

CONTROLLED DOCUMENT

Cirrhosis Care Bundle

CATEGORY:	Clinical Guidelines
CLASSIFICATION:	Clinical
Controlled Document Number:	CG201
Version Number:	1
Controlled Document Sponsor:	Clinical Guidelines Group
Controlled Document Lead (Author):	Consultant Hepatologist
Approved By:	Clinical Guidelines Group
On:	August 2015
Review Date:	August 2018

CIRRHOSIS CARE BUNDLE

<u>CONTENTS</u>	<u>PAGE</u>
INTRODUCTION	3
SCOPE	3
BASELINE INVESTIGATIOS	4
ALCOHOL	4
INFECTION	4
ACUTE KIDNEY INJUSY AND / OR HYPONATRAEMIA	5
GI BLEEDING	5
ENCEPHALOPATHY	6
REVIEW	6
FURTHER READING	6
CIRRHOSIS CARE BUNDLE	7

INTRODUCTION

This guideline is a synopsis of the joint British Society of Gastroenterology and British Association for the Study of the Liver paper on the introduction of a national Cirrhosis Care Bundle. For all supporting papers justifying the statements made in this Guideline please see the full paper in 'Further Reading'. Over the last 20 years, there has been a significant increase in the prevalence of chronic liver disease in the UK, with the major causes being alcohol-related liver disease (ARLD), hepatitis B and C and obesity-related liver disease. There has also been a substantial rise in hospital admissions with complications of liver disease, as well as a steady rise in liver related deaths in the UK. Liver disease is now one of the major causes of premature death in the UK. Decompensated cirrhosis and acute on chronic liver failure are common causes for hospital admission and are associated with a high mortality rate. The 2013 National Confidential Enquiry into Patient Outcome and Death (NCEPOD) of patients with ARLD 'Measuring the Units' raised concerns about suboptimal care of patients in hospital with complications of cirrhosis (due to ARLD), which might have contributed to the rise in liver-related mortality. The report found that less than half (47%) the patients who died from ARLD received 'good care', and avoidable deaths were identified. It is likely that similar findings would be observed for the management of patients with cirrhosis of other aetiologies. In order to improve the care of patients admitted with ARLD, the NCEPOD report recommended that a 'toolkit' for the acute management of patients admitted with decompensated cirrhosis be developed and made widely available. Such a toolkit has been developed by the British Society of Gastroenterology and British Association for the Study of the liver in response to the NCEPOD report and this Guideline introduces its use at University Hospital Birmingham Foundation NHS Trust. The toolkit is applicable to the initial management of all patients with features of decompensated cirrhosis of any aetiology, not just ARLD.

SCOPE

The care bundle is designed to be completed for all patients presenting with decompensated cirrhosis and should be commenced within 6 h of admission. 'Decompensated cirrhosis' is defined as an acute deterioration in liver function in a patient with cirrhosis that can manifest with the following: jaundice, increasing ascites, hepatic encephalopathy, renal impairment / hypovolaemia, gastrointestinal (GI) bleeding, or signs of sepsis. This care bundle is primarily designed to provide recommendations on management for the first 24 h only. After this period, it is expected that specialist GI/liver input will be available to provide guidance on further management. . The Decompensated Cirrhosis Care Bundle should be completed in all patients presenting to UHB with decompensated cirrhosis, and added to the admission documentation, The Decompensated Cirrhosis Care Bundle form is designed to act as a prompt to appropriate investigations and management in the first 24 hours after admission. This form is available as part of this Guideline. Copies will also be available in the Clinical Decisions Unit .and addresses the following areas:

BASELINE INVESTIGATIONS

On admission, it is vital that patients presenting with decompensated cirrhosis have a full history and clinical examination to look for the cause of deterioration in liver function, infection or evidence of GI bleeding. A Standardised Early Warning Score (SEWS) should be recorded and used to monitor the patient's physiological status. Blood tests should be taken to assess liver and renal function, and this should include: full blood count, urea and electrolytes (including calcium/phosphate/magnesium), liver function tests, coagulation profile and glucose. As sepsis is a frequent cause of admission, patients should also be screened for infection with clinical examination, urinalysis and urine culture, chest X-ray, blood cultures and C-reactive protein (CRP).

Additionally, an ascitic tap should be performed for all patients who have clinically detectable ascites to exclude spontaneous bacterial peritonitis (SBP). Coagulopathy is not a contraindication to this procedure and measures to correct coagulopathy are NOT required. Ascitic fluid should be sent to the lab for analysis of the fluid: polymorphonuclear (PMN) and white cell count, microscopy and culture, protein and albumin content.¹¹ An abdominal ultrasound should be requested and performed at the earliest opportunity (unless an ultrasound has been conducted in the last month), ideally within 24 h, and should include an assessment of portal vein patency.

ALCOHOL

70% of all hospital admissions have alcohol as the major aetiological factor. An alcohol history should be taken and patients should be management in accordance with the Trust Guideline on Alcohol Withdrawal.

INFECTION

Patients with cirrhosis have impaired defence against bacteria due to immune dysfunction, and as a result, bacterial infections are one of the most frequent reasons for admission and are associated with a high mortality rate. A careful assessment for infection is critical in all patients admitted with decompensated cirrhosis, as prompt treatment with antibiotics improves prognosis. Importantly, patients with cirrhosis do not always display typical signs of infection, such as pyrexia or rise in CRP, so clinicians must have a high index of suspicion for infection.

SBP is defined as infection of the ascitic fluid in the absence of a secondary cause, such as intestinal perforation, and occurs in approximately 10% of hospitalised patients with cirrhosis. Although abdominal pain and fever are commonly seen in patients with SBP, symptoms are frequently absent. It is, therefore, recommended that all patients presenting with ascites have a diagnostic ascitic tap to exclude SBP on admission to hospital, or if there is deterioration in their clinical status. Upon diagnosis, SBP should be empirically treated with a 3rd generation cephalosporin such as Cefotaxime 2G daily and modifications made in light of culture results.

ACUTE KIDNEY INJURY AND/OR HYPONATRAEMIA

Acute Kidney Injury (AKI) in patients with cirrhosis is commonly multifactorial, but prerenal AKI is most common (45%), followed by acute tubular necrosis and glomerulonephritis (32%), Hepatorenal Syndrome (23%) and rarely postrenal (<1%). It is important to identify the cause of AKI, as the underlying cause has implications on prognosis. Type 1 HRS (acute) carries a particularly poor prognosis with mortality rates approaching 100% without treatment. Diagnostic criteria for HRS are in Box 1.

Box 1: Diagnostic Criteria for Hepatorenal Syndrome

- ▶ Cirrhosis with ascites
- ▶ Serum creatinine >133 $\mu\text{mol/L}$
- ▶ No improvement of serum creatinine to <133 $\mu\text{mol/L}$ after at least 2 days with diuretic withdrawal and volume expansion with albumin (1 g/kg up to a maximum of 100 g/day)
- ▶ Absence of shock
- ▶ No current treatment with nephrotoxic drugs
- ▶ Absence of parenchymal kidney disease as indicated by proteinuria (>500 mg/day) or microhaematuria (>50 red blood cells per high-power field) and/or a normal renal ultrasonography

Hyponatraemia is common in patients with decompensated cirrhosis and can be caused by multiple factors including: hormonal dysregulation of salt and water balance in response to portal hypertension, dehydration, treatment with diuretics, infections and excessive use of hypotonic fluids, such as 5% dextrose. Patients with serum sodium levels <125 mmol/L are at risk of seizures. A careful history, clinical examination, medication review and review of other biochemistry are essential to determine the cause of hyponatraemia. Assessment of the patient's volume status is fundamental, and subjects who are hypovolaemic should be fluid-resuscitated. Patients with hypervolaemic hyponatraemia should be treated with fluid restriction. However, it should be noted that some patients with significant ascites, peripheral oedema and low serum albumin levels who appear hypervolaemic are actually intravascularly deplete, and treatment with intravenous albumin can correct the hyponatraemia.

AKI, Hyponatraemia and HRS should be managed in accordance with the Trust Renal Guidelines in Cirrhosis.

GI BLEEDING

Bleeding oesophageal and gastric varices are another important complication of cirrhosis with a high mortality rate. A UK-wide audit showed the overall 30-days mortality was 15% for patients with acute variceal bleeding, with higher mortality rates in subjects with more advanced cirrhosis. In patients with known varices or portal hypertension, upper GI bleeding should be regarded as variceal until proven otherwise, although a significant proportion will have non-variceal bleeding. The management should be in accordance with Trust guideline on Management of Gastro-Oesophageal Varices.

ENCEPHALOPATHY

Encephalopathy is another common complication in patients with cirrhosis.⁴⁶ A careful assessment of the cause of encephalopathy should be undertaken looking for causes such as infection, electrolyte disturbance, occult bleeding, constipation or sedative drugs. For patients with grade 4 encephalopathy (coma) consider orotracheal intubation to reduce the risk of developing aspiration pneumonia. In conscious patients, oral lactulose (20 mL–30 mL four times per day) should be administered aiming for 2 soft stools per day. For patients with a reduced conscious level, phosphate enemas or lactulose administered by nasogastric tube should be considered. Consider a CT head examination in patients where the confusion is unexplained to exclude intracranial pathology, such as subdural haematoma.

REVIEW

Patients with decompensated cirrhosis should be reviewed by a consultant at the earliest opportunity as they typically have multiple medical needs and have a high mortality. Additionally, these patients should be reviewed by a specialist (gastroenterologist or hepatologist), ideally within 24 h, but not more than 72 h after admission to hospital. Escalation to a higher level of care needs to be considered in all patients not responding to treatment when reviewed after 6 h, particularly in patients with WHO performance status 0–1 prior to the recent illness and those with first presentation.

FURTHER READING

1. McPherson S, Dyson J, Austin A, et al. Frontline Gastroenterology Published Online First: 28 Sept 14. doi:10.1136/flgastro-2014-100491

Decompensated Cirrhosis Care Bundle - First 24 Hours

Decompensated cirrhosis is a medical emergency with a high mortality. Effective early interventions can save lives and reduce hospital stay. This checklist should be completed for all patients admitted with decompensated cirrhosis within the first 6 hours of admission.

1. Investigations							
a)	NEWS <input type="checkbox"/>	FBC <input type="checkbox"/>	U/E <input type="checkbox"/>	LFT <input type="checkbox"/>	Coag <input type="checkbox"/>	Gluc <input type="checkbox"/>	Ca/PO ₄ /Mg <input type="checkbox"/>
b)	Blood cultures <input type="checkbox"/>			Urine Dip/MSU <input type="checkbox"/>	CXR <input type="checkbox"/>	Request USS abdo <input type="checkbox"/>	CRP <input type="checkbox"/>
c)	Perform ascitic tap in all patients with ascites using green needle irrespective of clotting parameters and send for ascitic PMN/WCC, culture and fluid albumin						Done <input type="checkbox"/> Y N <input type="checkbox"/>
d)	Record recent daily alcohol intake		 Units			
2. Alcohol - if the patient has a history of current excess alcohol consumption (>8 units/day Males or >6 units/day Females) N/A <input type="checkbox"/>							
a)	Give IV Pabrinex (2 pairs of vials three times daily)					Y N	
b)	Commence CIWA score if evidence of alcohol withdrawal					Y N N/A	
3. Infections - if sepsis or infection is suspected N/A <input type="checkbox"/>							
a)	What was the suspected source?.....						
b)	Treat with antibiotics in accordance with Trust protocol					Y N	
c)	If the ascitic neutrophils >0.25 x 10 ⁹ /L (>250/mm ³)(i.e. SBP) then give:					Y N	
i)	Treat with antibiotics as per trust protocol					Y N NA	
ii)	IV albumin (20% Human Albumin solution) 1.5g/kg (20g of albumin in 100ml of 20% Human Albumin Solution)					Y N NA	
4. Acute kidney injury and/or hyponatraemia (Na <125 mmol/L) N/A <input type="checkbox"/>							
AKI defined by modified RIFLE criteria		1: Increase in serum creatinine ≥ 26μmol/L within 48hrs 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 hrs based on dry weight or					
		4: Clinically dehydrated					
a)	Suspend all diuretics and nephrotoxic drugs					Y N NA	
b)	Fluid resuscitate with 5% Human Albumin Solution or 0.9% Sodium Chloride (250ml boluses with regular reassessment: 1-2L will correct most losses)					Y N	
c)	Initiate fluid balance chart/daily weights					Y N	
d)	Aim for Mean Arterial Pressure >80mmHg to achieve UO>0.5ml/kg/hr					Y N	
e)	At 6 hrs, if target not achieved or EWS worsening then consider escalation to higher level of care					Y N NA	
5. GI bleeding – if the patient has evidence of GI bleeding and varices are suspected N/A <input type="checkbox"/>							
a)	Fluid resuscitate according to BP, pulse and venous pressure (aim MAP >65 mmHg)					Y N	
b)	Prescribe IV terlipressin 2mg four times daily (caution if known ischaemic heart disease or peripheral vascular disease; perform ECG in >65yrs)					Y N NA	
c)	Prescribe prophylactic antibiotics as per Trust protocol (cefuroxime unless contraindicated)					Y N	
d)	If prothrombin time (PT) prolonged give IV vitamin K 10mg stat					Y N NA	
e)	If PT> 20 seconds (or INR >2.0) – give FFP (2-4 units)					Y N NA	
f)	If platelets <50 – give IV platelets					Y N NA	
g)	Transfuse blood if Hb <7.0g/L or massive bleeding (aim for Hb >8g/L)					Y N NA	
h)	Early endoscopy after resuscitation (ideally within 12 hours)					Y N	

Initials:

Time:

Initials:

Time:

Initials:

Time:

Initials:

Time:

Initials:

Time:

Please place in medical notes - Continues overleaf..→

6. Encephalopathy		N/A <input type="checkbox"/>
a)	Look for precipitant (GI bleed, constipation, dehydration, sepsis etc.)	Y N
b)	Encephalopathy – lactulose 20-30ml QDS or phosphate enema (aiming for 2 soft stools/day)	Y N
c)	If in clinical doubt in a confused patient request CT head to exclude subdural haematoma	Y N N/A
7. Other		
a)	Venous thromboembolism prophylaxis – prescribe prophylactic LMWH (patients with liver disease are at a high risk of thromboembolism even with a prolonged prothrombin time; withhold if patient is actively bleeding or platelets <50)	Y N NA
b)	GI/Liver review at earliest opportunity (ideally within 24 hrs)	<input type="checkbox"/>

Initials:

Time:

Initials:

Time:

Name.....Grade.....Date.....Time.....

Decompensated Cirrhosis Care Bundle - First 24 Hours

The recent NCEPOD report 2013 on alcohol related liver disease highlighted that the management of some patients admitted with decompensated cirrhosis in the UK was suboptimal. Admission with decompensated cirrhosis is a common medical presentation and carries a high mortality (10-20% in hospital mortality). Early intervention with evidence-based treatments for patients with the complications of cirrhosis can save lives. This checklist aims to provide a guide to help ensure that the necessary early investigations are completed in a timely manner and appropriate treatments are given at the earliest opportunity.

- Decompensated cirrhosis is defined as a patient with cirrhosis who presents with an acute deterioration in liver function that can manifest with the following symptoms:
 - Jaundice
 - Increasing ascites
 - Hepatic encephalopathy
 - Renal impairment
 - GI bleeding
 - Signs of sepsis/hypovolaemia
- Frequently there is a precipitant that leads to the decompensation of cirrhosis. Common causes are:
 - GI bleeding (variceal and non-variceal)
 - Infection/sepsis (spontaneous bacterial peritonitis, urine, chest, cholangitis etc)
 - Alcoholic hepatitis
 - Acute portal vein thrombosis
 - Development of hepatocellular carcinoma
 - Drugs (Alcohol, opiates, NSAIDs etc)
 - Ischaemic liver injury (sepsis or hypotension)
 - Dehydration
 - Constipation

When assessing patients who present with decompensated cirrhosis please look for the precipitating causes and treat accordingly. The checklist shown overleaf gives a guide on the necessary investigations and early management of these patients admitted with decompensated cirrhosis and should be completed on all patients who present with this condition. The checklist is designed to optimize a patient's management in the first 24 hours when specialist liver/gastro input might not be available. Please arrange for a review of the patient by the gastro/liver team at the earliest opportunity. Escalation of care to higher level should be considered in patients not responding to treatment when reviewed after 6 hours, particularly in those with first presentation and those with good underlying performance status prior to the recent illness.