

1. Introduction

- 1.1 Over the last 20 years, there has been a significant rise in the prevalence of CLD in the UK (1). Decompensated CLD is also associated with a high mortality rate (2). The 2013 National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report on patients with alcohol-related liver disease also raised the alarms on the lack of good quality of care to this particular cohort of patients in hospital (3).
- 1.2 These guidelines set out the identification and prompt management of patients with decompensated cirrhosis within the first 24 hours of being admitted to the University Hospitals of Leicester NHS trust. This includes recognition of the different ways decompensated cirrhosis can present and identification and management of precipitants and complications.
- 1.3 These guidelines and associated sticker have been written with the aim of providing a pragmatic overview of the key steps in the assessment and management of patients with decompensated chronic liver disease (CLD) within the first 24 hours of admission. As any guideline, it should not be used to replace clinical judgement but rather, as an aid to ensure the delivery of good clinical care.
- 1.4 This guideline should be used in conjunction with other associated UHL policies (reference made where appropriate) AND the UHL decompensated CLD sticker (see appendix 1).

2 Scope

- 2.1 These guidelines apply to the first 24 hours of inpatient care of adult patients presenting to UHL with features suggestive of decompensated cirrhosis. It would be directly applicable to the emergency department, the acute medical unit, the ambulatory care unit and the wards. While every single step is not expected to be performed within one single clinical area (recognising limitations such as time constraints within the Emergency Department), it is expected that all the steps will be performed as promptly as possible within the first 24 hours of care. (see section 2.4)

3 Aims

- 3.1 The aim of this document is to reduce complications of decompensated cirrhosis by identifying and managing precipitants in a timely fashion through the promotion of evidence based practice in line with recommendations from the British Society of Gastroenterology and the British Association for the Study of the Liver (4). This process is facilitated by the use of the attached sticker (Appendix 1).

4 Recommendations, Standards and Procedural Statements

It is expected that all patients admitted with decompensated CLD should have the attached sticker (Appendix 1) stuck on their medical notes and filled in appropriately and contemporaneously by the doctors (of any grade) looking after the patient. The first box (with title ON ADMISSION) includes tasks that should be performed at the time of initial presentation.

The associated sticker will be made available in the relevant clinical areas (Acute Medicine, ED and Gastroenterology).

4.1 Presentation of Decompensated Cirrhosis

Decompensated Cirrhosis or CLD is defined as an acute deterioration in liver function in patients with established cirrhosis or those with new presentations of cirrhosis, that can present as one (or more) of the following:

- Jaundice
- Worsening ascites
- Confusion/ psychomotor slowing due to hepatic encephalopathy
- Renal Impairment
- Hypovolaemia

- Gastrointestinal Bleeding
- Signs of sepsis

Common precipitants of decompensated cirrhosis include:

- Gastrointestinal bleeding
- Dehydration
- Infection/ sepsis
- Constipation (may precipitate hepatic encephalopathy)
- Alcoholic Hepatitis
- Acute Portal Vein Thrombosis
- Hepatocellular Carcinoma (HCC)
- Drugs (alcohol, opiates, NSAIDS, etc)
- Ischaemic hepatitis

Initial Assessment and Investigations

- Full History (inc. potential aetiological factors if not known cirrhotic (such as full alcohol history, Type 2 Diabetes (leading to NASH cirrhosis)
- Examination with particular attention to the following
 - Fluid status (JVP, BP, HR, Urine output, skin turgor, weight, peripheral/ sacral oedema)
 - Signs of infection (inc. urine dipstick +/- MCS)
 - Signs of GI bleed
 - Ascites
 - Nutrition status
 - Signs of withdrawal
- Full set of bloods
 - FBC, UE, INR, LFTs, Ca, Mg, PO₄, CRP, Glucose, Blood cultures (if signs of sepsis)
 - Alpha Fetoprotein if not done in last 6 months (development of Hepatocellular carcinoma can be a cause for decompensation)
 - Consider Hepatitis B surface antigen/ Hepatitis C antibody and HIV 1 and 2
- Diagnostic ascitic tap if ascites present clinically (please [refer to UHL Ascites guideline](#) (C36/2010) – remember to dipstick the specimen before sending it for polymorphonuclear (PMN) and White cell count, microscopy and culture, protein and albumin content and start antibiotics if positive for leucocytes/nitrites) and consider sending for cytology.
- Abdominal Ultrasound (assess liver parenchyma, splenomegaly, hepatoma, portal vein patency, Common bile duct calibre and ascites)
- Chest X-Ray

4.2 Sepsis

Patients with cirrhosis are susceptible to infections due to their poor immune system (5). A keen investigative eye (as patients with cirrhosis do not always have pyrexia or a rise in CRP) is needed to look for sources of infection and a low threshold for initiation of antibiotics are required (6).

- [Refer to UHL Sepsis pathway](#) (B11/2014) if any signs of sepsis
- In CLD, the common sources are:
 - Chest
 - Urine
 - Spontaneous Bacterial Peritonitis

4.3 Ascites

- Perform diagnostic paracentesis (asepsis/local anaesthetic/green needle) and send specimens for microbiology and fluid albumin as per guidelines (C36/2010)
- Dipstick Ascitic fluid and start antibiotics if leucocyte/nitrite positive
- Definite diagnosis if PMN >250/mm³
- [Refer to UHL Ascites Guidelines](#) (C36/2010) for most up to date antimicrobial advice
- Hold off non-specific beta blockers (propranolol, carvedilol) (7)
- IV human albumin solution 1.5g/kg (~5 bottles of 500mls 4.5% HAS in the first six hours followed by 1 g/kg on day 3 (~3 bottles of 500mls 4.5% HAS) (8).

4.4 GI Bleeding

- [Refer to UHL Acute Upper GI Bleeding policy](#) (C33/2002) and [UHL variceal bleed guidelines](#) (C15/2008)
- Cirrhotic patients are likely to develop varices, which upon bleeding, carry high mortality rates.
- If known varices/portal hypertension – treat as variceal bleed.
- Particularly note the transfusion threshold of 7g/dl aiming for aHb of about 8 g/dl. Recent studies in patients with cirrhosis and GI bleeds have shown that a more conservative transfusion strategy (i.e. transfuse when Hb<7 g/dl) had lower mortality when compared to a more liberal strategy of transfusing when Hb<9 g/dL(9, 10).

4.5 Acute Kidney Injury And/Or Hyponatraemia

- [Refer to UHL AKI guidelines](#) (B21/2009) on inSite
- Definition of AKI:
- Absolute rise in serum creatinine of $\geq 26 \mu\text{mol/L}$ within 48 hours
- **OR** $\geq 50\%$ rise in serum creatinine within last 7 days
- **OR** Drop in urine output to less than 0.5 ml/kg/hour for 6 hours based on dry weight (11).
- AKI is associated with a poor prognosis in patients with cirrhosis (12). AKI in patients with cirrhosis can often be multi-factorial but pre-renal AKI is most common.
- Early intervention includes:
 - Stop all diuretics and nephrotoxins
 - Fluid resuscitation with 250 ml boluses of 4.5% HAS ideally (most losses will be corrected with 1-2 L of fluid). An alternative would be 0.9% sodium chloride though large volumes can worsen ascites. Aim for an improvement in urine output to more than 0.5ml/kg/hour based on dry weight. HAS can also correct hyponatraemia if the patient is intravascularly deplete ([Refer to UHL Ascites policy – section on hyponatraemia](#)) – C36/2010

4.6 Alcohol excess

- [Refer to UHL Acute Alcohol Withdrawal Management Policy](#) (B30/2014)
- Refer to Alcohol liaison team on 0753565839 or viaswitchboard
- Alcohol is the major cause of chronic liver disease affecting 70 % of patients admitted with cirrhosis to UK hospitals.
- For all patients, record the following in your clerking:
 - Current Alcohol history
 - Previous Alcohol history
 - Time of last drink
 - Symptoms/ Signs of alcohol withdrawal
- If current excess alcohol intake (above recommended limits), start
- Pabrinex 2 pairs of vials TDS for 3 days

- If dependant, please prescribe regular/prn benzodiazepines as per [the UHL acute alcohol withdrawal management policy](#) (B30/2014)

4.7 Hepatic Encephalopathy (13)

Assess for symptoms/signs:

- Signs of psychomotor slowing
- Confusion
- Reversal of sleep-wake cycle
- Liver Flap
- Drop in GCS

Assess for potential triggers

- Constipation
- Sedative drugs
- Signs of infection
- Occult bleeding
- Liver Flap
- Dehydration/ Electrolyte disturbance

If encephalopathic

- Start lactulose 20-30ml QDS aiming for 2 soft spontaneous bowel motions per day
- If low GCS, consider lactulose via NG tube and phosphate enema

Is this truly encephalopathy?

- Patients with CLD may be at risk of other intracranial causes of drops in GCS such as delirium from infections and from intracranial causes such as acute and chronic subdural (therefore a CT head may be warranted if clinically appropriate).
- Consider referral to ITU if Grade 3 – Grade 4 encephalopathy if clinically appropriate.

4.8 VTE thromboprophylaxis(14)

- [Refer to UHL VTE thromboprophylaxis policy](#) (B24/2006)
- Unless the patient is bleeding/ has a platelet of less than $50 \times 10^9/L$ other contraindication as per risk assessment, prescribe prophylactic low molecular weight heparin (recent evidence shows that even if patients have a high INR, patients with advanced cirrhosis may still be hypercoagulable if their platelet count > 50).

4.9 Escalation of care

This needs to be thought through from admission and reassessed periodically if the patient is not improving (current recommendations include a reassessment at 6 hour (4) but this should not stop escalation before if the patient is deteriorating).

The decision for escalation of care needs to be individualised and remains a senior-led clinical decision. Decisions regarding escalation of care should be discussed with the patient (if not encephalopathic/ confused) and available next of kin, if deemed appropriate.

Individual policies for [sepsis](#) (B11/2014) , [AKI](#) (B21/2009) and [GI bleed](#) (C33/2002) / [Variceal Bleed](#) (C15/2008) should be referred to for decisions on escalation of care in these specific circumstances.

5 Education and Training

No specific new skills or additional training is required for implementation of these guidelines.

Increased awareness of the guidelines will be done through departmental teaching sessions, through Grand Round presentations and further sessions can be arranged upon request.

Guidelines on the Assessment and Management of Decompensated Cirrhosis– The First 24 hours

6 Monitoring and Audit Criteria

The following standards are expected to be audited every 24 months.

Audit lead: Dr Allister Grant

Method: Audit tool – retrospective case note review

Ref	Audit standards- within first 24 hours of admission	Target	Exceptions
1	All patients with decompensated CLD should have a decompensated CLD sticker in their medical notes filled from admission	100%	
2	All patients with decompensated CLD should have FBC, UE, LFT, INR, CRP	100%	
3	All patients with decompensated CLD and with signs of infection (raised temp, high CRP, high WCC) should have Urine Dip, CXR and Blood Cultures	100%	
4	All patients with ascites secondary to decompensated CLD should have a diagnostic ascitic tap	100%	Technically challenging to do blind tap (hence needing US guided tap)
5	All patients with a history of alcohol excess should be prescribed IV Pabrinex - 2 pairs TDS; and referred to the Alcohol liaison team	100%	
6	All patients with suspected hepatic encephalopathy should be prescribed lactulose and/or phosphate enema	100%	
7	All patients with suspected infections, should be prescribed an appropriate antibiotic	100%	
8	All patients with suspected SBP should be prescribed an appropriate antibiotic and receive IV HAS	100%	
9	All patients with AKI or with Na <125 and with decompensated CLD should have their diuretics and nephrotoxins stopped	100%	
10	All patients with AKI and with decompensated CLD should have an accurate input/ output chart	100%	
11	All patients with pre-renal AKI and with decompensated CLD should receive IV fluids if signs of hypovolaemia present	100%	
12	All patients with a GI bleed and with signs of CLD should receive terlipressin, antibiotics and appropriate fluid resuscitation	100%	contraindication to terlipressin (such as IHD, PVD)
13	All decompensated CLD patients with a GI bleed and with a Hb<7 should receive blood transfusion	100%	
14	All decompensated CLD patients with a GI bleed should have an upper endoscopy within 12 hours of admission if haemodynamically stable	100%	

7 Supporting Documents and Key References

See Appendices 1 and 2

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9. Key Words

Cirrhosis, chronic liver disease, decompensated chronic liver disease, decompensated cirrhosis, ascites, variceal bleed, GI bleed, gastrointestinal bleed, haematemesis, malaena, hepatic encephalopathy, AKI, sepsis, alcohol withdrawal

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Author / Lead Officer:	Mohammad Farhad Peerally Toby Delahooke	Job Title: SpR Gastroenterology Consultant Gastroenterologist	
Reviewed by:	Toby Delahooke		
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Guidelines on the Assessment and Management of Decompensated Cirrhosis– The First 24 hours

APPENDIX 1

UHL DECOMPENSATED CIRRHOSIS CARE BUNDLE – THE FIRST 24 HOURS

Date: _____ Time: _____ Doctor: _____

<p>ON ADMISSION</p> <ul style="list-style-type: none"> - BLOODS - CXR - URINE DIP <p>If applicable:</p> <ol style="list-style-type: none"> 1. Treat sepsis 2. Manage GI Bleed 3. Manage AKI 4. Start Pabrinex 5. Manage withdrawal symptoms 6. CT Head 	<p>TESTS</p> <p>BLOODS: FBC/UE/LFT/INR/ Ca/MG/PO4/GLUC/Blood Cultures/CRP []</p> <p>ASCITIC TAP (send for MCS and Fluid Albumin) [] n/a</p> <p>URINE: Urine Dipstick/ MSU []</p> <p>IMAGING: Abdo US [] n/a CXR []</p> <p>SEPSIS</p> <p>ARE YOU SUSPECTING AN INFECTION? IF YES - REFER TO UHL SEPSIS PATHWAY n/a</p> <p>Source:</p> <p>Antibiotic</p>	<p>GI BLEEDING-see UHL GI bleed protocol [] n/a</p> <p>IV Terlipressin 2mg QDS (unless contraindicated) []</p> <p>Prophylactic antibiotics []</p> <p>If raised INR – IV Vit K 10mg stat []</p> <p>INR > 2.0 / Platelets <50 – liaise with Haematology SpR for IV FFP / IV platelets []</p> <p>Transfuse RBC if Hb <7.0 or massive bleeding []</p> <p>Early endoscopy after resuscitation (liaise with gastro SpR/GI bleed consultant) []</p>	<p>AKI or HYPONATRAEMIA (NA <125) – see UHL AKI guideline [] n/a</p> <p>Stop All Diuretics and Nephrotoxins []</p> <p>Fluid balance chart and daily weights []</p> <p>Commence IV fluids – ideally 4.5% HAS as replacement (1-2 L should correct most losses). Aim UO > 0.5ml/kg/hr []</p>	<p>ALCOHOL (excess alcohol - >8u/day males or >6u/day females) [] n/a</p> <p>Refer to Alcohol liaison 0753565839 []</p> <p>Prescribe IV Pabrinex – 2 Pairs of vials TDS []</p> <p>If withdrawing – refer to UHL alcohol withdrawal guidelines []</p>	<p>OTHER ENCEPHALOPATHY [] n/a</p> <ul style="list-style-type: none"> - Look for precipitants (GI Bleed, constipation, dehydration, sepsis, etc) [] - Lactulose 20-30ml QDS or Phosphate enema (aim is 2 soft motions/day) [] - Consider CT Head if clinically appropriate [] <p>VTE PROPHYLAXIS []</p> <p>GASTRO REVIEW []</p>
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Consider escalation of care (if appropriate) if physiological markers deteriorating/ not improving in spite of above interventions

Guidelines on the Assessment and Management of Decompensated Cirrhosis– The First 24 hours

APPENDIX 2

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