# Liver disease

## *Executive summary*

## Introduction

The liver is responsible for many critical functions within the body and should it become diseased or injured, the loss of those functions can cause significant damage to the body. Liver disease is a broad term that covers all the potential problems that cause the liver to fail to perform its designated functions. Usually, more than 75% or three quarters of liver tissue needs to be affected before a decrease in function occurs.

Management of these patients is divided into the assessment and treatment of patients with a new diagnosis of liver disease and that of those with established chronic liver disease (CLD) who have decompensated.

## Target User

* Doctors
* Nurses

## Target area of use

* Ward

## Key areas of focus/new additions/changes

This guideline outlines the initial diagnosis of liver disease in our setting as well as the management of patients with established disease who decompensate.

## Limitations

We have limited access to most of the diagnostic tests for causes of liver disease.

Access to higher level care is largely not available – except for dialysis, which might be possible at the teaching hospital.

## Presenting symptoms and signs

If the patient is in extremis, they should be resuscitated prior to any other intervention – the most likely causes are an acute GI bleed or bacterial septicaemia.

Patients with liver disease can present with many different symptoms including:

* Weakness and fatigue
* Jaundice
* Nausea
* Vomiting
* Weight loss
* Right upper quadrant pain
* Confusion
* Reduced GCS
* Clubbing
* Hematemesis.

They may present with signs of anaemia, sepsis, acute liver disease or chronic liver disease.

The patient’s background history must be taken in order to establish the cause of the underlying liver disease. This may affect the treatment given.

The history of the current event must also be taken with particular note of any gastrointestinal bleeding, change in girth size, pain, confusion, difficulty breathing, increase in jaundice, recent medications and traditional treatments taken.

## Examination findings

* General findings: anaemia, fever, tachycardia, altered level of consciousness or confusion, peripheral oedema.
* Signs of chronic liver disease: liver flap, clubbing, palmar erythema, bruising, spider naevi, gynaecomastia, testicular atrophy, ascites, venous collaterals.
* Liver: small in cirrhosis, large in acute hepatitis or neoplasm.
* Splenomegaly: suggests portal hypertension.
* PR: this is essential if there is any suggestion of a GI bleed.
* Signs of excess alcohol – Dupuytren’s contracture – parotid enlargement.
* Stigmata of HIV: Oral thrush, Herpes Zoster scar.

## Investigations

Tests which may be appropriate to assess patient’s current condition include:

* FBC, LFTs, albumin, INR, U&Es, blood glucose
* Urinary dipstick
* Blood cultures and chest X-ray
* Ascitic tap (if ascites is present this is essential) – send for MC&S, albumin, AFBs.

Note that FBC, INR, U&Es, LFTs and ascitic tap microscopy and albumin are all urgent tests and ***the results should be reviewed within 4 hours of admission*** to the ward in any acutely unwell patient.

Tests to identify possible causes include:

* Viral serology: HIV, HBsAg (HCV, HAV when available)
* Hb genotype
* Ultrasound – for jaundiced patients ask directly about duct dilatation.
* Consider autoantibodies, ferritin and AFP when available.

## Causes of decompensation in CLD

For patients with chronic liver disease, management will depend on the cause of the decompensation. Patients with CLD may decompensate because of a complication of their underlying liver disease or because of a second insult to the liver or their overall system.

Complications of CLD are:

* Variceal haemorrhage
* Portal vein thrombosis
* Ascites
* Spontaneous bacterial peritonitis
* Hepatic encephalopathy
* Hepatorenal syndrome
* Hepatopulmonary syndrome
* Hepatocellular carcinoma

Common causes of a second insult are:

* Acute viral hepatitis
* Drug-induced liver injury (which may be caused by traditional treatment as well as prescribed medications)
* Bacterial infection (UTI, LRI etc)

## Management

### Newly diagnosed liver disease

Treat precipitants and possible causes.

Ensure adequate hydration and nutrition (give e-pap if there is any doubt).

Avoid hepatotoxic drugs.

Manage according to particular complications (hepatic encephalopathy is common).

Seek specialist support if acute liver disease is suspected.

### All patients with CLD

All patients admitted with CLD should have their Child-Pugh score calculated in order to provide prognostic information.

Use the table below to assign a score for each item:

|  |  |  |  |
| --- | --- | --- | --- |
| **Points** | **1** | **2** | **3** |
| Ascites | None | Slight | Moderate or above |
| Bilirubin | <34.2 | 34.2-51.3 | >51.3 |
| Serum albumin | >35 | 28-35 | <28 |
| INR | <1.7 | 1.7-2.3 | >2.3 |
| Encephalopathy | None | Grade 1-2 | Grade 3-4 |

Add the score and interpret the findings according to this table:

|  |  |  |  |
| --- | --- | --- | --- |
| **Class** | **Score** | **1 year survival rate** | **2 year survival rate** |
| A | 5-6 | 100% | 85% |
| B | 7-9 | 80% | 60% |
| C | 10-15 | 45% | 35% |

In the rare cases where the patient can control a cause of deterioration (especially alcoholic liver disease), if they remove this risk factor (i.e. stop drinking) they can improve their prognosis significantly.

If the INR is greater than 3.5, then vitamin K should be prescribed. In most cases, 10 mg OD orally for 3 days is adequate.

### Variceal haemorrhage

This should be suspected whenever there is GI bleeding. It may present with either haematemesis or melaena.

Other causes of GI haemorrhage are possible, but are less serious. Only 50% of bleeding varices will stop bleeding spontaneously. The outlook is even worse for patients with Child-Pugh Class C disease.

Re-bleeding is also very common and is more likely for older patients, those with encephalopathy, ascites, renal failure, alcoholic liver disease or low platelets.

Management begins with urgent fluid resuscitation including the siting of 2 large bore cannulas, iv normal saline to restore BP and urgent cross-matching of blood. Whole blood should be transfused as soon as possible aiming for an Hb of 9 g/dl.

Endoscopy should be arranged if available.

* Unstable patients with severe acute upper GI bleeding should be scoped as soon as possible after resuscitation.
* Stable patients with upper GI bleeding should be scoped within the next working day after admission.
* Any varices should be banded. If another cause of bleeding is identified, management should be altered to reflect this.

None of the vasoactive medications shown to be effective in controlling acute bleeding are available here.

Patients are at high risk of concomitant infection and the presence of infection increases the risk of re-bleeding so all patients should be given prophylactic antibiotics – usually ciprofloxacin 500 mg BD for 7 days.

Patients are at high risk of encephalopathy and should be treated with lactulose if they are not passing stool at least twice each day – 20 ml TDS to start.

*Secondary prophylaxis*

This should be offered to all patients who have had a variceal bleed.

Both non-cardioselective beta blockers and variceal band ligation should be given.

Give propranolol 40 mg BD and titrate up the dose until the HR is 50-55 bpm. The maximum dose is 320 mg per day. This should also be given to patients identified as having varices who have not yet had a bleed (primary prophylaxis).

### Spontaneous bacterial peritonitis (SBP)

The signs of SBP can be very subtle and therefore it should be considered a possibility in all patients with ascites due to portal hypertension.

Ascitic fluid should be aspirated and sent for immediate fluid albumin and microscopy including cell count. If plenty of fluid is obtained – some can be used to inoculate blood culture bottles.

SBP is considered to be present if the granulocyte count is greater than 250 cells/mm3. It should be suspected if there are typical clinical signs such as Temp > 37.8ºC, abdominal pain or tenderness or change in mental status.

As soon as the samples are obtained, if there are clinical signs of SBP, then treatment should be started. iv ceftriaxone 1 g OD or oral ciprofloxacin 500 mg BD are both appropriate empirical options. Most patients will recover with a 5 day course of treatment.

If the patient is on propranolol, this should be stopped permanently.

When the patient has recovered, they should be placed on antibiotic prophylaxis. The best choice is Septrin 960 mg OD. Ciprofloxacin 500 mg OD is an alternative.

Hepatic encephalopathy

This is a common complication which can be difficult to diagnose in its subtler forms. It is graded according to the following table:

|  |  |
| --- | --- |
| Grade 1 | Changes in behaviour, mild confusion, slurred speech, disordered sleep |
| Grade 2 | Lethargy, moderate confusion |
| Grade 3 | Marked confusion, incoherent speech, sleeping but rousable |
| Grade 4 | Coma |

It may be precipitated by GI bleeding, infection (including SBP), low K+, renal failure, hypovolaemia, hypoxia, sedatives, hypoglycaemia, constipation.

Patients should be treated with lactulose, beginning with a dose of 20 ml TDS. The dose should be increased until they pass stools at least twice each day.

Any potential triggers of encephalopathy should be corrected.

### Hepatorenal syndrome

This is the cause of about 15% of cases of renal failure in chronic liver disease. It refers to renal failure arising from falling renal perfusion as a direct effect of liver dysfunction. It has a very poor prognosis and there is no treatment available for it in this setting. Other causes of renal failure must therefore be excluded or treated before this diagnosis is made.

Consider alternative diagnoses of renal failure such as hypovolaemia, sepsis, glomerulonephritis, or obstruction.

* Dip the urine – if there are white cells or red cells, consider glomerulonephritis or a urinary infection with septic shock.
* Stop any diuretics.
* Give 1 litre of normal saline over 1-2 hours and monitor urinary output and observations. If the renal failure is caused by hypovolaemia, you should see an increase in urine output and improving observations.
* Treat any infection.
* Arrange for a renal and bladder ultrasound to exclude obstruction.

Any patient who has no other diagnosis identified and who has not had an improvement in their renal function with these interventions may have hepatorenal syndrome. The diagnosis can be confirmed if the creatinine rises by 25 or more within 48 hours or if there is a 50% rise from baseline within 7 days. The treatment is supportive. A small number will recover if they can be supported until their liver function improves.

### Hepatopulmonary syndrome

Diagnosed when a patient with CLD becomes hypoxic and is found to have a hyperdynamic circulation. They may become hypoxic only on sitting upright.

Treat with oxygen and exclude other causes of hypoxia. There is no specific treatment and the prognosis is very poor.

### Ascites

Ascites is a very common complication of chronic liver disease. It is usually diagnosed clinically and confirmed by abdominal ultrasound.

All CLD patients presenting with ascites should have an immediate ascitic tap with samples sent for albumin, cell count and culture.

Calculate the serum ascites albumin gradient (SAAG):

SAAG = serum albumin – ascites albumin

If SAAG is greater than 11, the ascites is caused by portal hypertension – usually because of liver disease, but could be due to heart failure or Budd-Chiari syndrome. If SAAG is less than 11, an alternative diagnosis must be sought.

If the cell count shows > 250 granulocytes/mm3, then the patient has SBP and should be treated accordingly.

Ascites that is due to portal hypertension in the context of CLD should be treated with salt restriction and diuretics. Start with 40 mg furosemide OD and 100 mg spironolactone OD. If there is no response after 3-5 days, the doses can be increased in the same ratio up to a maximum of 160 mg furosemide and 400 mg spironolactone. A patient with ascites and oedema may lose up to 2 kg/day whilst a patient with only ascites typically loses no more than 0.75 kg/day.

If the ascites is tense or the ascites needs to be removed more quickly, large volume paracentesis can be undertaken. Up to 5 litres can be removed each day without replacement with albumin or other fluids.

***Patients who develop ascites should not continue treatment with propranolol or an ACE inhibitor.***

### Hepatocellular carcinoma

In our setting, many patients do not present with CLD until they already have a large hepatocellular carcinoma. There is little that can be done to treat them apart from symptom control.

Patients with CLD who present with a sudden deterioration in liver function may have developed a HCC and should be sent for ultrasound. If a new HCC is present, they should be referred to the liver clinic for assessment once they have recovered from their acute decompensation.

Patients should be treated with analgesia according to the WHO analgesic ladder if they are in pain. Paracetamol is safe in this setting. NSAIDs should be used with caution, but are the most effective option for pain due to stretching of the liver capsule. Codeine and morphine may trigger hepatic encephalopathy, but should be used if the patient’s pain is severe and unresponsive to other options.

## Key Issues for Nursing care

* Refer all patients with suspected liver disease or complications to a doctor.
* Assess need for pain control.
* Check on frequency of bowel movements and if less than or greater than two stools / day advise doctor to adjust lactulose dose accordingly.
* Ask patient if they have melaena stools.
* Document presence of confusion and or changes in behaviour which may be signs of evolving hepatic encephalopathy.
* Order a low salt diet in patients with ascites.
* Ensure adequate nutrition (with epap supplements if necessary) for all patients with liver disease.

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| **Written by:** | Name: Amie Secka | Date: 6 June 2018 |
| **Reviewed by:** | Name: Karen Forrest | Date: 14 June 2018 |
| **Version:** | **Change history:** | **Review due date:** |
| 1.0 | New document |  |
| 2.0 | Adapted from previous guideline on CLD and format updated. | 31 July 2020 |
| 2.1 | Executive summary added | 31 July 2020 |
| Review Comments (*if applicable)* |  |  |