Royal College of Nursing

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Understanding oesophageal varices

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Joseph McArdle describes the risk factors associated with oesophageal varices and the nursing contribution to the various techniques used to treat them.

Aims and intended learning outcomes

This article describes the aetiology of oesophageal varices, the variety of treatment options available and the physiological and psychological nursing needs of patients undergoing these treatment regimes. Its purpose is to produce a balanced overview, looking at nursing care in the acute period and the long-term support required to meet the complex needs of these patients and their carers.

After reading this article you should be able to:

- Identify what lifestyle risk factors are associated with patients who have oesophageal varices.
- Describe the underlying physiology that promotes the development of oesophageal varices.
- State what treatment options are available and their associated complications.
- Recognise the needs of carers and staff when attending to this client group.
- Prepare an action plan for the long-term support of a patient prone to further bleeding episodes.

Introduction

Varicose veins, a series of enlarged, twisted superficial veins, may occur in almost any part of the body and, when damaged, can result in severe bleeding episodes. Oesophageal and gastric varices are varicose veins that occur in the upper gastrointestinal (GI) tract, forming either in the oesophagus itself, or in the fundus of the stomach, at the gastro-oesophageal junction. These too are prone to damage and severe bleeding, leading to a large haematemesis, and potentially a prolonged period of hypovolaemia. This in turn can develop into an acute renal failure that is almost always fatal.

Oesophageal varices account for 5-10 per cent of all medical admissions with GI bleeding (Bennett *et al* 1988, Rhodes and Tsai 1995). The risk of mortality for those presenting with a first variceal bleed is between 30 and 50 per cent (Bennett *et al* 1988, Clearfield and Borowsky 1989, Sauer *et al* 1997), although Conn (1997) suggests that this may even be as high as 70 per cent. The one-year survival rate may be as little as 30-35 per cent (Given and Simmons 1979), although this is also dependent on any associated liver dysfunction.

Any episode of severe internal or external bleeding causes great distress to patients, their carers and nursing staff. It can have huge social and financial implications for the patient, especially if the associated complications are not identified early.

Aetiology

Oesophageal varices are formed as a direct consequence of liver dysfunction, which results in a raised venous pressure in the superficial oesophageal and gastric veins. When in its normal state, the liver is made up of small hexagonal lobules consisting of sinusoids (open spaces) that are supplied with oxygenated blood from the hepatic artery, and nutritionally rich blood from the portal vein. Running alongside these blood vessels is a small canal that joins the hepatic ducts which transport bile to the gall bladder. In the middle of these lobules there is a central vein that feeds into the hepatic veins and subsequently the inferior vena cava. Blood flows from the portal vein and hepatic artery through these sinusoids, and out via the hepatic veins and inferior vena cava back to the heart (Fig. 1).

If for some reason there is a restriction of flow through the liver, the back pressure in the portal vein opens previously latent anastomoses (collateral vessels) between the portal and systemic circulation. This opening of the portosystemic venous collaterals causes the veins of the gastro-oesophageal junction to shunt blood from the left gastric and splenic veins to the superior vena cava via the oesophageal plexus.

These veins are not very elastic and become more fragile as they keep enlarging and their lumens become thinner. The size of these varices, severity of any underlying liver disease and presence of red spots on endoscopy (demonstrating that the walls of the vein are thin) are considered the three principal risk factors in predicting a first variceal bleed (NIECSTOV 1988).

key words
- Gastrointestinal disorders
- Alcohol and alcoholism

These key words are based on the subject headings from the British Nursing Index.
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This restriction of blood flow, the causes of which are not always clear, can be divided into three categories:

- Pre-hepatic.
- Intra-hepatic.
- Post-hepatic.

Further consideration of the intra-hepatic factor separates it into three more subdivisions. They are:

- Pre-sinusoidal.
- Sinusoidal.
- Post-sinusoidal.

A list of causes in these different fields can be found in Table 1.

TIME OUT 1

Revise the structure and function of the liver. Make a list of its key functions.

Portal hypertension

As stated earlier, varices are formed as a result of increased pressure in the portal venous system. When the hydrostatic pressure in the system is beyond the normal 10mmHg, hypertension can be said to occur and the patient is at risk of developing a varix (Garcia-Tsao *et al* 1996). There are three ways to measure this pressure (Cooper *et al* 1987):

- Wedged hepatic venous pressure via a brachial catheter.
- Trans-hepatic venography.
- Direct splenic puncture.

There is still some disagreement about when this hypertension should be clinically defined. Some authors suggest the clinical indicator should be a portal pressure of greater than12mmHg (Elias and Hawkins 1985, McIntyre 1996), while for others it is if the venous pressure rises above 7mmHg (Cooper *et al* 1987).

Although there are numerous causes of portal hypertension (Table 1), the dominant cause is cirrhosis of the liver. A variceal bleed due to non-cirrhotic portal hypertension has a better prognosis, but is rare (Smith-Laing 1981).

Liver cirrhosis

With cirrhosis of the liver, the normal architecture is disrupted by diffuse fibrosis, which is produced by a process of cell necrosis and regeneration. When this process develops slowly, the liver can compensate, but when it is unable to, the prognosis is poorer and treatment options limited. To help assess this, patients may be scored against the widely accepted Child-Pugh (Pugh *et al* 1973) grading system (Table 2).

In the western world, the most prevalent cause of cirrhosis is alcohol. Worldwide however, laval forms of parasitic schistosome worms are responsible for 200 million cases (Gillespie SH *et al* 1996). All patients with liver dysfunction are prone to develop ascites and subsequently retain sodium and water. Although the cause of this is not fully understood, it is suggested that the damage limits the liver's function of deactivating aldosterone, the hormone that regulates the reabsorption of sodium and water, and potassium excretion from the distal renal tubule. It is imperative that an accurate fluid balance be maintained, paying particular attention to a decrease in urine output (hourly measurements are recommended) and gaining a positive balance.

Table 1. Causes of portal hypertension

POSITION OF RESTRICTION	CAUSE
Pre-hepatic	Portal vein thrombosis (umbilical and portal sepsis, malignancy, pancreatitis)
	Splenic vein thrombosis (pancreatic disease)
Intra-hepatic – pre-sinusoidal	Schistosomiasis
	Primary biliary cirrhosis
	Sarcoidosis
	Fibropolycystic disease
	Toxins – arsenic, vinyl chloride and methyldopa
	Sarcoidosis
	Myelosclerosis
Intra-hepatic – sinusoidal	Cirrhosis
	Alcoholic hepatitis
	Chronic active hepatitis
	Congenital hepatic fibrosis
Intra-hepatic – post sinusoidal	Veno-occlusive disease (bush tea, chemotherapy)
	Budd-Chiari syndrome
	Sclerosing hyaline necrosis
Post-hepatic	Right sided congestive cardiac failure
	Constrictive pericarditis
	Inferior vena cava obstruction (thrombus, malignancy)
	Webbing of hepatic vein or inferior vena cava

Fig. 1. Bloodflow through the liver

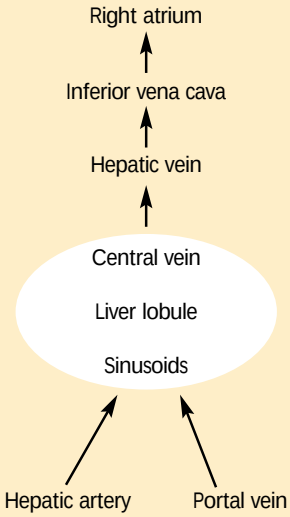


Table 2. Child-Pugh modified grading system for liver disease

FACTOR	SCORE 1	SCORE 2	SCORE 3
Albumin	>35 g/l	30-35 g/l	<35g/l
Bilirubin	<34	34-51	>51
Prothrombin time	< 4 seconds prolonged	4-6 seconds prolonged	>6 seconds prolonged
Ascites	absent	moderate	severe
Encephalopathy	absent	moderate	severe

Grade A = total score 5-6
Grade B = total score 7-9
Grade C = (poorest prognosis) total score >9

Urea and electrolyte levels are checked daily to ensure that both the potassium and sodium levels are kept within their therapeutic ranges. This is managed by using a parenteral replacement therapy for potassium in the acute stage, followed by oral dietary supplements. The use of intravenous saline should be avoided and when the patient is able to eat, a low salt diet is essential. To improve urinary output, a potassium-sparing diuretic may also be prescribed.

An additional function of the liver is the synthesis of clotting factors. When its capacity for this function becomes impaired, prothrombin times increase, heightening the risk of uncontrollable bleeding, which may be exacerbated by the patient's poor nutritional state. To counter these risks, vitamin K is administered, and in some cases it might also be necessary to give fresh frozen plasma.

Hepatic encephalopathy

All patients with an acute bleed are at risk of developing a confusional state, hepatic encephalopathy. The precise mechanism that leads to this syndrome (also known as portosystemic encephalopathy – PSE) is unclear, but it is thought that the toxins not cleared through the liver cross the blood-brain barrier, leading to altered mental states. Hepatic encephalopathy can be considered as having three stages (Bouchier 1982):

- Stage one – this stage is reversible and symptoms include disorders of mood, personality, intellect and sleep, irritability, childishness, untidiness, apathy, paranoia, constructional apraxia and disorientation.
- Stage two – here the symptoms are more pronounced. The patient may start smacking his or her lips or pulling abnormal facial expressions. He or she may also have a tremor.
- Stage three – by this stage, coma may occur, ultimately proving fatal.

Alcohol detoxification may also contribute to the confusion and may require treatment with a benzodiazepine sedative, providing there is no liver toxicity or renal impairment present.

Treatment methods

There are four principal approaches to treating bleeding oesophageal varices – pharmacological, endoscopic, balloon tamponade and surgery. There

Table 3. Potential side effects of pharmacological treatments

TYPE OF DRUG	DRUG NAME (REGIME)	SIDE EFFECTS
Antidiuretic hormone (Posterior pituitary hormone)	Vasopressin (20 units in 100ml dextrose 5 per cent over 15 minutes) Terlipressin (2mg bolus then 1-2mg 4-6 hourly)	Angina secondary to coronary artery constriction. This may progress to a full myocardial infarction so extreme care is required when given to patients with cardiovascular disease. Abdominal cramps. Pallor and blanching. Nausea. Headache. (These side effects are noted to be less with terlipressin)
Beta adrenoreceptor blocking drugs (Beta blockers)	Propronalol (40-80mg 12hrly)	Bradycardic hypotension which might exacerbate any already present due to hypovolaemia. Bronchospasm – not recommended for people with asthma. Nausea and vomiting. Caution is also required if administered to diabetic patients, as their hypoglycaemic early warning mechanisms may become impaired.
Somatostatin	Octreotide (50µg iv bolus then 50µg/hr via infusion for an acute bleed when used for preventative therapy, then 50µg subcutaneously 12 hourly)	Pain and irritation at local subcutaneous injection site. Diarrhoea, bloating, flatulence and nausea. Abdominal pain. A drop in serum insulin levels leading to hyperglycaemia. A cytoprotective agent such as sucralfate may also be given to promote the healing effect in the gastric lesion.

(Martindale 1993)

Fig. 2. Minnesota adaptation of Sengstaken-Blakemore tube

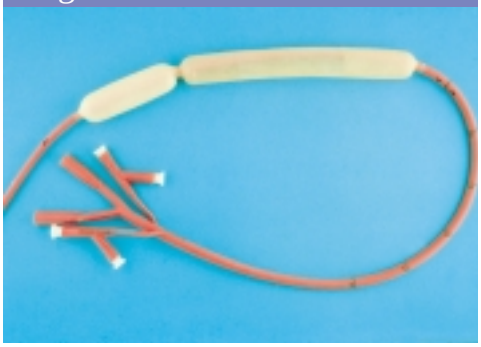
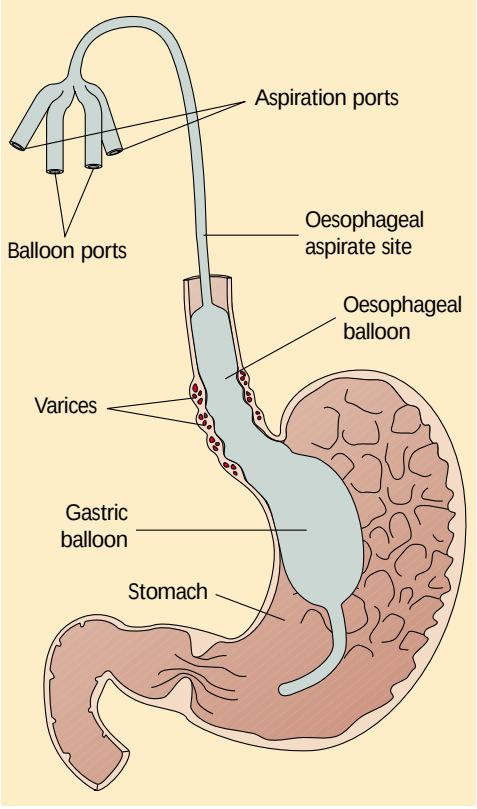


Fig. 3. Minnesota tube in position



continue to be numerous studies comparing the efficacy of individual and combined approaches to treatment as emergency and long-term therapy.

The pharmacological advances Some medications can decrease the portal venous pressure and so reduce the risk of variceal haemorrhage. Drugs currently used are beta-blockers, antidiuretic hormone and octreotide. Nurses administering drugs must always be aware of their potential side-effects and be able to recognise them in practice (Table 3). Despite recent studies showing that octreotide (an analogue of somatostatin) has a significant role in pharmacological therapies, it is still in the evaluation stage of randomised controlled trials (Avengerinos *et al* 1997, Besson *et al* 1995, Gillespie IE *et al* 1996) and has not yet been licensed for this practice. It therefore remains available only by consultant prescription (Sutton 1998).

Endoscopic treatments There are two endoscopic techniques used. Each is intended to stop blood getting to the bleeding point:

- Variceal banding – here the varix is occluded by a strong elastic band. This method is similar to the surgical procedure of transoesophageal ligation, with a staple gun via a gastrotomy (Burroughs 1987, Jones 1985).
- Sclerotherapy – in this approach the varix is injected with a sclerosing agent such as ethanolamine oleate. If any extravasation occurs, the surrounding tissue may necrose.

Both techniques compare favourably with surgery and are advocated as the first-line treatment in controlling an acute haemorrhage, and as long-term preventive therapy. They are palliative treatments as they do not affect the underlying physiology. Both demonstrate a marked improvement in survival from the initial bleed, but the overall mortality risk for the patient continues to be associated with the degree of liver failure, rather than the bleeding episode.

The potential complications of both procedures are similar, with a risk of oesophageal perforation, ulceration, medianitis and stricture formation, especially if used on gastric varices. A patient will need to be re-examined by gastroscope several times until the varices are obliterated, and at six-monthly intervals thereafter to observe and manage recurrence.

Balloon tamponade This procedure is particularly frightening and unpleasant for the patient, and distressing for the family. It must be clearly explained to all concerned.

A balloon tamponade device is a gastric tube that has one (gastric only – Linton-Nachlas) or two (gastric and oesophageal – Minnesota, Sengstaken-Blakemore) reservoirs surrounding it (Fig. 1). These reservoirs can be inflated with air or sterile water. The tube may also have a separate channel leading from an oesophageal opening to allow drainage above the balloon. The device may therefore have three or four ports which must be clearly labelled.

Every first-aider knows that to stop bleeding, pressure needs to be applied to the wound. A balloon tamponade device is able to do this unhindered by the oesophagus (Fig. 3). Pressure is applied to the vessels in the oesophageal gastric junction via the gastric balloon, restricting blood flow to the oesophageal vessels. This pressure also helps to secure the tube in place. If the bleeding in the oesophagus continues, the oesophageal balloon can also be inflated to a pressure that is slightly greater than portal pressure (20-30mmHg). This pressure can be checked by using a manometer attached to a Y connection. Each balloon should be checked for leaks prior to insertion by hyperinflating to 50-60mmHg while under water.

All of the devices carry a complication risk of 10-20 per cent. These include:

- Oesophageal rupture following hyperinflation within the oesophagus or the tube curling back on itself during insertion (Crerar-Gilbert 1996).

- Pressure necrosis, similar to pressure sore formation. If the oesophageal balloon is inflated, it must be deflated for five minutes every hour.
- Aspiration pneumonia secondary to blood inhalation from above the oesophageal balloon. With devices that have no oesophageal aspiration channel, it is essential that regular oropharangeal suctioning occurs. If this channel is available, a low-grade suction device may be attached, provided that over-suctioning does not occur, causing trauma and exacerbating the bleed.
- Re-bleeding on tube removal.

Oral medication can be administered via the gastric aspiration channel and flushed through with 30-50ml of water. It is imperative, though, that any gastric suctioning be stopped for 30 minutes to allow absorption.

It is good practice for the balloons to be inflated for no more than 12-24 hours. Once the balloons are deflated, the tube should be kept *in situ* until it is certain that bleeding has stopped. When this has been established the tube is then gently removed, bearing in mind that this process itself may re-activate bleeding by removing the wound clot, especially if the balloons are not fully deflated. An accurate record of the volume inflated must be kept, especially as the tube may have been inserted in an endoscopy unit and removed in the ward. If the bleed is re-activated, the tube may be re-inserted for a further 24 hours.

TIME OUT 2

Discuss with your colleagues what fears and anxieties a patient might have concerning balloon tamponade therapy. Write a short statement on how you would explain the treatment to patients .

Surgical decompression techniques The creation of an artificial shunt, moving blood into the inferior vena cava prior to entering the liver, decompresses the hypertension in the portal venous system. This can be by a full diversion (portocaval), partial diversion (splenorenal, mesocaval) or by selective variceal decompression (Warren shunt). These procedures carry a significant increase in the risk of hepatic encephalopathy. They are a tertiary rather than emergency option, as they have a 50-90 per cent mortality risk in the non-elective approach (Rhodes and Tsai 1995). The operation involves an abdominal incision so the associated complications include the usual post-operative risks of shock, haemorrhage, anoxia, urinary retention and pain. However, the complications also carry the specific risks of GI bleeding, encephalopathy, ascites and hepatic anoxia.

It is critical that post-operative oxygen therapy is maintained for a minimum of 24 hours. Another complication is that the grafts themselves have a potential to clot, requiring further surgery and reducing the potential of liver transplantation, a technique that some centres are considering as treatment for end stage liver disease.

A less invasive operation is the transjugular intrahepatic portalsystemic shunt (TIPS). Here, a venous connection is made between the hepatic and portal vein by passing a stent via the internal jugular vein into the hepatic vein. The long-term performance of this technique is currently under review and it too carries a high risk of encephalopathy and shunt dysfunction (Sauer *et al* 1997). A further advantage of this procedure is that it does not compromise future transplantation.

Nursing care of the patient

Regardless of treatment, the patient will continue to be at risk of the generic problems associated with hospitalisation:

- Dangers from hospital and self.
- Personal care.
- Socio-spiritual care.

When thinking about safety factors, those primarily considered are:

- Haemorrhage.
- Mental state.

Any acute haemorrhage needs close observation of pulse, blood pressure, oxygen saturation, central venous pressures, urine output, full blood count and clotting times. Where there is a haemoglobin count of less than 10g/l, a blood transfusion might be indicated, but the risk of over-transfusion, causing increased portal pressure and worsening the bleeding episode, needs to be considered (Rhodes and Tsai 1995). More appropriate indicators are the clinical signs of hypovolaemia or shock. These are a systolic blood pressure of <90mmHg, a tachycardia >120mmHg, an oxygen saturation <95 per cent, a drop in central venous pressure, urine output <30ml/Hr and the visual signs of peripheral shutdown – pallor, cyanosis and clamminess. Regular suctioning is required to prevent aspiration, especially if a tamponade device is *in situ*.

TIME OUT 3

Think about the frequency of observations you would carry out on the patient. Write up a short rationale on when and why you would change this frequency.

Mental state The mental state of patients can determine if their safety and that of the nurse is compromised. Pre-procedural information can reduce anxiety, and it is essential that we let patients and their carers know the rationale for our actions at all times. Consideration must be given to the bedside environment, ensuring that all risks are minimised, in particular if the patient has become encephalopathic or unco-operative, which may be manifested by attempts to remove any lines or tubes attached.

If a tamponade device is in position, vigilant observation is required to ensure that the patient does not pull the tube out and cause a further bleeding episode. Some patients may also attempt to move

unaided without the necessary co-ordination or cognitive skills, increasing their chance of fall injuries. Local protocols will determine the safety measures to be used, with some centres nursing the patient on a mattress on the floor and others using bedside supports (cot-sides). Both approaches require an explanation to the patient and his or her family. The nurses' safety may also be at risk and an agreement between the medical team, nursing team and patient/patient's carers must be achieved. Only rarely will a sedative be given due to the amount of liver impairment present.

TIME OUT 4

Take some time to think about what local nurse management issues would influence your delivery of care to this client group. List the issues and make a note of any key actions you could take to address them.

Personal care Most hospitalised patients require support in meeting their personal care needs and a patient with an oesophageal bleed requires special attention to be paid to nose and mouth care. Regular oral hygiene must be administered as the taste of blood in the mouth is very unpleasant and distressing. If tamponade therapy is in progress, the mouth is prone to be coated in debris. Cleaning away any excess blood and applying a water-soluble lubricant to the lips and whichever nostril is intubated helps to alleviate any irritation for the patient.

Any blood in the bowel may be absorbed as ammonia, causing encephalopathy, so it is important to expedite the passage of the blood through the bowel. A two-pronged approach is practised:

- The use of an oral laxative.
- The administration of a magnesium sulphate enema given as often as twice a day.

The administration of an enema relies on high quality nursing care to maintain skin integrity and patient dignity, especially as melaena is notoriously tar-like and difficult to remove from the skin unless meticulous care is taken.

Many patients are undernourished and have poor appetites. The fact that they require a low salt diet is difficult for them, especially as the meals might not be particularly appealing. Excellent teamwork between the dietician, kitchen and nursing staff must be in place and nutritional supplements must be readily available for the patient.

Psychological support Providing information to a patient helps reduce anxiety and promotes a therapeutic relationship between a patient, patient's carer and nursing staff. For nurses to provide reassurance, however, they must (Salter and Berretta 1998):

- Have a positive attitude towards the patient.
- Perceive the patient as a valuable person.
- Exhibit positive interpersonal skills toward or on behalf of the patient.
- Possess knowledge related to the causes of anxiety.

It is a cardinal requirement, therefore, that the nurse does not become judgmental, even when the patient has become unco-operative. The very moment the nurse judges the patient, the relationship becomes tarnished.

We are human and can become stressed, frustrated and irritable when confronted with the self-destructive behaviour demonstrated by some patients. The importance of being aware of body language and non-verbal cues, and using them to maintain this positive relationship cannot be over-emphasised. As trusted carers, nurses are able to promote the positive lifestyle changes that are crucial if long-term survival is to be achieved.

TIME OUT 5

Think about what non-verbal communications you send as part of your everyday life and how they might affect your relationship with self-destructive patients.

Long-term support

As alcohol is the biggest contributor to variceal bleeding in the western world, nurses have a key role in challenging patients on their drinking habits, providing them with brief advice, helping them to identify factors that make compliance to abstinence orders difficult and directing them to appropriate agencies.

These agencies include an alcohol specialist nurse, if available, substance dependency units that provide alcohol support and charitable organisations or self-help groups. Some useful contact details are included at the end of this article.

Regardless of the cause of admission, patients benefit if they are assessed on their alcohol consumption and counselled early. A study by Chick *et al* (1985) showed a significant improvement in alcohol related problems over a subsequent 12-month period in patients who received this ward-based counselling. The administration of the simple 'CAGE' questionnaire on admission can determine if further advice is required (Dawson 1995). This asks the patient the following four questions.

- Do you ever feel you should CUT down on your drinking?
- Does others' criticism about your drinking ever ANNOY you?
- Have you ever felt GUILTY about drinking?
- Have you ever had a drink (EYEOPENER) to steady your nerves or get rid of a hangover?

One of the reasons given for not stopping drinking is social deprivation, so a referral to a social worker to meet the needs that drive the patient to drink may be obligatory. Isolation and social circles also contribute to the patient's dilemma, so it is important to emphasise the need for long-term support to the patient's carers, and recognise that this may include lifestyle changes of their (the carers') own.

Patients who have an acute episode of haematemesis secondary to oesophageal varices present with a major threat to their lives. It is imperative that they are supported through both the acute and long-term stages of recovery.

In the initial stage of admission the predominant focus will be on maintaining patient survival and safety, and the psychological support of carers. It is imperative that nurses are aware of possible factors that may compromise the patient's recovery. These include:

- Airway obstruction/aspiration.
- Hypovolaemia due to incessant bleeding.
- Confusional states – detoxification or encephalopathy leading to falls or self-harm.
- Oesophageal rupture.
- Re-bleeding upon removal of tamponade device.
- Over-sedation secondary to hepatic failure.
- Renal failure.
- Poor nutritional state slowing the healing process.

Long-term support needs to be planned with referral to appr