: 2-3 days ideal

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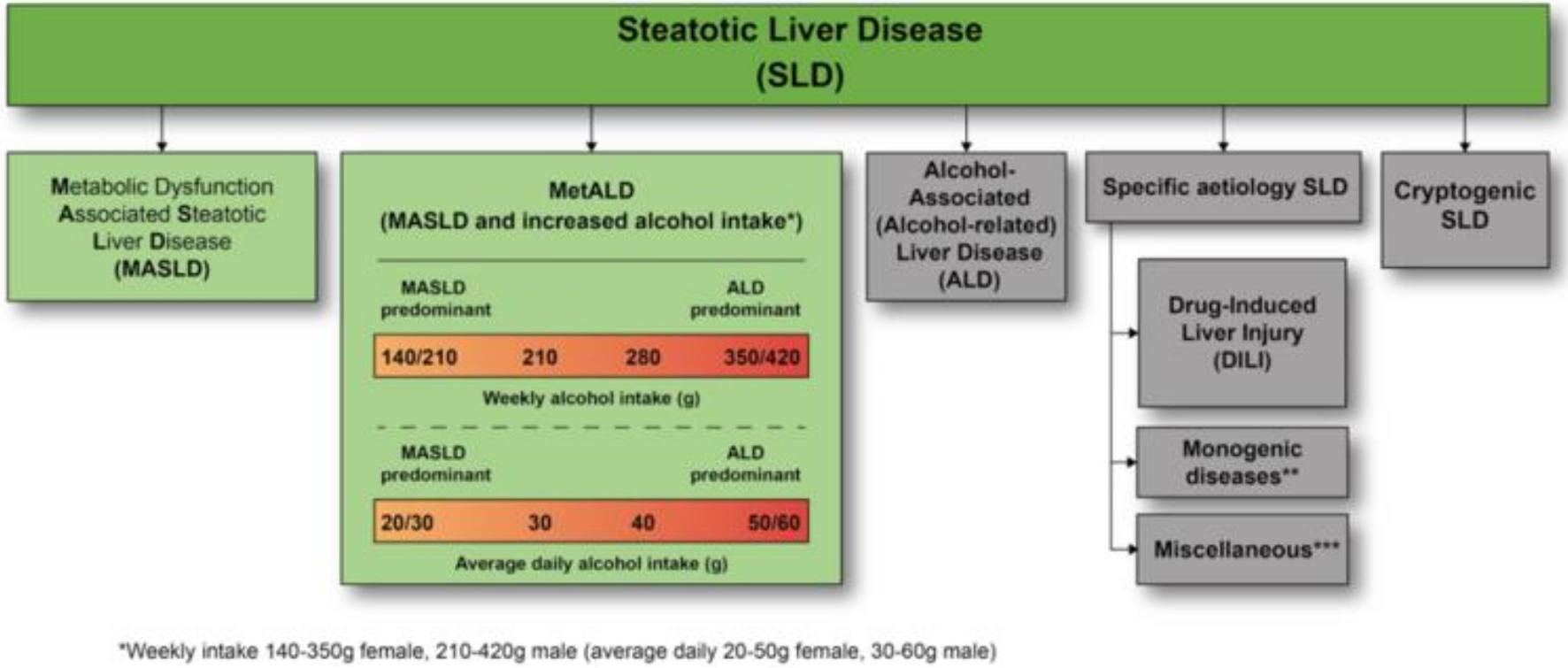
Incidental finding of liver disease – who needs liver clinic?

Steatotic Liver Disease Sub-classification



*Weekly intake 140-350g female, 210-420g male (average daily 20-50g female, 30-60g male)

- MASLD is the hepatic manifestation of metabolic syndrome
  - Very common in T2DM
  - Cardiovascular disease is leading cause of death in MASLD
- Most MASLD should be managed in primary care

In-patient liver fibrosis assessment

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- Advancing fibrosis is key predictor of liver-related events and mortality in MASLD
- Finding steatosis or unexplained abnormal LFT should prompt fibrosis risk assessment
  - Low risk – manage in primary care to reduce cardiovascular mortality
  - Higher risk – refer to GI/Hep to reduce risk of liver-related mortality
- Fib4 or ELF can be done on admission bloods
  - Fib4 not validated in <35s or ArLD
  - ELF <9.5 is OK
- Fibroscan – requires referral

Summary

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- Decompensation is a medical emergency
  - In-hospital mortality is mostly to do with other organ failure
  - Look for, and aggressively target, renal dysfunction and infection
  - Use the BSG/BASL/SAM bundle to guide management

- Liver disease is preventable and treatable
  - Opportunities to avoid the worst outcomes should be seized
  - Take action on incidental findings of liver disease

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Thank you