Review Article

Anaesthesia for biliary atresia and hepatectomy in paediatrics

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ABSTRACT

The scope of this article precludes an 'in depth' description of all liver problems and I will limit this review to anaesthesia for biliary atresia — a common hepatic problem in the very young — and partial hepatectomy in older children. I will not be discussing the problems of anaesthetising children with hepatitis, cirrhosis, congenital storage diseases or liver failure. Extrahepatic biliary obstruction is an obliterative cholangiopathy of infancy which is fatal if untreated. Diagnosis involves exclusion of other causes of neonatal jaundice and treatment involves a hepatico portoenterostomy carried out at the earliest. This is a review of current concepts in anaesthesia and postoperative management of neonates with extrahepatic biliary atresia. Anaesthesia for hepatic resection has seen great changes in recent times with the improvement in surgical techniques, technology and a better understanding of the underlying physiology. These are reviewed along with the problems of postoperative pain management.

Key words: Anaesthesia, biliary atresia, hepatectomy, paediatrics, pain relief

BILIARY ATRESIA

Biliary atresia is an obliterative cholangiopathy of infancy that is fatal if untreated.

It is characterized by a lack of gross patency of the extra hepatic bile duct of which there are three main types. Type I atresia of the common bile duct; Type II with additional atresia of the common hepatic duct but residual patency of the right and left ducts; and Type III with atresia of the whole of the extrahepatic duct system. If left untreated it leads to worsening cholestasis, hepatic fibrosis, cirrhosis, portal hypertension and liver failure.[1,2]

Though biliary atresia is considered a congenital lesion, it has dynamic properties in that the biliary structures gradually disappear by 2-4 months and are replaced by fibrous tissue. Kasai[3] suggested that survival rates are better if the infant is operated on before 60 days of age. Kelly and Davenport[4] suggest 30 days of age. However, others believe that survival is more closely related to the severity of intrahepatic cholangiopathy.[5]

Clinically, biliary atresia presents in infants from 1-6 weeks of age with jaundice, pale stools, dark urine, coagulopathy, failure to thrive, hepatosplenomegaly and ascites.[6] There is a rise in conjugated bilirubin, alkaline phosphatase and gamma glutamyl transferase. Prothrombin and albumin are normal in the early phase.[4] About 50% are anicteric until the 2nd or 3rd week of life.[7] Liver function is usually fairly well preserved in the first few months of life. These properties change as the child gets older and fibrosis extends into liver tissue. Characteristically, there is worsening cholestasis, fibrosis and cirrhosis which lead to portal hypertension and a decrease in synthetic function. Other abnormalities present are associated with embryological development like polysplenia, cardiac defects, situs inversus, anomalous hepatic artery, absent IVC and intestinal malrotation.[2,8]

The differential diagnosis of persistent 'pathological jaundice' in the infant includes neonatal hepatitis, intrahepatic biliary hypoplasia, choledochal cyst, inspissated bile syndrome and extra hepatic biliary atresia (EHBA).[2]

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Diagnosis by abdominal ultrasound shows an enlarged liver and an absent or contracted gall bladder after 4 h fasting.[4] This is confirmed by liver biopsy or an intraoperative cholangiogram with visual assessment of the biliary tree.[9]

Medical management is by preventing cholangitis with low dose oral antibiotics (amoxicillin, cephalexin or trimethoprim), improvement of bile flow with ursodeoxycholic acid and corticosteroids (controversial) and nutritional support with oral fat soluble vitamins and high caloric protein feeds.[4,9]

Surgical palliation which remains the standard of care for first line intervention is achieved by portoenterostomy (Kasai's procedure).[2,10] The surgical procedure involves three stages: 1) mobilisation of the liver and dissection of the atretic bile ducts at the porta hepatis with the removal of all remnants of the bile tracts upto the capsule of the liver; 2) preparation of a Roux-en-y loop of jejunum; and 3) anastomosis of the end of the Roux-en-y loop to the area prepared at the porta hepatis. This is then used as the conduit for bile drainage.[2]

Predictive factors[11] in the success of Kasai's procedure include a direct bilirubin less than 2.0, the absence of bridging liver fibrosis[12] and the number of episodes of cholangitis after surgery. Cholinesterase levels can be an early indicator of protein synthesis by the liver independent of albumin synthesis,[13] Short and long term outcomes of hepatoportoenterostomy are closely related to the experience of the surgeon and the center.[4]

Combined therapy of early portoenterostomy and hepatic transplantation, where cure is not possible, has greatly improved the prognosis for infants with EHBA with 5 year survival, now exceeding 90% in some series.[2,4]

Complications of surgical repair and the underlying disease states include cholangitis, portal hypertension and fat soluble vitamin deficiency.[14] This is especially important when considering the patient who returns to the OT for revision of biliary drainage, intra-abdominal sepsis and relief of intestinal obstruction. Due to these problems liver transplantation is being considered more frequently as a primary option.[7]

Preoperative preparation:[2,4,9] Continue medical management as detailed above, Vit K IM for at least 4 days oral neomycin and metronidazole for 24 h at 4-h intervals, Hb and clotting factors are checked, one unit of blood <5 days old cross matched and kept available, clear fluids administered orally for 24 h and the patient starved for 4 h prior to surgery. Atropine 0.05-0.1 mg are given 30-60 min before surgery.

Anaesthesia for babies with biliary atresia follows the same general principles of anaesthesia as for neonates and infants.[7] The degree of liver dysfunction and the drug's ability to bind to plasma proteins are important variables in determining drug kinetics in patients with liver disease.

If there is an IV cannula *in situ*, Inj. propofol 2-3 mg/kg and atracurium may be used. In those without a venous access inhalational anaesthesia with oxygen/nitrous oxide and sevoflurane may be used, an IV cannula inserted, atracurium given and the baby intubated and ventilated. (Insertion of an IV cannula may prove difficult due to previous venepuncture attempts in the early diagnostic period. In that case a 5 Fr triple lumen catheter may be placed centrally). The inhalational agent may then be changed to isoflurane/(air and oxygen) as hepatic blood flow and oxygen supply are better maintained. Nitrous oxide is avoided at this stage to avoid troublesome bowel distension. A nasogastric tube also helps to decompress the stomach.

Maintenance crystalloid solutions must contain dextrose so as to avoid hypoglycaemia. Maintenance fluids should contain at least 1% dextrose increasing to 5% as required. Blood sugar estimations should be done at least after induction and at the end of the surgery. Replacement may be with Ringer lactate. Unlike in adults with obstructive jaundice the use of diuretics or volume loading is not required for prevention of renal failure because this virtually does not occur in this age group.[2] Blood loss is assessed by swab weight and suction loss. Generally, there is very little blood loss and blood replacement is not necessary. However, warm blood is replaced as soon as the loss exceeds 10% of the estimated blood volume. Ascites is not usual but if present 4.5% albumin in 0.9% sodium chloride should be used to replace losses. Urine output is not usually easy to assess in this age group.

Monitoring is usually with an ECG, pulse oximeter, noninvasive blood pressure and temperature (both core and peripheral). Ventilation should be controlled and end tidal gases monitored for oxygen, carbon dioxide and volatile anaesthetic agents.

In general, haemodynamic stability is maintained without the need for vaso active substances. There is usually no need for invasive arterial or venous monitoring unless the baby has associated sepsis, pneumonia, cholangitis or severe cirrhosis.[7] However, one must watch out for decreasing venous return, and therefore decreased cardiac output, if there is compression or kinking of the inferior vena cava.

Hypothermia is a very real problem as in addition to the large surface to volume ratio in infants and their lack of insulation, a large surface of the liver is often exposed to the cold operating room. OT temperatures should be kept higher than normal, IV fluids warmed, all cleaning solutions warmed and hot air blowers used to maintain surface warmth.

Elective postop ventilation is considered in children with other organ system failures (specifically sepsis, cholangitis and pneumonia), those who are cold at the end of the procedure (<35°C) or those who have undergone a transfusion of more than one blood volume.

Intravenous antibiotics are given for 5 days post op to prevent ascending cholangitis which can lead to significant morbidity. Wong *et al*. suggests the use of meropenem as an effective first line empirical antibiotic.[15]

POST OP ANALGESIA

Morphine is now the agent of choice at the King's College Hospital.[2] It is made up as weight (in kg) in 50 ml saline and then administered at the rate of 0.5-2 ml/h to a max of 1 mg/kg/day. However, as these children are extubated and breathing spontaneously great care must be taken to avoid respiratory depression.

The Boston group advocate the use of epidural analgesia concluding that it is safe providing that coagulopathy is corrected or absent and close post op monitoring and pain management is available. Lower doses of local anaesthetic infusion are advocated in view of the risk of increased unbound bupivacaine levels in neonates. Epidural analgesia is effective when properly placed and closely monitored. However, a number of complications have been reported with epidurals. They include bradycardia, ventricular and atrial ectopics, transient apnoea, leakage around the epidural catheter, relocation of the catheter position and necessity to abandon the epidural.[16]

LIVER TUMOURS AND HEPATIC RESECTION

Liver tumours are uncommon in children but 72% of those that occur are malignant. Of these the most common are hepatoblastomas and hepatocarcinomas. Unlike in adults, metastatic tumours only account for 10% of cases.[17] Neuro endocrine tumours, though rare, are notorious for their intraluminal venous extension and may have to be removed on cardiopulmonary bypass.[18]

Hepatoblastomas[7] usually occur in low birth weight babies below the age of 2 years. The presenting symptom is an abdominal mass that is not usually associated with ascitis, anaemia or jaundice. Liver functions are often normal. They are often associated with isosexual precocity as a result of the liver's production of ectopic gonadotrophic hormone, Beckwith Wiedemann's syndrome, polyposis coli, Wilm's tumour and fetal alcohol syndrome. Survival rates are up to 90% if the histology type is foetal and about 50% if it is embryonal. Other types with poorer survival rates are macrotubular and anaplastic.

Hepatocellular carcinoma[7] occurs in two age peaks of below 4 years and between 12-15 years. The survival rates in both groups are poor at 5-10%. Patients present with weight loss, jaundice, fever and lethargy. Only about 5% have cirrhosis. Other associated diseases include von Gierke's disease, type I glycogenesis, cystinosis, extrahepatic biliary atresia, hypoplasia of intrahepatic bile ducts, Wilson's disease, giant cell hepatitis and Solo's syndrome. Many of these children would have been subjected to chemotherapy and the protocol should be reviewed prior to surgery. Adriamycin and anthracycline are often used and these are associated with dose dependent irreversible cardiomyopathy. They will require special tests to assess cardiotoxicty and cardiac reserve, like history, physical examination, ECG, chest X-ray and echocardiogram.

Anatomic hepatic resection is dependent on the segmental infrastructure of the liver and saves a considerable amount of normal liver tissue. Non-anatomic resection is independent of structural planes and excessive bleeding is a common problem.[19] In children residual liver is often normal unlike in adults, whose liver is often cirrhotic. However, patients with pre-existing liver disease often present with coagulation defects, acidosis, electrolyte disturbances and hypoglycaemia. Pulmonary

function may be impaired due to the presence of shunts, or diaphragmatic splinting due to ascites and hepatomegaly.[17] They should be specifically assessed for gas exchange impairment, nutrition associated cardiomyopathy, infection, cirrhosis, decompensation and kidney impairment. The response of hypoxemia to 100% oxygen should be measured to assess the degree of shunt as well as an assessment for post op weaning of ventilation.[20]

The influence of anaesthetic techniques and drugs on liver function after liver resection has not been studied. However, the pharmacokinetics of fentanyl and sufentanil are not altered nor is liver function responsible for the elimination of remifentanil.[20] Theoretically, muscle relaxants used should be those not metabolised or eliminated by the liver. However, a review of literature shows that the most commonly used muscle relaxants in hepatic resection are rocuronium and vecuronium.

Sevoflurane is the inhalational agent of choice for induction and isoflurane or desflurane are 'marginally preferred' for maintenance.[17] All three are preferable to halothane as they cause less decrease in splanchnic and hepatic blood flow. Also hepatitis is believed to be more common with halothane due to its increased metabolism — 20% as compared to 2-5% of sevoflurane, 0.2-0.6% of isoflurane and 0.02% of desflurane.[17] Though there seems to be little evidence to support the avoidance of halothane on the grounds of hepatitis risk certain precautions should be taken such as not using halothane within 3 months of the first exposure. Unexplained jaundice and pyrexia after its first use should also preclude further use. Nitrous oxide has an additive effect on depressing cardiac function when used with other volatile agents.[21]

There is a potential for massive blood loss in these children and invasive monitoring with intra-arterial blood pressure (IABP) and central venous pressure (CVP) with a Foleys catheter for measuring urine output are considered mandatory. Pulmonary artery catheterisation is rarely required.[20] Trans-oesophageal echocardiography may be used to assess cardiac function and volume status if a multifactorial problem arises.[20]

Intra operative blood loss has been correlated to high CVP and venous congestion. Therefore, intravenous fluid replacement should be restricted as far as the haemodynamics allow until parenchymal resection is completed.[20,22] Maintaining a CVP of less than 5 cms of H_2O has been shown to reduce bleeding, thus decreasing the need for blood transfusion, prolonged dissection, prolonged portal occlusion time and increase in the hepatic ischemic reperfusion injury.[22] However, any intraoperative decrease in systemic arterial blood pressure (SAP) should be treated aggressively to preserve liver blood flow and post-operative impairment of liver function.

The recording of CVP may, however, prove fallacious as it is much less reliable during liver resection. CVP may increase during surgery when the pressure of surgical retractors on the diaphragm raises the intra thoracic pressure or compression on the liver releases a significant amount of blood into the circulation.[23] Clamping of liver vessels decreases venous return to the heart and thus the CVP.[23] This may also occur when the liver is mobilised and the vena cava and the portal vein may get compressed or twisted. The potential for air embolism, while maintaining a low CVP, during liver dissection is significant. A negative CVP can rapidly allow large volumes of air to be entrained through small, unrecognised lacerations of the hepatic veins. The presence of an air embolus may be diagnosed by a sudden fall in EtCO2, hypoxemia, hypercarbia, fall in SAP and rise in pulmonary artery pressure (PAP).[24] This problem has been minimised by newer techniques of surgery and the maintenance of CVP between 3 and 5 cms of H_2O.

Should massive bleeding occur, the major criteria for blood volume expansion are the adequacy of systemic arterial pressure, expired carbon dioxide plus the adequacy of the wave form of the peripheral pulse oximetry curve.[23]

Massive blood volume replacement with volume expanders, blood and blood products may create physiological derangements requiring close monitoring and correction of arterial blood gases, acid base balance, electrolytes, coagulation profiles and tissue oxygenation. Haemodynamic instability can be accentuated during surgical manipulation.[17,20] Lower haemoglobin values are well tolerated and restricting intraoperative fluids to decrease haemodilution may contribute to a reduction in the need for blood transfusion.[25,26]

Liver vessel occlusion may be resorted to decrease blood inflow to the liver. This may be done by clamping of the vascular triad and more rarely with total vascular occlusion. In these cases ionotropes, vasopressors

and vasodilators must be used appropriately.[20] Intermittent clamping of the portal vascular triad is better tolerated than prolonged continuous periods of ischemia.[20]

Antibiotics[20] are given pre- and intra-operatively because of long operating times, large postsurgery dead spaces and inevitable areas of dead tissue Translocation of intestinal enterobacteria to the systemic circulation and peritoneal inoculation is seen after major liver resection. The drug recommended is cefazoline. Biliary obstruction is a potential source of sepsis and piperacillin with tazobactam (or sulbactam) combined with an aminoglycoside or a fluroquinolone are recommended.

Hypothermia is commonly associated with this surgery and adequate warming during and after surgery is important.

Post op ventilation should be considered in children who are cold, have had massive transfusions, still have some acid base or coagulation abnormalities and have other associated anomalies.

Postoperative ascites always develops at the expense of circulating fluid and occurs whether the liver is healthy or diseased. Intra and postoperative fluid limitation does not prevent this ascites and volume expansion with diuretics and vasopressor therapy should be initiated early to prevent kidney failure.[20]

POST OP PAIN RELIEF

Many reports suggest that a combination of general anaesthesia and epidural analgesia can provide good operating conditions and decrease the requirement for blood transfusions.[27,28] However, liver surgery may cause postoperative liver disturbances even in patients with normal preoperative coagulation functions undergoing uncomplicated hepatectomy. The extent of liver resection appears to affect the magnitude and duration of the coagulation defect with the PT and platelets showing an inverse relationship to the amount of hepatic tissue resected.[29] Matot *et al*.[30] found that PT returned to normal within a day in minor resections but took 5-7 days in uncomplicated major resections necessitating the use of FFP prior to the removal of the epidural catheters. PT and platelets should therefore be monitored post-operatively and the patient observed closely for early signs of cord compression (spinal haematoma).[27,31]

If epidural is the analgesic method of choice improvement in analgesic management should focus on three phases where 'analgesic gaps' may occur, namely, transition from the OT to the immediate post-operative care and transition from epidural to systemic analgesics.[32,33]

Morphine infusions have been used successfully with close monitoring of sedation, respiratory depression and hepatotoxicity.[20,33] Acetaminophen reduces morphine requirements and can be used cautiously for a limited period of time after major hepatic resection.[20] However, the nonspecific clinical features associated with acetaminophen like nausea, vomiting, jaundice, dizziness, elevated transaminases and moderate kidney impairment may be masked after major abdominal surgery.[20] NSAID's are better avoided as they worsen pre-existing poor renal function especially in the presence of cirrhosis.

Right thoracic paravertebral blocks have been used in managing post-hepatectomy pain in adults[34] but there are no reports of their use in paediatrics.[30] Perhaps they may be used in the older children.

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Announcement

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