36 Intensive Care Issues in Post-operative Pediatric Liver Transplantation

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36.1 Introduction

Pediatric liver transplantation (LT) has become a standard and definitive treatment for end stage liver disease over last few decades. Advances in pre-transplant care, operative techniques, and post-operative intensive care have led to consistent improved survival and better outcomes. Although the pre-operative condition of patient has an important role in affecting outcome, yet it is not the sole determinant [1]. LT can be successful even in critically ill patients with appropriate supportive care. In more recent years, sicker patients with complicated comorbidities are undergoing LT. Recent addition of certain metabolic diseases as an indication for LT makes the post-operative care even more complex and challenging. This underlines the larger and more crucial role of intensive care management of these children peri-operatively. Pre-existing liver dysfunction associated comorbidities such as hepatopulmonary syndrome, hepatorenal syndrome, etc. along with post-transplant variables such as allograft dysfunction, infection, and surgical issues makes them an unique cohort which requires certain level of expertise to be handled in intensive care. Careful monitoring, anticipation of problems, and pre-emptive action are essential components to post-LT intensive care management for successful outcomes.

36.2 General Principles

The anesthetist dealing with the child during the liver transplant (LT) operation should accompany the patient to the intensive care unit and a detailed handover should be given to the pediatric intensivist. Handover details should cover all the important intra-operative events such as blood loss, ionotropic requirement, and change in ventilatory requirements when the abdomen is closed, etc. In the liver intensive care unit (ICU) continuous monitoring of vital parameters such as arterial blood pressure (ABP), electrocardiogram (ECG), peripheral oxygen saturation (SpO2), central venous pressure (CVP), and temperature is continued. Some units do additional monitoring with NIRS (near infrared spectroscopy), ScvO2, and SjvO2 monitoring. Bedside ultrasound (USG) and echocardiography are now a routine practice in most of the liver ICU.

Head end elevation, 30–45°, is preferred in normotensive patients. All drains and catheters (nasogastric, bladder, intra-abdominal, biliary, etc.) must be emptied initially and amounts noted. Subsequently hourly output should be measured

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V. Vohra et al. (eds.), *Peri-operative Anesthetic Management in Liver Transplantation*,
https://doi.org/10.1007/978-981-19-6045-1_36

and recorded. Drain output is usually replaced on hourly basis. In addition the colour and consistency of drains is monitored to assess the intra abdominal bleeding. Chest radiograph is performed in all patients to check endotracheal tube position (if intubated) and for assessing lung condition (like collapse, effusion, consolidation etc.). Lung imaging may be repeated if required and regular bedside lung USG (no radiation exposure) can also be performed to assist in diagnosing and decision making. Laboratory investigations include arterial blood gas, complete blood count, coagulation profile (prothrombin time PT/activated partial thromboplastin time PTT, thromboelastogram TEG, fibrinogen), liver and renal function tests, and serum electrolytes (sodium Na, potassium K, magnesium Mg, calcium Ca, chloride Cl, and phosphorous P). Blood investigations are typically repeated 6–12 hourly depending on patient condition. Culture surveillance (blood, urine, endotracheal, and drain fluid) are usually sent depending on institutional protocol. USG Doppler for hepatic vessels and portal vein flow, as a standard should be done daily in children for up to 1 week post-operatively. In infants and small children, some units do twice daily USG Doppler for early detection of vascular issues.

Short-acting, non-hepatotoxic medications are preferred for analgo-sedation during the duration of ventilation (propofol, fentanyl, etc.). Prophylactic antibiotic and antifungal therapy (as per institution protocol) and immunosuppressive therapy are administered. Anticoagulation in the form of either intravenous heparin infusion or low molecular weight subcutaneous heparin may be initiated in high risk patients (small size artery, Budd-Chiari or previous hypercoagulopathy, etc.), with close monitoring of coagulation and clinical parameters to avoid bleeding.

36.3 Post-op Ventilation and Oxygenation

Lung protective strategy should be generally adopted with tidal volumes 6–8 ml/kg to target a plateau pressure < 30 cm H_2O. High positive end expiratory pressure (PEEP) (>10 cm H_2O) can elevate intrathoracic pressures and impair venous return form inferior vena cava and hepatic veins resulting in graft congestion and loss. As a rule extubation is done as early as possible provided biochemical parameters and hemodynamics are stable, with a good liver vessel doppler study. However, the decision to extubate has to be individualized based on pre-operative comorbidities (such as encephalopathy, hepatopulmonary syndrome, etc.) or post-LT complications (such as graft dysfunction, fluid over load, etc.).

Pediatric patients on an average require <48 h of mechanical ventilation post-LT [2]. In selected subgroup of stable patients, tracheal extubation is occasionally feasible immediately at the end of surgical procedure (fast tracking) [3]. Few adult studies have shown that adequate gas exchange can be maintained in cases of early extubation (in O.R or within 3 h of surgery) with no significant difference in the rate of reintubation [3]. However, optimal selection criteria for candidates for early extubation in pediatric LT are not well defined. Also, it is not correct to extrapolate results from a center which advocates fast tracking to any another due to differences in pre-operative condition, surgical skill and duration, graft size and abdominal status, and other factors [4]. Early extubation is generally more feasible in the older children compared to the younger ones and infants [5].

There is an emerging important role for non-invasive ventilation (NIV) and heated humidified high flow nasal cannula (HHHFNC), post-LT. By providing PEEP, NIV is useful to prevent lung atelectasis post-extubation, especially in children with compliant chest walls. This also allows for earlier and safer extubation subsequently leading to lower rates of pneumonia and sepsis post-LT [6].

36.4 Fluids and Hemodynamics

For the liver graft to function well, it is important to maintain euvolemic state during immediate post-transplant period. Though input–output chart during surgical procedure gives a rough idea on fluid status, it is really difficult to assess

the exact status particularly in small infants as insensible and abdominal fluid losses cannot be judged accurately. Several parameters such as heart rate, inferior vena cava (IVC) filling, urine output, post-transplant weight, and CVP are used in combination to access the intravascular volume status. The ultrasonic cardiac output monitors (USCOM) are a more reliable tool in accessing fluid status in such scenario [7].

Hypovolemia can lead to impaired hepatic perfusion, whereas excessive fluids can lead to venous congestion in allograft and can cause graft loss. The pre-existing liver disease induced vasodilatory state, myocardial dysfunction, etc. may resolve slowly over few days post-LT; this may also contribute to hypotension and can explain the need for continued minimal vasopressor/inotrope support in such cases. Dyselectrolytemia, metabolic derangements, reperfusion syndrome, and primary graft dysfunction can all lead to postoperative hemodynamic instability.

In cases of hypovolemia, careful controlled fluid resuscitation should be carried out, with meticulous and continuous reassessment. Synthetic colloids have been demonstrated to be as effective as albumin for fluid resuscitation [8]. Norepinephrine is the usual vasopressor of choice in pediatric post-LT patients. In cases of myocardial dysfunction, dobutamine is conventionally used. Hypertension has also been described in 20–70% patients of pediatric patients undergoing LT [9, 10]. This occurs due to interplay of multiple variables: high volume status, steroid, calcineurin inhibitor, and high renin levels. Medications used to treat hypertensive crisis include sodium nitroprusside infusion or calcium channel blockers initially. Angiotensin converting enzyme inhibitors and beta blockers are subsequently added as needed. Hypertension must be well controlled in the post-LT patient as there is higher risk of bleeding due to the presence of coagulopathy/thrombocytopenia in these patients.

36.5 Electrolytes and Metabolic Issues

Lactate in post-LT patient have a special role. In addition to diagnosing and treating shock, lactate is a good surrogate marker of good graft function during immediate post-transplant period. The lactate reaches peak during anhepatic phase of surgery and once the new allograft is implanted the lactate should start showing a declining trend. Persistent hyperlactatemia or progressive increase after surgery is a marker for graft dysfunction. Usual maintenance fluid in children is 5% or 10% dextrose with additives. This is to provide at least 4–6 mg of glucose/min. In case of hyperglycemia, rather than decreasing the dextrose concentration, providing adequate glucose along with insulin infusion should be carried out. This hyperglycemia can be due to transient insulin resistance during postoperative period. On the other hand, persistent hypoglycemia should alert the possibility of graft dysfunction.

Most of the children with chronic liver disease (CLD) would be having long standing hyponatremia before surgery. This may require low sodium fluids during post-LT period, to avoid rapid increase in serum sodium levels. Regular electrolyte monitoring is essential. Hypocalcemia may be present, especially if a patient has received massive amounts of blood products (citrate-induced). This needs to be corrected. Hypokalemia and hypomagnesemia may also occur and have to monitored regularly and should be appropriately supplemented.

36.6 Pulmonary Issues

1. Pleural effusion: Pleural effusions are very common post-LT (around 30%) and seen most often on the right side [4]. This pleural fluid collection is usually reactionary, and is due to movement of ascitic fluid across the diaphragm. Fluid collection also occurs due to surgical

handling and placement of a foreign graft tissue below the diaphragm. Most of these effusions resolve spontaneously with careful restriction of volume intake and/or cautious diuretic therapy. Chest tube insertion is rarely needed; only if respiratory compromise occurs.

2. Atelectasis: This is a unique concern, especially in the smaller children, which can delay extubation or lead to respiratory distress post-extubation. Patients who are malnourished or have abdominal distension have added risk. Regular chest physiotherapy post-LT plays both a preventive as well as therapeutic role to achieve lung expansion.
3. Pulmonary edema: This can occur due to fluid overload and/or myocardial dysfunction. Careful attention to volume status and fluid restriction is the key to prevent this.
4. Ventilator induced pneumonia (VAP): prolonged post-op ventilation could predispose to VAP. Strict asepsis, implementation of VAP bundle, early extubation, non-invasive ventilation, and regular physiotherapy helps in reducing the risk.
5. Acute respiratory distress syndrome (ARDS): The mechanism for development of ARDS post-LT are manifold. These include transfusion related acute lung injury (TRALI), severe reperfusion injury, and infection induced ARDS. Low tidal volume with high PEEP strategy is the dictum for ventilator strategy in ARDS, the same is applicable to the post-LT scenario. Inhaled nitric oxide may be used in cases of refractory hypoxemia. Role of HFOV and prone ventilation is unclear, but if indicated it is used.
6. Right hemidiaphragm paralysis due to phrenic nerve injury is a rare complication which may hamper weaning process [11].

36.7 Post-operative Hematological Issues: Bleeding and Coagulopathy

Most of the children who undergoes LT are coagulopathic. Due to the usage of various blood products during the surgery, PT/International normalized ratio (INR) measured at 4–6 h after surgery would reflect the true coagulation profile. Coagulation is also used as marker of graft functionality. During immediate post-operative period PT/INR is monitored on regular interval to check that it is improving. In case of progressive worsening of INR, fibrinogen and platelets are to be checked and are usually corrected before giving fresh frozen plasma (FFP). While correcting coagulopathy risk of bleeding must be carefully balanced against risk of thrombosis of hepatic vessels. Overcorrection of coagulopathy and thrombocytopenia should be avoided. Thromboelastography (TEG) is useful to guide the type and amount of blood component transfusion needed along with PT/PTT, fibrinogen, and platelet counts [12]. The usual target levels are INR 1.5–2, fibrinogen >100 mg/dl and platelet >50,000/mcL [13]. But these target parameters are not the rule always, clinical examination and surgical team inputs are necessary before giving blood products.

Thrombocytopenia is commonly seen post-LT and may persist for about 2 weeks: it occurs due to platelet activation and consumption following reperfusion and hypersplenism. Some centers advocate target threshold of <20,000/mcL along with active bleeding to transfuse platelets [14]. Monitoring of serial blood Hb levels along with drain Hb level and drain amount will help to identify active ongoing bleeding. Imperfectly achieved surgical hemostasis (e.g.: slippage of surgical knot), hypocalcemia, and thrombocytopenia can cause postsurgical bleed. In cases of persistent or increasing bleeding, re-exploration is indicated. Intra-operative use of recombinant factor VII has been shown to control profuse bleeding without increasing rate of thrombotic events. Restrictive transfusion strategy is advocated as it has been shown to improve patient outcomes [15].

36.8 Gastrointestinal (GI) Concerns

1. Ileus: Post-operative ileus may occur due to the use of analgesia, intra-operative gut handling, dyselectrolytemia (e.g.: hypokalemia),

and occasionally sepsis. In patients with biliary atresia with kasai portoenterostomy, LT procedure is complicated by bowel adhesions, thereby increased risk of perforation in such patients. Bowel perforation during post-LT period may be silent with absence of classical signs such as rigidity, fever, etc. due to immunosuppressive drugs. In cases of clinical suspicion (persistent abdominal pain, increasing gastric aspirates, and abdominal distension) further evaluation with imaging (abdominal CT) is essential and abdominal re-exploration would be required in some cases. Elevated abdominal drain amylases and visibly dirty abdominal drain could give clue towards intestinal perforation.

2. Gastrointestinal (GI) bleeding: Stress-induced and steroid induced gastroduodenal ulceration can lead to upper GI bleeding post-LT. Massive upper GI bleed is usually from esophageal varix secondary to portal vein (PV) thrombosis, hence urgent evaluation by endoscopy and ultrasound doppler must be done. Any lower GI bleed or hematochezia requires CT angiography to look at bleeding site in small and large bowel. The management consists of cessation of antiplatelet drugs and anticoagulants. PT/aPTT, platelet count, fibrinogen need to be evaluated and blood product support needs to be given according to laboratory and clinical parameters. Upper GI endoscopy is essential in cases of significant bleeding; it has both diagnostic and therapeutic value.
3. Intra-abdominal hypertension: Intra-abdominal hypertension can occur occasionally in the smaller babies, where graft size is large, leading to a tight closure. This is detrimental to liver perfusion and may also lead to progressive shock and organ dysfunction, particularly kidneys. There must a high alert for this complication whenever large grafts are used, and occasionally the abdomen is left open and temporary closure devices are used in order to prevent this complication.

36.9 Neurological Issues

Children who were ventilated due to encephalopathy prior to LT, neurological recovery might be slow as the cerebral oedema takes time to resolve. Seizures during post-LT period is not uncommon and could occur in around 30% cases [15]. Etiology for seizures during post-LT would include dyselectrolytemia, hypertension, calcineurin inhibitor toxicity, posterior reversible encephalopathy syndrome (PRES), acute cerebral infarctions, hemorrhages, continue myelinolysis (CPM), [9, 16] etc. Though serum electrolytes levels might be normal, rapid fluctuations in serum electrolytes due to multiple colloids and crystalloids are also implicated as important risk factor. Screening CT of brain to be performed in all children with seizure to rule out any bleed or vascular event. Self-limiting brief seizures usually does not require treatment. For recurrent seizures or a single seizure with potentially epileptogenic abnormalities on brain imaging or EEG, antiepileptic drug (AED) therapy should be initiated. Levetiracetam is the drug of choice for post-LT seizures due to lack of significant hepatic metabolism or drug interactions. AEDs can usually be discontinued after 1–3 months, provided the child is seizure-free and EEG is normal. Other neurological disturbances such as headache, confusion, hallucinations, tremor, and speech apraxia can be seen due to adverse effects of immunosuppressive drugs.

36.10 Acute Kidney Injury (AKI)

AKI during post-LT is multi-factorial. Pretransplant status (severe liver dysfunction, shock, hepatorenal syndrome), intra-operative hemodynamic instability, prolonged use of vasoconstrictors, massive blood transfusions, post-op use of immunosuppressive agents (tacrolimus, cyclosporine), antibiotics (aminoglycosides, colistin), and antifungals (amphotericin B) with nephro-

toxic side effects could all contribute towards AKI. Hemodynamic-mediated AKI generally resolves spontaneously. Several renoprotective agents (dopamine, prostaglandins) have been studied but none have been proven to be effective to prevent post-LT (AKI) [17]. In case of worsening AKI: reducing/withholding tacrolimus should be considered. Alternative agents like sirolimus, and/or mycophenolate mofetil can be given for immunosuppression [18]. Whenever feasible nephrotoxic drugs like aminoglycosides, colistin, and amphoterin B must be avoided. When AKI is advanced causing fluid and metabolic issues in critically sick children, continuous renal replacement therapy (CRRT) is the preferred modality of management.

36.11 Immunosuppression

Calcineurin inhibitors (CNIs) particularly tacrolimus forms the mainstay of immunosuppression after LT. It is a potentially nephrotoxic agent and has many undesirable side effects. Tacrolimus doses are adjusted by drug level monitoring. Of particular note is the drug interaction between fluconazole and tacrolimus. Azole antifungals inhibit the metabolism of tacrolimus mediated by CYP3A4. Steroid are used as adjutants during immediate post-transplant period and gradually weaned in 3–6 months. Different institutes have their own policy/protocol on steroid weaning. Other drugs which are used include mycophenolate mofetil (B & T cell proliferation inhibitor, renal sparing, no drug level monitoring needed), cyclosporine (T helper cell inhibitor), sirolimus (IL-2 transduction inhibitor), and IL2 receptor blocking antibodies (Daclizumab, basiliximab). Detailed discussion of immunosuppression is beyond the scope of this article. Table 36.1 enlists the side effect profile of the commonly used agents. Research is ongoing to develop new immunosuppressive protocols which are renal sparing and steroid free.

Table 36.1 Common drugs used in liver transplant and their side effect profile

Drug	Side effects
Glucocorticoids	Hypertension Dyselectrolytemia Gastroduodenal ulceration Mood disturbances Fluid retention Hyperglycemia Pancreatitis
Tacrolimus	Hypertension Nephrotoxicity Hyperglycemia Seizures and neurological symptoms (tremor, headache, etc.)
Cyclosporine	Hypertension Neurological symptoms (seizure, headache, confusion) Nephrotoxicity Dyselectrolytemia Gingival hyperplasia
Mycophenolate mofetil	Anemia, thrombocytopenia, leukopenia Hypertension Dyselectrolytemia Myopathy Tachycardia
Sirolimus	Anemia, thrombocytopenia, leukopenia Hyperlipidemia Poor wound healing

36.12 Infections

Post-LT patients are at high risk for infection. Pre-operative liver dysfunction and malnutrition along with the use of post-operative immunosuppression and presence of multiple devices (central lines, drains, etc.) in situ make them prone for acquiring infections. Post-transplant prophylactic antibiotics are given for 5 days and are usually stopped. Antifungal prophylaxis (oral fluconazole) is given as a institutes protocol for 2–3 weeks or until patient is discharged from hospital. If the recipient is cytomegalovirus (CMV) negative and the donor is CMV positive, treatment with intravenous ganciclovir is given for at least 2 weeks. Clinical signs of sepsis may

be subtle due to steroids/immunosuppression. Laboratory septic parameters must be closely followed for development of leukocytosis/leukopenia and positive cultures; and focus of infection must be sought. Prompt treatment with appropriate broad spectrum or specific antibiotics/antifungal is an essential component.

36.13 Nutrition

Nutritional support is an important part of post-LT care. Pre-operative malnutrition, surgical stress, and steroid administration all contribute to increased nutritional demand post-op. Pre-operative malnutrition is associated with higher risk of post-LT infections and longer ICU stay. Caloric intake should be aimed at 120–130% of calculated basal energy expenditure and protein intake of 1.5–2 g/kg must be provided [19]. Early enteral nutrition has been shown to be beneficial and causes lesser metabolic issues and decreased infection rates [20]. Since most of the infants with LT would have hepaticojejunostomy immediate enteral nutrition might not be practically possible. In such children with malnutrition, total or partial parenteral nutrition is recommended from post-op day one until 50–70% enteral feeds are achieved. Replacing abdominal drain loss with intravenous 5% albumin is another strategy to prevent protein loss.

36.14 Early Post-operative Complications Specific to Liver Graft

36.14.1 Primary Graft Failure

This is a dreaded complication of LT where the new liver graft fails to function. Though the exact reason is unclear, many factors such as advanced donor age, prolonged ischemic time (>18 h), >30% macrosteatosis, and reperfusion injury have been implicated [21]. Liver dysfunction is severe (transaminases usually >5000 U/L) and hypoglycaemia and coagulopathy occur. Management is mainly supportive: high concentration dextrose infusion, regular FFP transfusion, and appropriate neuroprotective measures and renal support are given. Prostaglandin E1 and N-acetyl cysteine infusion have been used across various centers, however, results have not been consistent. If there is no graft recovery in 24–36 h, emergency re-transplantation is indicated, without which progressive multiorgan dysfunction and eventually death results.

36.14.2 Size Discrepancy of Graft

The ideal graft should be 0.8–2% of recipient body weight. Size discrepancy is problematic in both ways—both large and small sized grafts have their own set of issues. When Graft to recipient weight ratio (GRWR) < 0.8% (small for size) congestion of the graft may occur due to high portal flow resulting in delayed synthetic liver function, cholestasis and increased susceptibility for infections. However, this is not common in the small-sized pediatric patient. Large for size transplants (GRWR > 4) even with use of split/reduced grafts can occur especially in patients <10 kg [22]. Abdominal closure in these cases may be tight or not completely possible. Higher intrabdominal pressures may lead to abdominal compartment syndrome and compromised blood perfusion to the new liver graft. In some cases full abdominal closure is not possible; and temporary abdominal mesh/bogota bag need to be placed followed by closure after 2 weeks [22].

36.14.3 Rejection

Acute rejection can occur at any time after LT but is common in first 5–10 days post-LT. Clinical manifestations may be absent or subtle in mild to moderate rejection, while in severe rejection non-specific symptoms such as fever, abdominal pain, etc. can be present. Any rise in transaminases above base line should raise the possibility of rejection. Liver biopsy is essential for confirmation and will show lymphocytic infiltrate in the portal space. Duct and endothelial damage can be seen and in severe cases hepatocyte destruction occurs. Adjustment in baseline immunosuppressive drug doses along with pulse dose steroids

usually suffices as treatment [20]. Rejection must be recognized and managed early, as it is detrimental for graft survival. A close differential is a sepsis, differentiating this needs careful interpretation of clinical and laboratory parameters.

36.14.4 Vascular Issues

Hepatic artery thrombosis (HAT): This is a potentially fatal complication with an incidence of 1.5–25% in children [23]. It is more common in children compared to adults and more so in living donor transplants as the anastomosis is between smaller sized vessels. Sensitivity of doppler ultrasound to detect HAT can be 100% and should be performed daily for the first week post-LT. CT arteriography is confirmatory when vessel cannot be identified on ultrasound. After detection, urgent thrombectomy is indicated either by surgical or radiological interventional technique [24]. Failure to detect or intervene in cases of HAT leads to rapid graft necrosis and death. Urgent retransplantation is indicated if revascularization of hepatic artery is not achieved. Prophylactic low molecular weight heparin, antiplatelet drugs (aspirin), systemic anticoagulation (in high risk cases) are used based on institution-specific protocols. HAT and biliary complications are interlinked as the biliary tract is almost exclusively supplied by hepatic artery.

Portal vein thrombosis (PVT): Clinical manifestations include worsening signs of portal hypertension (GI bleeding due to varices, increasing ascites, encephalopathy) and worsening liver function tests. The diagnosis is by doppler ultrasound. Early thrombosis is usually successfully relieved by surgical/radiological interventional procedure or by emergency shunt surgery to relieve the portal hypertension. Late thrombosis are usually persistent and tend to get compensated over time.

36.14.5 Biliary Issues

These are known as the "Achilles heel" of LT and are more frequent in living donor transplantation [25]. Bile leak can occur due to faulty surgical anastomosis, necrosis or biliary tract ischemia. Cut surface leak can also occur especially in case of split/partial grafts. Bile leaks usually occur towards the end of the first week post-LT. They can present as fever, bilious abdominal drain, increased gamma-glutamyl transferase (GGT), and leukocytosis. Biliary leak increases the risk of fungal sepsis. While in older children who had duct to duct anastomosis; ERCP and stenting can help while in small children with hepaticojejunostomy, usually surgical intervention is required to detect and repair site of leak. Biliary strictures may occur late in the post-LT course which are treated by percutaneous/endoscopic dilatation or surgical re-exploration.

36.15 Conclusion

Post-LT intensive care management involves anticipation of problems and pre-emptive intervention. Essential components of PICU care include hemodynamic stabilization, early extubation, appropriate fluid-electrolyte therapy, strict asepsis, early recognition, and appropriate management of sepsis, AKI, and organ dysfunction. Unique aspects of monitoring in post-LT children include monitoring graft function and flow in hepatic vasculature. Prompt identification and treatment of complications is the key to reduce mortality and morbidity post-LT.

Key Points

- Post-operative intensive care in pediatric liver transplantation is complex and challenging.
- Optimal fluid management and hemodynamics are essential for liver graft functioning and successful outcomes.
- Early extubation must be targeted; NIV is an emerging useful adjunct to prevent pulmonary complications and allow early extubation.
- Thorough knowledge of graft and vascular issues are essential for anticipation and timely management of complications which are unique to liver transplantation.

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