



**Clinical Cases  
for AMU –  
Case Six:  
Haematemesis**

## **Introduction**

These cases are designed to support your learning during your time in Acute and General Medicine. You can use them when you have free time on the ward. They can be done either alone, or in a small group. They use fictional scenarios to demonstrate learning points from common presentations to the Acute Medical Unit (AMU) and on the General Medical wards. As you work through the cases, you will find a mixture of case discussions, practical activities, and practice questions to assess your learning.

If there is a knowledge check or interpretation exercise, the answer can be found on the back of the same page that the question is on.

## **Case History**

Tracy is a 57-year-old woman who has been referred to the Medical Assessment Unit by her GP after she had an episode of vomiting blood. Tracy describes three episodes of vomiting fresh red blood over the course of yesterday evening into this morning. She thinks there has been a cupful of blood on each occasion. She continues to feel nauseated. She describes passing black, tarry stool for the last 48 hours. She feels generally unwell and has noticed that she is dizzy when she stands up. Tracy describes some epigastric discomfort.

Tracy's past medical history includes osteoarthritis for which she takes regular paracetamol and ibuprofen.

Tracy is a non-smoker. She drinks a bottle of wine every night with additional spirits at the weekend. She works as a civil servant.

Her observations can be seen on the attached chart. They are marked observations one.

On examination, Tracy looks unwell. She is cool peripherally with a capillary refill time of 4 to 5 seconds. Her sclerae look jaundiced. 5 spider naevi can be seen on her upper chest and neck. Her heart sounds are normal, with no additional sounds. Her chest is clear. Her abdomen appears swollen, with evidence of caput medusae. Shifting dullness is present. You are unable to feel the liver. There is bilateral lower limb oedema at the ankles.

You suspect an upper GI bleed.

[illegible]

## Knowledge Check One

1. Based on the clinical information available, what other diagnosis would you be suspicious of?
2. What risk factors for upper GI bleeding does Tracy have?
3. What immediate actions would you take in Tracy's case?

Knowledge Check One - Answers

1. Tracy's history of alcohol excess, and several clinical signs, are suggestive of alcoholic liver disease with cirrhosis. Abdominal swelling and shifting dullness are examination signs of ascites. The caput medusae suggests portal hypertension. Other signs of liver disease include spider naevi, palmar erythema, and jaundice.
2. Alcohol excess, ibuprofen use, and alcoholic liver disease are all risk factors for upper GI bleeding.
3. Initial steps for a haemodynamically stable patient in whom you are suspicious of a GI bleed include:
  - a. Urgent bloods including coagulation screen and group and save
  - b. Insertion of 2 wide bore cannula
  - c. Fast the patient until it is clearer when they will have an upper GI endoscopy

## Results

Test	Result	Normal range
Hb (g/L)	63	130 - 180
WCC ( $10^9/L$ )	5.2	4.0 - 11.0
Plt ( $10^9/L$ )	84	150 - 400
Na (mmol/L)	130	135 - 145
K (mmol/L)	5.4	3.6 - 5.0
Urea (mmol/L)	13.9	2.5 - 6.6
Creatinine (umol/L)	86	64 - 111
eGFR (ml/min)	>60	>60
Bil (umol/L)	81	3 - 21
ALT (U/L)	96	10 - 50
AST (U/L)	210	8 - 33
ALP (U/L)	108	40 - 125
GGT (U/L)	385	1 - 35
Albumin (g/L)	23	36 - 47
PT (sec)	14.1	9.0 – 12.0
APTT (sec)	31	21.0 – 28.0
CRP (mg/L)	<1	0 - 5

## Interpretation One

1. There are multiple abnormalities in these blood results. Which two changes are most supportive of an acute GI bleed?
2. The Glasgow-Blatchford Score (GBS) is used to differentiate between high risk and low risk upper GI bleeds. Patients with scores of 0 or 1 are suitable for outpatient management. Search the Glasgow-Blatchford score online and use the information available to calculate the score for Tracy.
3. Which of these results would support a diagnosis of alcoholic liver disease with cirrhosis?
4. What other blood tests might you consider in a patient with new derangement of their liver function tests?

Interpretation One - Answers

1. The drop in haemoglobin with an associated rise in urea suggest a GI bleed. In patients with upper GI bleeding, a rise in urea is expected. This is because blood is digested to protein in the GI tract, and the protein is metabolised to urea by the liver.
2. Tracy's Glasgow-Blatchford Score is 13. This score indicates that she is having a high risk GI bleed, and requires an inpatient endoscopy.
3. There are several blood results which support a diagnosis of alcoholic liver disease:
  - a. Coagulation disturbances with PT prolongation occur due to impairment of the liver's synthetic function in cirrhosis. The cirrhotic liver is unable to produce the clotting factor required for a normal coagulation cascade, resulting in PT prolongation
  - b. Similarly low albumin occurs due to impairment of the synthetic function of the liver
  - c. Hyponatraemia in cirrhosis is common. In cirrhosis, the ability of the kidney to excrete solute-free water is impaired, resulting in water and sodium retention. Proportionally, more water is retained than salt, resulting in dilution of the blood and hyponatraemia. This is hypervolaemic hyponatraemia.
  - d. Thrombocytopenia is a common finding in cirrhosis. This is multifactorial with reasons including reduced production, sequestration of platelets to the spleen, and increased destruction.
  - e. LFT derangement with raised ALT, AST and bilirubin also support the diagnosis. GGT is often raised in the context of recent alcohol use. The AST:ALT ratio can be a useful indication of the aetiology of liver disease. An AST:ALT ratio of  $>1$  is suggestive of alcoholic liver disease.
4. In patients with a new hepatitis on their blood tests, we often perform a 'liver screen', which is a set of blood tests that assesses for causes of hepatitis. This includes viral hepatitis serology, autoantibodies for autoimmune liver diseases, ferritin and iron studies (assessing for haemochromatosis).

**Case continued...**

While you are writing up Tracy's notes, the emergency buzzer goes off, and you hear the nurse looking after Tracy call for help. You are the first to attend.

Activity One

You perform an A to E assessment on Tracy. Using an A to E assessment, document what steps you would take in the immediate assessment and management of a patient who is bleeding. Compare this with the A to E assessment on the back of this page.

<b>A</b>	
<b>B</b>	
<b>C</b>	
<b>D</b>	
<b>E</b>	

Activity One - Answers

<b>A</b>	<p>Assess airway for signs of obstruction. If patient is speaking normally, then no airway obstruction present.</p>
<b>B</b>	<p>Check respiratory rate and oxygen saturations. Oxygen if saturations are &lt;94%. Examine chest. If evidence of respiratory compromise, perform ABG and organise CXR. In Tracy's case, there is no evidence of respiratory disease, so ABG and CXR would not be essential parts of her initial management. For the purpose of the case, I have included both for practice.</p>
<b>C</b>	<p>Check pulse and blood pressure. Examine for externalisation of blood (e.g., is there any further melaena/haematemesis). Ensure two wide bore cannulas inserted, and emergency bloods have been sent. Send VBG as an urgent point of care test to find current haemoglobin and assess for acidosis. Bolus of fast IV fluid and arrange urgent blood transfusion. Contact relevant team who will be required to achieve haemostasis in bleeding patient. Determine whether there is any coagulation disorder that needs reversed, or whether the patient is taking anticoagulants. ECG</p>
<b>D</b>	<p>Assess conscious level Check glucose</p>
<b>E</b>	<p>Check temperature Examine abdomen</p>



## Assessment

The outcome of your assessment can be seen below.

<b>A</b>	Patient is talking, with no evidence of airway obstruction
<b>B</b>	<p>SpO<sub>2</sub> 96% on room air, RR 26 Chest clear CXR clear ABG on air</p> <ul style="list-style-type: none"> <li>• H<sup>+</sup> 43 (35 – 45 nmol/L)</li> <li>• pO<sub>2</sub> 12.1 (11.1 – 14.4 kPa)</li> <li>• pCO<sub>2</sub> 4.7 (4.7 – 6.4 kPa)</li> <li>• HCO<sub>3</sub><sup>-</sup> 22 (22 – 28 mmol/L)</li> <li>• Base excess -2 (-2 – 3 mmol/L)</li> <li>• Hb 55 (130 – 180 g/l)</li> <li>• Lactate 4.1 (0.5 – 1.6 mmol/L)</li> </ul>
<b>C</b>	<p>HR 130, BP 72/40 Cool peripherally, CRT 5s HS pure 2 x large bore cannulas already in place. Repeat bloods sent including VBG for immediate result. Further bolus of IV fluid given Tracy has just had a further large volume haematemesis, with 500mls of fresh red blood in bowl ECG – sinus tachycardia</p>
<b>D</b>	Alert, though a little agitated
<b>E</b>	<p>Epigastric tenderness Swollen abdomen with evidence of ascites BS present Nurses show you melaena in bedpan</p>

### Knowledge Check Two

1. What kind of shock is Tracy suffering from?
2. Tracy has evidence of active GI bleeding. Her blood sample is currently at the transfusion lab being crossmatched, but it is not yet ready. Should you:
  - a. Activate the major haemorrhage protocol to arrange an urgent O negative blood transfusion.
  - b. Wait until the crossmatch is complete so that Tracy can be given a transfusion which is more specific to her blood type and give fast intravenous fluid in the meantime.
3. What other clinical teams would you want to be involved in Tracy's care at this stage? Why?
4. If Tracy's blood group was B positive, what blood groups would be compatible with her for red cell transfusion?
5. Identify some potential complications of blood transfusion.

### **Case continued...**

Following transfusion, Tracy is transferred to theatre as an emergency for an endoscopy. Here, a bleeding oesophageal varix is identified. The bleeding varix is successfully banded to achieve haemostasis. In line with guidelines on the management of bleeding oesophageal varices, Tracy is started on IV terlipressin and antibiotic prophylaxis with IV co-amoxiclav. She is transferred to HDU for overnight monitoring before being transferred to the Gastroenterology ward.

You review Tracy on the Gastroenterology ward round the next morning. She is noticeably drowsier than she was on admission.

Her observations can be seen on the enclosed chart. They are marked observations two.

Tracy is drowsy and confused. There is minimal verbal interaction. She denies being in any pain, but you are otherwise unable to elicit any history.

On examination, she is breathing comfortably, and her chest is clear. She is peripherally warm and well perfused, and her capillary refill time is less than 2s. Her heart sounds are normal, with no additional sounds. She has a flapping tremor in both hands. There is evidence of palmar erythema. Her abdomen is soft but generally tender, and there continues to be abdominal swelling consistent with ascites. There is mild peripheral oedema. She is rousable to voice, but quickly falls asleep again after you speak with her. You feel her GCS is 13 (E3VM6) There is no focal neurology on examination.

Knowledge Check Two - Answers

1. Tracy is suffering from hypovolaemic shock secondary to haemorrhage
2. A - Activate the major haemorrhage protocol to arrange an urgent O negative blood transfusion.  
After an appropriate sample is sent to the transfusion laboratory, group-specific blood can be ready in 20 minutes, and fully crossmatched blood can be ready in 40 minutes. However, a patient who is haemodynamically unstable with evidence of bleeding requires urgent O negative blood. To access this, the major haemorrhage protocol would need to be activated. Who attends and who is alerted to a major haemorrhage varies across health boards. In NHS Lothian, it results in a porter being ready to collect blood, and the blood transfusion laboratory being on stand-by.
3. This situation is a medical emergency, and you would not be expected to manage it on your own. In this case an emergency call (activated by dialling 2222) should have been put to switchboard to gather the medical emergency team, which will include the medical registrar as the team leader. Specific teams you would want to be involved in Tracy's case would be:
  - a. Gastroenterology – in any bleeding patient, a priority must be to stop the bleeding. In the case of an upper GI bleed, this is done with endoscopy performed by the Gastroenterologists.
  - b. Anaesthetics – Tracy is critically unwell and needs a procedure to stop the bleeding. The procedure will need to be supported by anaesthetics.
  - c. Critical Care – Once Tracy has had the procedure, she will need to go to an area in the hospital with advanced monitoring to continue her treatment. This kind of enhanced monitoring is provided by Critical Care.
4. If Tracy is B positive, she has antibodies in her blood to the A antigen. Therefore, she cannot receive any A-type blood. Therefore, she can receive:
  - a. B positive/B negative
  - b. O positive/O negative
5. There are many potential complications to blood transfusion, and to cover them in detail would be outwith the scope of this case. A good summary can be found on the following site: <https://patient.info/doctor/blood-transfusion-reactions>. Some possible complications include:
  - a. Acute haemolytic transfusion reaction (due to ABO incompatibility)
  - b. Febrile non-haemolytic reactions
  - c. Transfusion-associated circulatory overload (TACO)
  - d. Transfusion-associated acute lung injury (TRALI)
  - e. Allergic or anaphylactic reactions
  - f. Transfusion-transmitted bacterial infection
  - g. Transfusion-transmitted viral infection

### Knowledge Check Three

1. What diagnosis could explain Tracy's drowsiness?
2. What are the potential triggers for this condition developing?
3. What further investigations should you consider doing?
4. How is this condition treated?

### **Results**

The Gastroenterology consultant suggests some repeat blood tests to further investigate the drowsiness. He requests an ultrasound of the abdomen. He also requests that one of the medical trainees performs an ascitic tap to get a sample of the ascitic fluid. Abbreviated blood results and the results of the ascitic tap are shown below.

Test	Result	Normal range
Hb (g/L)	85	130 - 180
WCC ( $10^9/L$ )	14.7	4.0 - 11.0
Plt ( $10^9/L$ )	91	150 - 400
Na (mmol/L)	131	135 - 145
K (mmol/L)	5.1	3.6 - 5.0
Urea (mmol/L)	8.2	2.5 - 6.6
Creatinine ( $\mu\text{mol/L}$ )	78	64 - 111
eGFR (ml/min)	>60	>60
Bil ( $\mu\text{mol/L}$ )	87	3 - 21
ALT (U/L)	118	10 - 50
ALP (U/L)	96	40 - 125
GGT (U/L)	155	1 - 35
Albumin (g/L)	22	36 - 47
CRP (mg/L)	143	0 - 5
Ammonia ( $\mu\text{mol/L}$ )	127	11 - 51

#### *Ascitic fluid*

Appearance: turbid, yellow fluid

White blood cell:  $1032 \times 10^6/L$

Red blood cells:  $123 \times 10^6/L$

Gram negative bacilli seen on microscopy

#### *US Abdomen*

Liver cirrhosis with mild splenomegaly. Large volume ascites.

Knowledge Check Three - Answers

1. Tracy's history of alcoholic liver disease with cirrhosis, the flapping tremor and the drowsiness are all suggestive of a diagnosis of hepatic encephalopathy.
2. Potential triggers for hepatic encephalopathy include GI bleeding (as in this case), infection, constipation, dehydration, increased protein intake and the use of sedative medications.
3. Investigations should be used to identify the trigger for hepatic encephalopathy, as well as to ensure that there is no other cause of reduced conscious level. A capillary blood glucose is an essential test. Bloods should be used to investigate for GI bleeding, infection, and electrolyte disturbances. If there is concern about infection, urine culture and CXR would be appropriate investigations. In patients with focal neurology, an intracerebral cause should be suspected, and a CT head performed. As patients with ascites are at high risk of spontaneous bacterial peritonitis (SBP), and infection often triggers encephalopathy, Tracy requires an ascitic tap.
4. Treatment should be focussed on identifying and reversing the trigger for encephalopathy, and by ensuring the bowels are moving regularly with a combination of regular lactulose, and enemas if required.

### Interpretation Two

1. When compared to Tracy's admission bloods, what new abnormalities can be seen on these blood tests?
2. Based on the above results, what other diagnosis can you make?

### **Next...**

Tracy is started on regular laxatives and phosphate enemas to treat hepatic encephalopathy. As per the local protocol, her antibiotics are switched to tazocin, and she receives 20% human albumin solution daily while she is being treated for spontaneous bacterial peritonitis (SBP).

With this treatment, Tracy's confusion resolves. She has no further GI bleeding. Once treatment for SBP is completed, an ascitic drain is inserted to drain the ascitic fluid. After drainage, Tracy is ready for discharge. Her discharge script is below

Medicine	Dose	Frequency
Lactulose 3.1-3.7g/5ml oral solution	15ml	Three times a day
Furosemide 40mg tablets	40mg	Once a day
Spirolactone 100mg tablets	100mg	Once a day
Paracetamol 500mg caplets	1g	As required, no more than four times a day
Propranolol hydrochloride 40mg tablets	40mg	Twice a day
Rifaximin 550mg tablets	550mg	Twice a day

### Activity Two

Go through Tracy's discharge script. Consider what each of these medicines does, and why they were introduced. Is there any monitoring requirements for these medicines?

### Activity Three

Familiarise yourself with the British Society of Gastroenterology Decompensated Cirrhosis Care Bundle. A copy is enclosed at the end of this pack. The bundle covers important considerations, investigations and management for patients presenting with decompensated cirrhosis, and is very useful when you find yourself managing these patients.

### Interpretation Two - Answers

1. Repeat blood tests show raised inflammatory markers, with a raised white cell count and a marked increase in CRP. This would suggest the presence of an infection. The raised ammonia supports a diagnosis of hepatic encephalopathy. The cause of hepatic encephalopathy is multifactorial but is thought to relate to brain exposure of ammonia.
2. The white cell count of greater than  $250 \times 10^6/L$  in an ascitic fluid sample is diagnostic of spontaneous bacterial peritonitis (SBP). The presence of bacteria in the fluid and the raised inflammatory markers both support this.

### Activity Two - Answers

Medicine	Indication
Lactulose 3.1-3.7g/5ml oral solution	Treatment for hepatic encephalopathy
Furosemide 40mg tablets	Diuretic for medical management of ascites
Spironolactone 100mg tablets	Diuretic for medical management of ascites
Paracetamol 500mg caplets	Analgesia
Propranolol hydrochloride 40mg tablets	Prophylaxis against variceal bleeding by reducing portal hypertension
Rifaximin 550mg tablets	Additional treatment for hepatic encephalopathy

Patients who are starting diuretics should have their kidney function monitored in the community. Introduction of the medicines, or a change in dose, should prompt repeat blood tests one week after the change.

### **Conclusion**

Tracy is discharged with outpatient follow up from the hepatology team.

Well done on completing this case. I hope that you have found it informative. If you have any questions, please contact ...

Dr Sophie Horrocks, [Sophie.horrocks@nhslothian.scot.nhs.uk](mailto:Sophie.horrocks@nhslothian.scot.nhs.uk)

Dr Toby Merriman, [Andrew.Merriman@nhs.scot](mailto:Andrew.Merriman@nhs.scot)

**Thank you for completing this long case. As these cases are new intervention, we would really value your feedback. We would be very grateful if you could complete the feedback form accessed from the QR code below.**



Patient details



### 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.

<b>1. Investigations</b>							
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 <sub>4</sub> /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 <b>all patients with ascites</b> using green needle <b>irrespective of clotting parameters</b> and send for ascitic PMN/WCC, culture and fluid albumin						Done <input type="checkbox"/> N/A <input type="checkbox"/>
d)	Record recent daily alcohol intake			..... Units			
<b>2. Alcohol</b> - if the patient has a history of current excess alcohol consumption (>8 units/day Males or >6 units/day Females) <b>N/A <input type="checkbox"/></b>							
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
<b>3. Infections</b> - if sepsis or infection is suspected <b>N/A <input type="checkbox"/></b>							
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 <sup>9</sup> /L (>250/mm <sup>3</sup> ) (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
<b>4. Acute kidney injury and/or hyponatraemia</b> (Na <125 mmol/L) <b>N/A <input type="checkbox"/></b>							
AKI defined by modified RIFLE criteria		1: Increase in serum creatinine ≥ 26μmol/L within 48hrs <b>or</b>					
		2: ≥50% rise in serum creatinine over the last 7 days <b>or</b>					
		3: Urine output (UO) <0.5mls/kg/hr for more than 6 hrs based on dry weight <b>or</b>					
		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 MAP>80mmHg to achieve UO>0.5ml/kg/hr based on dry weight						Y N
e)	At 6 hrs, if target not achieved or EWS worsening then consider escalation to higher level of care						Y N NA
<b>5. GI bleeding</b> – if the patient has evidence of GI bleeding and varices are suspected <b>N/A <input type="checkbox"/></b>							
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 <b>after</b> resuscitation (ideally within 12 hours)						Y N

Continues overleaf..→

Please place in medical notes



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.

Stuart McPherson, Jessica Dyson, Andrew Austin, Mark Hudson 2014