https://doi.org/10.60115/11055/1191

Perioperative considerations for the patient with portal hypertension: Anaesthesia management of common interventions

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Edited by Associate Professor Matthew Doane

BACKGROUND

In Australia and New Zealand, rates of chronic liver disease and its associated complications are on the rise. This increasing burden of liver disease highlights the importance for anaesthetists to understand the risks and complexities that make perioperative management challenging for this unique group. In general, patients with liver disease have a significant risk of morbidity and mortality after anaesthesia and surgery, with potential for deterioration into life-threatening hepatic failure. Overall, patients with liver disease have higher transfusion requirements, higher infection rates, increased rates of cardiac compromise and longer hospital stays compared to those without liver disease.[1]

Advances in screening and management of liver disease and its complications, namely portal hypertension, presents a challenge for the anaesthesia community. Historically, treatment of portal hypertension and its sequelae was limited to medical management or liver transplantation. There have been a number of recent expansions in screening and treatment options that require anaesthetic involvement, and a subsequent need for familiarity by the anaesthetists who are likely to encounter them. These interventions include screening endoscopy, variceal banding and transjugular intrahepatic portosystemic shunts (TIPS). Anaesthesia expertise is required for an increasing number of these procedures in both the acute and elective setting, and frequently in remote anaesthesia locations.

The intention of this article is to discuss the anaesthetic considerations related to these interventions. Of note, smaller procedures, such as drainage of ascites, are typically performed under local anaesthetic with no specialist anaesthetist involvement required. While trans-arterial chemoembolization (TACE) and microwave ablation of liver lesions often do require the involvement of an anaesthetist, these procedures are not specifically due to the presence of portal hypertension and are therefore not discussed.

PORTAL HYPERTENSION

Portal hypertension is a major consequence of liver cirrhosis and the cause of end stage liver disease complications such as ascites, variceal bleeding, and thrombocytopenia. While liver cirrhosis is the leading cause of portal hypertension, several other non-cirrhotic causes exist, with schistosomiasis (a parasitic infection) being the second most common cause.[2] Non-cirrhotic aetiologies of portal hypertension can be categorised into pre, intra and post hepatic causes. These categories are demonstrated below, in Table 1.

Table 1. Non-cirrhotic causes of portal hypertension based on perihepatic pathology[3,4]

Pre-hepatic - Portal or splenic vein thrombosis - Splanchnic arterio-venous fistulas
Intra-hepatic - Pre-sinusoidal ▪ Polycystic liver disease ▪ Congenital hepatic fibrosis ▪ Arterio-venous fistulas ▪ Biliary disease (biliary cholangitis, autoimmune cholangiopathy, primary sclerosing cholangitis, other biliary injury) ▪ Neoplastic occlusion of portal vein ▪ Granulomatous liver disease (schistosomiasis, sarcoidosis) ▪ Idiopathic noncirrhotic portal hypertension - Sinusoidal ▪ Amyloid ▪ Fibrosis of the space of Disse ▪ Sinusoid destruction in acute liver injury ▪ Infiltrative diseases (mastocytosis, Gaucher disease, idiopathic myeloid metaplasia) - Post-sinusoidal ▪ Sinusoidal obstruction syndrome ▪ Budd-Chiari syndrome ▪ Phlebosclerosis of hepatic veins (alcohol-associated liver disease, chronic radiation injury) ▪ Primary vascular malignancies ▪ Granulomatous phlebitis ▪ Lipogranulomas
Post-hepatic - Hepatic vein or IVC obstruction (Budd-Chiari syndrome) - Constrictive pericarditis, restrictive cardiomyopathy

The portal vein receives blood drained from the gastrointestinal tract, stomach, pancreas, gallbladder, and spleen. The blood flows into the liver via these pathways and back to the inferior vena cava via the hepatic vein. Under normal conditions, the portal vein supplies the liver with approximately 75% of its blood supply (the other 25% being from the hepatic artery). Portal hypertension develops when there is an increase in portal venous pressure. This leads to the development of collateral portosystemic vessels which shunt blood back to the systemic circulation, bypassing the liver.

Portal hypertension is typically a diagnosis made in patients with known risk factors (e.g. cirrhosis) who present with clinical manifestations of elevated portal pressures. The gold standard for quantification of portal hypertension is performed by measuring the hepatic venous pressure gradient (HVPG). This procedure is conducted by placing a pressure transducing catheter into a hepatic vein, and the difference between balloon wedged hepatic venous pressure and free hepatic venous pressure is measured. A normal HVPG is up to 5mmHg; portal hypertension is defined as an HVPG ≥ 6mmHg. Clinically significant manifestations of portal hypertension typically occur at a HVPG of > 10mmHg.[5] Measurement of a HVPG does offer prognostic value (particularly when ≥ 20mmHg), however it is not routinely performed.[6,7] Ultrasonography and transient elastography can also be used to assess portal hypertension, but neither are used to confirm diagnosis.

Table 2. Common complications of portal hypertension

- Gastroesophageal varices
- Portal hypertensive gastropathy
- Ascites
- Spontaneous bacterial peritonitis
- Splenomegaly
- Thrombocytopenia
- Hepatorenal syndrome
- Hepatic hydrothorax
- Hepatopulmonary syndrome
- Portopulmonary hypertension
- Cirrhotic cardiomyopathy
- Portal cholangiopathy

Cirrhotic liver disease can be broadly classified into two main stages: compensated and decompensated cirrhosis. The distinction depends on the presence of decompensating events such as variceal haemorrhage, ascites, and encephalopathy. This classification has significant implications for expected lifespan. With compensated cirrhosis, median survival exceeds 12 years, whereas patients with decompensated cirrhosis have a median survival of 1.8 years.[8] A list of common and important complications secondary to portal hypertension is presented in Table 2.

GASTROESOPHAGEAL VARICES

One of the earliest consequences of portal hypertension is the development of portosystemic collaterals and the subsequent formation of gastroesophageal varices. By definition, patients who have developed gastroesophageal varices have clinically significant portal hypertension (HPVG ≥ 10mmHg) and are more likely to progress to decompensated cirrhosis.

Incidence

Gastroesophageal varices are present in 25-40% of patients with compensated cirrhosis, and in up to 85% of those with decompensated cirrhosis.[9]

Surveillance

Patients with advanced chronic liver disease will commonly present to the endoscopy suite for elective screening or surveillance endoscopy. Since the introduction of the Baveno criteria, a validated tool used to rule out high risk varices based on non-invasive tests, screening endoscopy can now be avoided in a significant number of patients.[10-12] Depending on the result of previous endoscopy, surveillance can generally be performed every 2 years. Repeat endoscopy should be performed in the event of any decompensation.[11,12] Consequently, the indication for endoscopy (for example, decompensation) may inform the anaesthetist of the likelihood of varices being present and any intervention required.

Prevention of variceal haemorrhage

Advanced chronic liver disease patients with clinically significant portal hypertension commonly receive non-selective beta blockers (e.g. carvedilol or propranolol) as first line treatment to prevent variceal bleeding.[10,11] Patients with high-risk varices who have a contraindication to non-selective beta blockers will receive prophylactic endoscopic ligation, repeated until eradication is achieved.[11] Any contraindication to non-selective beta blockade therapy may be of interest to the anaesthetist, and may include second- or third-degree heart block, asthma, or persistent hypotension.

In the broader surgical population, a history of chronic liver disease and treatment with a non-selective beta blocker should trigger an awareness that there is probable significant portal hypertension and sequelae may be present. Care should be taken with any procedures in the oesophagus (for example, nasogastric/orogastric tube insertion or insertion of a transoesophageal echocardiogram probe).

Anaesthesia considerations for endoscopy and band ligation

Routine patient assessment should be performed and an assessment of the severity of liver disease made (compensated versus decompensated). The decision to use sedation or general anaesthesia with an endotracheal tube will be influenced by usual factors, including aspiration risk and the patient's ability to maintain their airway. Additionally, the risk of peri-procedural bleeding and a reduced respiratory reserve (from massive ascites, for example) will also impact this decision. Considerations for tailoring of anaesthetic agents and dose adjustments will be discussed later in this article.

ACUTE VARICEAL BLEEDING

Gastrointestinal bleeding is a common decompensating event in patients with end-stage liver disease. The most common cause of bleeding in these patients is gastroesophageal varices, followed by gastroduodenal ulceration.[13] Concomitant bacterial infection, HVPG >20mmHg and Child Pugh C score are risk factors for mortality after variceal haemorrhage.[10] Rebleeding is common after an index bleed but is significantly reduced by effective treatment of portal hypertension.[13]

Pharmacological treatments

Typically, patients will receive a bundle of care aimed to reduce the risk of rebleeding and the overall risk of mortality. This may include:

- Early antibiotic prophylaxis[13,14]
- Intravenous proton pump inhibitor therapy, continued until varices are confirmed[13,15]
- Intravenous splanchnic vasoconstrictor (e.g. octreotide, terlipressin)[16,17]
- Prokinetic agents (e.g. erythromycin, metoclopramide) to promote gastric emptying and to improve procedural visualisation[11]

Interventional procedures

All patients with suspected variceal haemorrhage should be referred for endoscopy within 12 hours of presentation, or sooner if haemodynamic instability is present.[11] Endoscopic band ligation is the intervention of choice when oesophageal varices are present. While band ligation is preferred for oesophageal varices, patients with gastric varices are generally treated with cyanoacrylate injections, as band ligation is typically less successful.[10,15]

If haemostasis of variceal bleeding varices cannot be achieved endoscopically, balloon tamponade can be used as a temporising intervention. Commonly used devices are the Sengstaken-Blakemore and Minnesota tubes. These methods should only be used as a bridge (<24 hours) to definitive therapy (that is, a transjugular intrahepatic portosystemic shunt). Patients must be intubated, ventilated and admitted to an intensive care unit. Balloon techniques are associated with a high risk of complications, including oesophageal ulceration, perforation and pneumonia.[18] Oesophageal stenting with self-expanding metal stents can also be considered as a bridging therapy. These stents can be left in place for 7 days or more. Potential complications of stenting include migration and ulceration, though complications of stenting may be less common compared to balloon tamponade techniques.[11,18]

ANAESTHESIA CONSIDERATIONS FOR PATIENTS WITH ACUTE VARICEAL HAEMORRHAGE

General measures

Large bore peripheral IV access should be established and resuscitation with a balanced crystalloid solution initiated. Consideration should be given to placement of an arterial line for haemodynamic monitoring and serial blood sampling. Activation of the massive transfusion protocol or discussion with blood bank may be prudent. Hypothermia should be avoided, given the pre-existing propensity to coagulopathy in this population and the possibility of large volume transfusion.

Transfusion strategy

It has been suggested that aggressive restitution of the intravascular volume increases portal pressure and, therefore, may worsen variceal bleeding or cause rebleeding.[11] A restrictive transfusion strategy (haemoglobin threshold of ≤ 70g/dL prior to packed red blood cell administration) has been associated with lower mortality and rebleeding rates.[19] This strategy is recommended for patients with acute gastrointestinal haemorrhage who are haemodynamically stable and do not have a history of significant cardiovascular disease. Patients with significant cardiovascular disease should have a more liberal transfusion threshold (e.g. ≤ 80g/dL).

It is critical, however, to recognise the distinction between patients who are haemodynamically stable and those who are exhibiting signs of shock and instability. Bleeding can be rapid and the clinical picture dynamic. Consequently, a haemoglobin value ≥ 70g/dL may be falsely reassuring in the face of active bleeding. Withholding resuscitative efforts and transfusion for an unstable patient is not appropriate and may lead to catastrophic outcomes.

Correction of coagulopathy

Anticoagulation agents should be ceased and reversed where possible. Thought should be given to general measures which support the coagulation system. This includes ensuring a core temperature of greater than 35 degrees Celsius, avoiding acidosis, and maintaining an ionised calcium level > 1.1 mmol/L, with particular vigilance required in the setting of a massive transfusion.

Despite thrombocytopaenia, prolonged prothrombin time and a raised INR being common features of advanced chronic liver disease, they may not necessarily correlate with bleeding risk in these patients.[20] Routine transfusion with platelets and fresh frozen plasma (FFP) is not recommended, nor can a threshold value for transfusion be definitively suggested.[10] Rather, correction of coagulopathy should be guided by viscoelastic testing where possible.[21] Unnecessary transfusion is not only a waste of a valuable resource, but it may cause harm.[22] Prothrombin complex concentrate may be a more effective option than FFP in correcting the INR, while avoiding additional intravascular volume expansion.[22] Recombinant factor VIIa has not been demonstrated to be of benefit.[11]

Haemodynamic goals

Early introduction of vasopressors to avoid haemodynamic compromise at induction is recommended in addition to any ongoing transfusion requirement. While vasopressin may provide theoretical benefit of splanchnic vasoconstriction, administration should be via a central venous line. Practically, metaraminol or phenylephrine are suitable. A mean arterial pressure target of 65mmHg is reasonable, acknowledging there may be patient factors which alter this target. Overzealous administration of vasopressors and subsequent hypertension prior to gaining control of the bleeding source is potentially harmful.

Airway protection

Generally, airway protection with rapid sequence induction and endotracheal tube placement will be required for patients having interventional procedures for variceal haemorrhage. Early extubation is encouraged but will be dictated by the haemostasis achieved and the patient condition at the end of the procedure.

TIPS PROCEDURE

Transjugular intrahepatic portosystemic shunts (TIPS) are used to reduce portal hypertension. A radiologically placed TIPS creates a low-pressure route between the intrahepatic portion of the portal and hepatic veins. This effectively shunts blood from the congested portal system, directly back to the hepatic vein for return to the systemic circulation. Offloading the portal system helps reduce complications of portal hypertension, such as ascites and variceal bleeding. Prior to the modern TIPS procedure, open surgical porto-caval shunts were performed, but these were associated with high rates of morbidity and mortality.[23]

TIPS may be used electively or as a rescue intervention where medical management has failed. TIPS have shown both survival and symptomatic benefit in patients requiring serial large-volume paracentesis.[3,24] While endoscopic treatment of variceal disease is the gold standard treatment, TIPS have shown a survival benefit when used for management of varices and variceal bleeding.[24] Less common indications for TIPS include portal hypertensive gastropathy, gastric antral vascular ectasia, refractory hypertensive hydrothorax, hepatorenal syndrome, Budd-Chiari syndrome, hepatic veno-occlusive disease and hepatopulmonary syndrome. Evidence for these indications, however, is more limited. Other indications for TIPS include elective placement prior to surgery in cirrhotic patients (as an attempt to reduce the risk of hepatic decompensation perioperatively), and as a bridge to liver transplantation.[25,26] Despite these indications, there are several conditions that are considered both absolute and relative contraindications for TIPS placement. These are presented below, in Table 3.

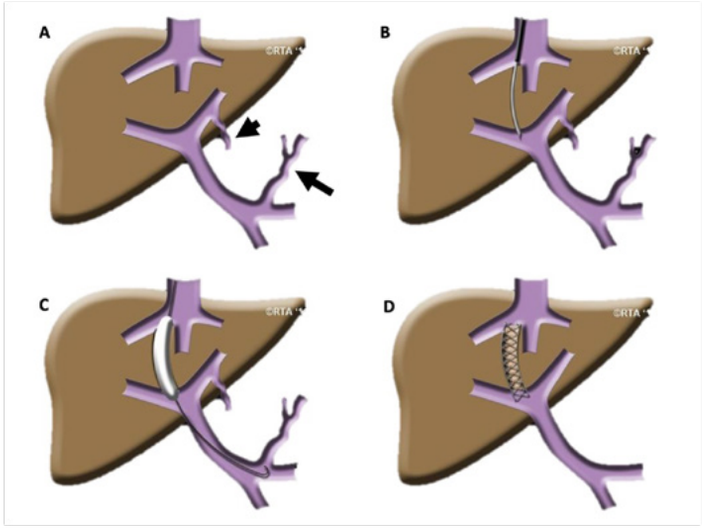
Table 3. Contraindications to insertion of a TIPS[23,27,28]

Absolute contraindications ▪ Congestive heart failure ▪ Severe tricuspid regurgitation ▪ Severe pulmonary hypertension ▪ Polycystic liver disease ▪ Active systemic sepsis ▪ Unrelieved biliary obstruction
Relative contraindications ▪ Hepatic encephalopathy ▪ Hepatocellular carcinoma, particularly if centrally located ▪ Obstruction of all hepatic veins ▪ Portal vein thrombosis ▪ Thrombocytopenia (platelet count <20) ▪ Severe coagulopathy (INR >5) ▪ Moderate pulmonary hypertension

THE PROCEDURE

TIPS is an interventional radiology procedure commonly performed under general anaesthesia. The internal jugular vein (usually right) is cannulated, and a catheter passed into the hepatic vein. A wedge pressure is measured and the HVPG is calculated. Contrast is used to identify the relevant hepatic vascular anatomy and a tract is made between a branch of the hepatic venous and the portal venous system. The newly formed tract is then dilated using a balloon, and a stent is deployed to maintain patency. Typically, a TIPS procedure is deemed successful if the HVPG returns to normal, with a minimum goal of <12mmHg.[24,28] The procedure is summarised in Figure 1. The patients selected for a TIPS often present with numerous comorbidities. These comorbidities, the invasive nature of the procedure and the physiological changes during and after TIPS placement present several periprocedural complications that should be familiar to those providing care to these patients. These complications are summarised in Table 4.

Figure 1. Steps in a TIPS procedure



A) portal hypertension has caused the coronary vein (arrow) and the umbilical vein (arrowhead) to dilate and flow in reverse; B) a needle has been introduced (via the jugular vein) and is passing from the hepatic vein into the portal vein; C) the tract is dilated with a balloon; D) stent is deployed and portal pressure is normalised, with the coronary and umbilical veins no longer dilated. ("Tips schematic," R. Torrance Andrews, MD, Wikipedia, CC BY 1.0)

Table 4. Potential complications from TIPS insertion[29-31]

Procedural related complications - Internal jugular vein access complications ▪ Carotid, right atrial or tracheal puncture ▪ Pneumothorax ▪ Haemothorax ▪ Thoracic duct injury ▪ Brachial plexus injury - Arrythmias due to catheter passage into right atria/right ventricle - Complications associated with portal vein puncture ▪ Catastrophic haemorrhage/haemoperitoneum ▪ Liver capsule puncture - Injury to surrounding structures ▪ Inferior vena cava ▪ Hepatic artery ▪ Biliary structures ▪ Right kidney - Stent complications ▪ Misplacement ▪ Stent recoil/migration - Radiation injuries
Other complications - Issues relating to porto-systemic shunting ▪ Hepatic encephalopathy ▪ Cardiac decompensation/failure - Liver failure - Infection/sepsis - TIPS dysfunction ▪ Stent occlusion/thrombosis/dislodgement - Rare complications ▪ Haemolytic anaemia ▪ Cardiac perforation due to stent embolization ▪ Mechanical haemolysis

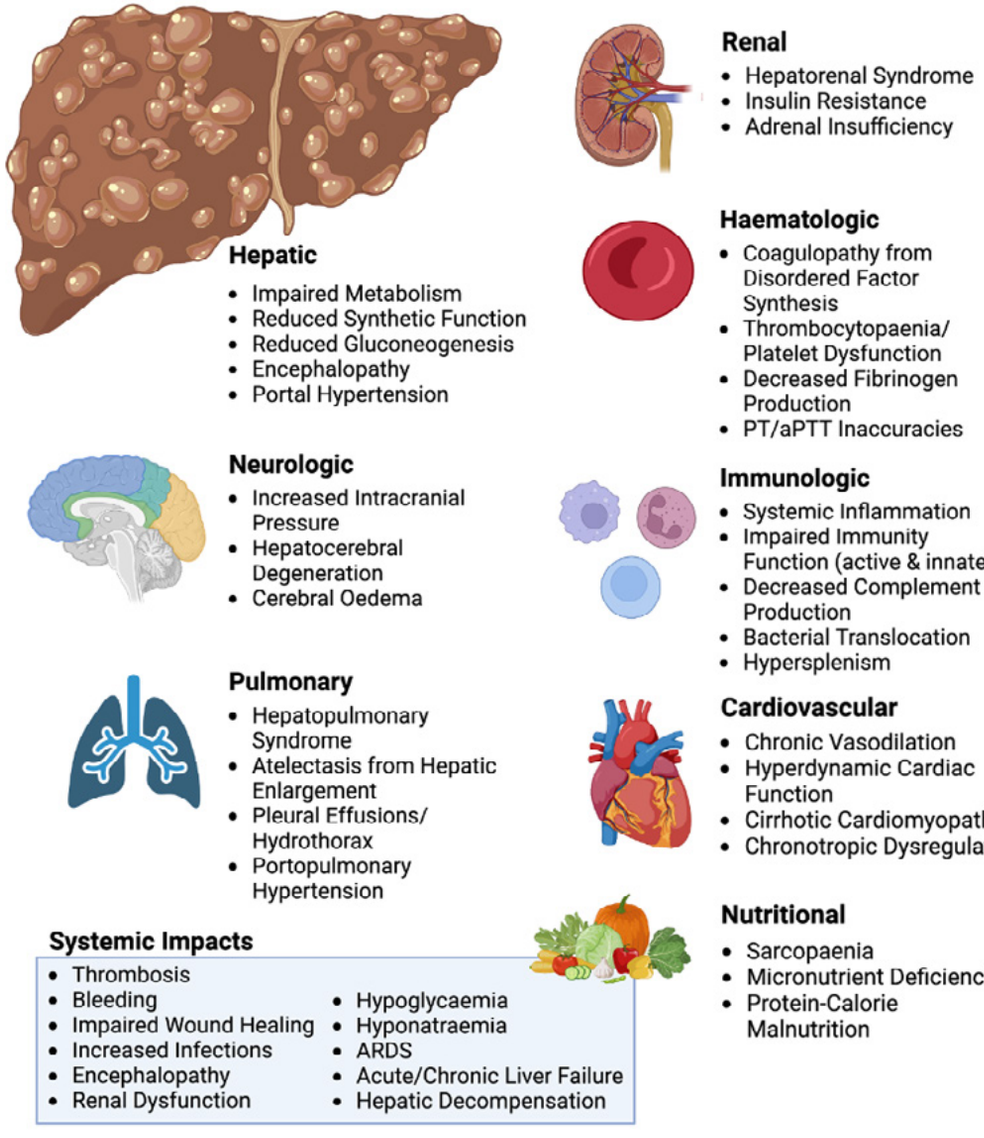
ANAESTHESIA MANAGEMENT

Pre-optimisation will involve the local hepatology team. The patient's eligibility for liver transplant is important to note. Although uncommon, TIPS can result in acute decompensation and deterioration into fulminant liver failure, potentially necessitating rapid assessment for liver transplantation.

In addition to a standard pre-operative assessment, consideration should be given to the physiological and pharmacological complications of advanced liver disease. A broad, by no means complete, list of systemic derangements that may need to be considered in the patient presenting with hepatic dysfunction is presented in Figure 2. Specific focus should be given to the cardiovascular system, identifying any history or clinical evidence of heart failure or pulmonary hypertension. Up to 50% of patients with advanced cirrhosis may have some form of cardiac dysfunction and often present with a hyperdynamic circulation. While these patients have an increased cardiac output at rest, there is reduced ability to respond to physiological stress.[30] Post TIPS, there is an increase in venous return as the portal system is offloaded and circulating volume returns to the systemic circulation. This fluid challenge results in an increase in cardiac output and exposure to inflammatory

mediators, which can lead to cardiac decompensation in the acute (post-procedural) setting. Patients with a chronically hyperdynamic circulation and low SVR are believed to be more predisposed to acute cardiovascular collapse immediately after a TIPS procedure. Up to one year after the procedure, patients are still at risk of cardiac decompensation, but via a different mechanism, which develops more slowly. It is estimated that up to 20% of patients will demonstrate cardiac decompensation post TIPS.[32] As well as a cardiovascular examination, a recent chest X-Ray, ECG and echocardiogram should be reviewed.

Figure 2. Common systemic pathophysiology in the patient with hepatic dysfunction



Ascites and hepatic hydrothorax impact on aspiration risk, respiratory function, and the likelihood of desaturation during induction. Patients who require regular ascitic drainage should ideally have this done prior to proceeding to TIPS. Any large volume paracentesis in the immediate pre-operative period should be paired with albumin replacement to reduce the risk of post-paracentesis circulatory dysfunction and renal impairment. Symptomatic or large volume hepatic hydrothorax may also require drainage pre-operatively.

Encephalopathy should be graded and documented. In an elective setting, moderate to severe encephalopathy may be a contraindication to TIPS due to the risk of worsening symptoms following placement.

There is no widely accepted consensus regarding pre-operative investigations prior to a TIPS procedure. Patient risk factors for bleeding and transfusion requirement, as well as the risk for hepatic or renal decompensation should be noted. A valid group and screen is required. This group of patients may have had repeated blood transfusions, leading to the possibility of red cell antibodies and cross match delays.

When a TIPS is performed in an acute setting, such as massive variceal haemorrhage, complications and mortality rates are significantly increased.[31]

INTRAOPERATIVE

TIPS procedures are performed in the interventional radiology suite. The usual challenges and safety concerns of both remote location anaesthesia and radiation exposure apply.

Limited literature exists around the use of local and sedation versus general anaesthesia for TIPS. Individual patient factors, as well as proceduralist preference will guide decision making regarding the optimal type of anaesthesia for each individual patient. The risk of intraoperative complications after shunt deployment (e.g. cardiac decompensation or pulmonary oedema) are arguably better managed in an anaesthetised, intubated patient.[31] TIPS procedures can be technically challenging and lengthy, with most centres in Australasia performing TIPS under general anaesthesia. Drug choices, venous access and monitoring should be guided by the severity of liver disease, concurrent patient comorbidities and local protocols.

In carefully selected patients, conscious sedation using a combination of propofol and/or remifentanil may be used. The patient must be able to lay flat for a prolonged period and have a low risk of aspiration. Balloon dilatation of the intrahepatic tract is painful and often poorly tolerated by conscious patients. The anaesthetist must be prepared for conversion to general anaesthesia at any point, acknowledging this can be difficult with limited access to the patient in the interventional suite once the procedure has begun.

Premedications, particularly benzodiazepines, should be used with caution due to the possibility of impaired hepatic metabolism and prolonged duration of action. Rapid sequence induction should be considered to mitigate the risk of aspiration in patients with severe ascites or hepatic hydrothorax. Tracheal intubation with paralysis also confers the benefit of controlled ventilation and the ability to perform breath holds.

Large bore intravenous access is recommended, and arterial cannulation should be considered. Although central venous access is not routine, if it is required, it will usually be via the left internal jugular or the femoral veins, depending on the radiologist's planned approach. In actively bleeding patients, rapid infusion devices and point of care coagulation testing should be available.

Suxamethonium is safe in advanced liver disease although it may have a prolonged duration of action. Atracurium and cisatracurium offer more dependable pharmacokinetic profiles due to their organ independent metabolism, but rocuronium and vecuronium can also be used safely. Maintenance of anaesthesia with either volatile or propofol TIVA is appropriate.[31] Dose reduction of propofol may be necessary secondary to an increased sensitivity. Fentanyl and remifentanil are the opioids of choice for this group as their metabolism remains largely unchanged in severe liver disease. Opioid use should be minimised where possible, and long-acting agents should be avoided.[33] Ketamine, clonidine, dexmedetomidine and gabapentinoids should be used with care due to their impaired metabolism and side effect profiles. Residual sedation will cloud the assessment of encephalopathy and should be a factor for consideration in anaesthetic planning. Broad spectrum antibiotics should be given in accordance with local guidelines.

The anaesthetist must be prepared to manage several potential intra-operative complications including massive haemorrhage, cardiac decompensation, and pulmonary oedema. Venous air embolism is a risk, particularly at the time of jugular sheath removal and in the spontaneously breathing patient. In the absence of complications, most patients are appropriate for extubation at the end of the procedure. Attention should be paid to the avoidance of coughing and straining on emergence, given the recent removal of a large jugular access sheath and the risk of bleeding and neck haematoma. If there is persisting haemodynamic instability, respiratory concerns, or evidence of acute cardiac or neurological deterioration, admission to the intensive care unit for haemodynamic and ventilatory support may be required.

POSTOPERATIVE

Postoperatively, there is potential for multisystem dysfunction. TIPS patients should be cared for in a monitored setting with ongoing specialist hepatology input. Patients with cardiac decompensation may require diuresis. In extubated patients, postoperative CPAP may be beneficial if there is evidence of pulmonary oedema. Encephalopathy occurs in up to 20% of patients following TIPS. Acute post TIPS hepatic encephalopathy occurs due to a rapid increase in ammonia levels with associated cerebral oedema. Conversely, chronic/late post TIPS encephalopathy does not present with cerebral oedema. Encephalopathy is generally managed medically; with a low protein diet and lactulose or rifaximin to reduce intestinal neurotoxin production and absorption. In refractory encephalopathy, the TIPS may need to be revised, reduced in size or re-occluded.[34]

Monitoring for worsening hepatorenal syndrome, contrast induced nephropathy and haemolytic anaemia is necessary. Shunt patency is checked using ultrasound, usually within one week post TIPS, and then for ongoing surveillance thereafter.

Post TIPS, close attention should be given to signs of developing sepsis and a low threshold exists for initiation of antibiotics. Responsible pathogens are typically *Escherichia coli, Klebsiella, enterococcus and Streptococcus.* Early identification and initiation of antibiotics to reduce deterioration in organ function is important.

CONCLUSION

Patients with advanced chronic liver disease are increasingly encountered by the general anaesthetist for interventions relating to their disease complications. Understanding the pathophysiology of portal hypertension, the perioperative interventions these patients often present for, the steps to consider for optimisation, and the potential pitfalls of each procedure are key in improving perioperative outcomes and effectively managing acute events for this high-risk group of patients.

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