Liver transplantation (LT) is now a standard treatment for children with end-stage liver disease with excellent 1- and 5-year survival. This has been achieved through improvement of surgical techniques and anti-rejection treatment and management. The donor pool for children has been extended by the use of cut-down, split, living-related and, recently, non-heart-beating donor and isolated hepatocyte transplantation. Though the majority of transplanted children enjoy an excellent quality of life, there remain a high number of possible complications, including short-term primary non-function, vascular and biliary problems, bowel perforation, severe rejection, infection, hypertension and long-term renal impairment, chronic rejection, de novo autoimmunity, lymphoproliferative disease and cancer, most of which are related to anti-rejection drug toxicity. Hence, the focus of research for paediatric LT should be induction of tolerance, avoiding long-term immunosuppression and its toxicity.

1. Introduction

Over 40 years, liver transplantation (LT) has evolved from an experimental procedure to a standard treatment for children with end-stage liver disease. Early efforts at LT resulted in significant intraoperative and post-operative mortality. Despite advances in surgical techniques, one-year survivals remained poor throughout the 1970s, being only 30% in 1978. The advent of cyclosporin in 1981 produced a marked increase in graft and patient survival and reduced the need for long-term use of high dose steroids with their attendant growth-suppression. Newer immunosuppressive drugs, enhanced organ preservation and better donor management have contributed to improving outcome. Paradoxically, however, the success of LT, leading to a widening of its indications, resulted in a shortage of organs for children. To increase the donor pool, new surgical procedures to cut down adult livers to fit small children were introduced. Early referral, improved pre-LT nutritional support and increasing experience in managing immunosuppression and its complications have also contributed to ameliorating outcomes. Current one-year survival of children undergoing LT for chronic liver disease is 80-90%, the majority enjoying a good quality of life.

2. Indications (Table 1)

All children with life-threatening liver disorders should be considered for LT. These include decompensated chronic liver disease, acute liver failure, non-cirrhotic liver-based metabolic disorders and liver tumours.

2.1. Chronic liver disease

LT should be considered in children with end-stage liver disease and a predicted survival of <1 year or a very poor quality of life. Impaired synthetic function, disordered metabolism, portal hypertension, lethargy, intractable pruritus are indications for transplantation [1]. Timing of LT is important. Too early, it jeopardizes
the child’s life unnecessarily; too late, the chances of success are reduced. A number of factors, including aetiology of underlying liver disease, patient’s age, quality of life, growth retardation, increased hepatic artery resistance index and past medical/surgical history, influence the timing of transplantation. Biliary atresia, if hepatoportoenterostomy fails, is the most common indication for LT, accounting for half of the cases. Genetic disorders resulting in cirrhosis, including α1-antitrypsin deficiency, progressive familial intrahepatic cholestasis, Wilson disease and cystic fibrosis constitute the second most common group.

2.2. Acute liver failure

Acute liver failure is rare, but associated with high mortality [2]. The commonest cause is cryptogenic, which may be associated with subsequent bone marrow failure. In Western countries, hepatitis A, B or E are rarely implicated. Drugs associated with liver failure are acetaminophen (inadvertent overdose or homicide), anti-tuberculous/anti-epileptic agents, non-steroidal anti-inflammatory agents, halothane and ecstasy in adolescents. Direct toxic injury occurs with Amanita phalloides ingestion. Other potential causes include Wilson disease, tyrosinaemia type 1, autoimmune hepatitis and neonatal haemochromatosis. Poor prognosis is predicted by younger age, severe coagulopathy, presence of encephalopathy, severe metabolic acidosis, cardiovascular instability, a rapidly shrinking liver and the presence of renal failure. An international normalised prothrombin ratio (INR) >4 carries a mortality of >80% without LT. One-year survival is worse than for LT for chronic liver disease, being 75–80% [3].

2.3. Non-cirrhotic liver-based metabolic disorders

Metabolic conditions that are life threatening because of the lack of an essential enzyme, but are not accompanied by liver disease, may be treated by LT. Some of these metabolic disorders can be treated by auxiliary liver transplant [4] or hepatocyte transplantation [5–8].

2.4. Liver tumours

LT has a limited role in primary malignant liver tumours of childhood that cannot be successfully treated by subtotal hepatectomy and chemotherapy. Excellent results have been obtained in hepatoblastoma responsive to chemotherapy [9] while hepatocellular carcinoma usually recurs, unless small or an incidental finding at the time of LT.

3. Contraindications

There are few absolute contraindications to LT, including overwhelming bacterial, fungal, or viral infection outside the liver, severe cardiovascular disease, extrahepatic malignancy and inherited diseases with multisystem involvement, like mitochondrial disorders.
4. Operative techniques

Liver grafts come from living or cadaveric donors, either brain stem dead, heart-beating, or non-heart-beating (NHBD). Though the latter have been rarely used in children, the initial experience of successful NHBD segmental LT is promising [10]. Donor and recipient should be ABO blood group compatible, but preferably ABO matched. An appropriate size-match is also preferred, unless the liver is suitable for size reduction. There is no upper age limit for liver donation, but donors >50 years of age are rarely accepted. Absolute contraindications to organ donation are malignancy (excluding primary cerebral tumours), active systemic infection, hepatitis B and HIV infection, chronic liver disease and Creutzfeldt–Jakob disease. Donors should have satisfactory liver function tests and negative virology for hepatitis A, B, C and HIV. Cytomegalovirus (CMV) antibodies are tested to decide whether anti-CMV prophylaxis in CMV negative recipients is needed post-LT.

4.1. Orthotopic liver replacement

The recipient hepatectomy may be difficult in children who have undergone previous surgery such as biliary atresia, because of the presence of adhesions and portal hypertension. The portal vein is anastomosed end-to-end, while the donor hepatic artery is usually anastomosed to the recipient common hepatic artery. If the native artery is small or frail, an infra-renal donor iliac conduit may be used to re-arterialize the graft. Biliary drainage is established either by primary end-to-end anastomosis or by Roux-en-Y hepatico-jejunostomy.

4.2. Liver reduction

Reduction techniques are based on the segmental anatomy of the liver [11] and can be used to produce three types of liver graft of varying size: left lateral segment, left lobe and right lobe. The extent of reduction is determined by visual comparison between the donor liver and the recipient hepatic fossa. Transplantation of the left lateral segment involves resecting the recipient’s native liver off the inferior vena cava which is left in situ. The graft is then piggybacked onto the inferior vena cava by anastomosing the left hepatic vein to the common orifice of the hepatic veins using a triangulation technique to reduce the risk of outflow problems (Fig. 1).

4.3. Split

Familiarity with the techniques of liver reduction has led to splitting the organ between two recipients: the left lateral segment for a child, the right lobe for an adult. Results of 80 paediatric split transplants in our unit show a 93% patient and 89% graft actuarial 1-year survival [12]. Livers can be split in situ and ex situ, both techniques giving comparable graft and patient outcomes in experienced hands.

4.4. Living donor liver transplantation (LDLT)

LDLT was developed simultaneously in Japan and Chicago in the late 1980s [13,14]. The technique involves the resection of the left lateral segment of the liver of a parent and transplanting it into their child. The reported donor mortality is 0.5% in adult to adult LDLT, the risk being smaller in case of left lateral segment donation, and donor morbidity is 5%. LDLT offers clear benefits: it can be performed as an elective procedure, avoiding prolonged waiting times; the donor is carefully assessed to ensure a compatible size and blood group match; living donor grafts have an excellent early graft function and the overall hospital stay is shorter than for cadaveric transplants. Surprisingly, there is no immunological advantage over cadaveric grafts, as the incidence of acute rejection is similar. The reported graft and patient one-year survival is 89% and 92% [15]. LDLT programs have developed within transplant centres that are experienced in both segmental liver transplantation and hepatobiliary surgery and follow an ethical protocol regarding donor and recipient selection, informed consent and audit of results. LDLT is an option for suitable families but should represent a second-line choice in countries where cadaveric split organs are available. Paediatric LDLT is used for acute liver failure when a cadaveric graft cannot be found in time, in severely decompensated chronic liver disease or in hepatoblastoma.
post-chemotherapy, when choosing the optimal time point for transplantation is vital [16,17].

5. Rejection

5.1. Acute rejection

Almost half of the paediatric population experience at least one episode of acute rejection. In the majority of children, rejection is associated with elevated serum aminotransferase and \( \gamma \)-glutamyltranspeptidase or alkaline phosphatase levels whereas only half of the children show elevated serum bilirubin. Fever is a feature in 1/3 of rejection episodes. Risk factors include recipient age, ethnicity of recipients and baseline immunosuppression [18]. The lowest rate of rejection is seen in children <6 months of age and the highest in teenagers who suffer from poor compliance with their medication. The use of tacrolimus is associated with lower rates of rejection when compared to cyclosporin and it is now used for primary immunosuppression in most centres [19].

Tacrolimus may be used as single agent after withdrawal of steroids one-year post-LT, to allow optimal growth. Acute rejection is treated with 3-day high dose intravenous methylprednisolone (10–20 mg/kg/day), followed by weaning doses of oral prednisolone. Steroid-resistant rejection, previously an immediate indication for the use of anti-lymphocyte preparations, is effectively treated with chimeric or humanised IL-2 receptor monoclonal antibodies [20]. Recurrent acute rejection is treated by adding mycophenolate mofetil or rapamycin [21]. Induction therapy with IL-2 receptor antibodies can further reduce the rate of acute rejection and has been used effectively as a steroid sparing immunosuppressive regimen [22,23].

5.2. Chronic rejection

Risk factors for chronic rejection include younger age, steroid-resistant rejection, ethnicity of recipient, CMV infection, transplantation for autoimmune disease, occurrence of post-transplant lymphoproliferative disease, HLA match/mismatch and positive lymphocytotoxic crossmatching [24]. In paediatric LT its incidence has decreased from 10% to <5% and the Pittsburgh group has reported the absence of chronic rejection in children receiving tacrolimus-based immunosuppression, provided baseline immunosuppression is maintained [25]. Chronic rejection can occur as early as 6 weeks post-LT but is usually most common in the first year. The clinical presentation is with jaundice and pruritus. Rescue strategies for chronic rejection are evolving as new immunosuppressants become available. The first step in our unit is to add mycophenolate mofetil to tacrolimus, followed by anti-IL2-receptor monoclonal antibodies and then rapamycin in non-responsive patients. Non-response to medical management requires re-transplantation.

6. Complications

6.1. Primary non-function (PNF)

PNF occurs in <5% of patients (0–16%) [26]. The incidence of PNF is lower after living donor and in situ split liver transplantation, where cold ischemia and reperfusion injuries are minimised [27]. It results in graft loss, with only one-third of children with PNF surviving [28]. Several factors may contribute to PNF, including cause of donor death, time in intensive care before donation, organ preservation and retrieval as well as problems in the recipient due to technical complications or hyperacute rejection.

6.2. Vascular complications

Hepatic artery thrombosis (HAT) is a serious complication, as the transplanted liver is particularly dependent on an intact arterial inflow, all potential collaterals having been divided. The reported incidence is 3% in adults and 7–8% in children [29]. It may present insidiously, with fever, cholangitis or biliary leaks, strictures and abscesses. Risk factors for HAT include underlying prothrombotic disorders, elevated haematocrit, severe acute rejection with increased hepatic arterial resistance and prolonged cold preservation time. Surgical factors include small vessel size, particularly with whole grafts, intimal dissection and faulty technique. The diagnosis is suspected on routine Doppler ultrasonography and confirmed by three-dimensional multislice computed tomographic angiography [30]. With an aggressive imaging policy and the selective use of retransplantation, revascularisation, and conservative treatment, >80% of children survive HAT; 40% survive without needing retransplantation due to the development of a sufficient collateral arterial supply which sustains liver and biliary tree. Hepatic artery stenosis occurs in 5–10% of the cases and can be successfully managed with angioplasty or stenting by an experienced interventional radiologist.

Portal vein thrombosis (PVT), rare in adults, occurs in up to 33% of paediatric liver transplant recipients [31]. Risk factors include hypoplastic portal vein, the use of whole liver grafts, haemoconcentration, hypercoagulability, severe acute rejection and splenectomy. It presents with INR prolongation, persistent metabolic acidosis and, in severe cases, hypertransaminasaemia. Urgent re-exploration and revision of the portal vein anastomosis usually rescues the graft.

The risk of early and late PVT or stenosis after LDLT is significantly higher than for reduced size cadaveric transplantation (33% versus 4%) [32], presumably
through the stretching of a short length portal reconstruction after regeneration and remodeling of the graft. Use of cadaveric cryopreserved donor iliac or femoral vein is associated to 50% late PVT [33]. In patients with extrahepatic PVT and a healthy liver graft with patent intrahepatic portal system, the Roux loop (mesenteric-o-left portal bypass) is the treatment of choice [34].

6.3. Caval obstruction

Caval complications are rare and usually due to technical problems. Suprahepatic caval stenosis presents with lower trunk and leg edema and signs of portal hypertension with ascites, renal impairment, graft dysfunction and splenomegaly. Doppler ultrasound and cavography with pressure measurements reveal a significant gradient across the stenosis confirming the diagnosis. Percutaneous venous angioplasty and stenting may lead to a dramatic resolution. Late caval complications occur infrequently but their incidence increases with the use of the piggyback technique [33].

6.4. Biliary

Biliary complications are common in paediatric recipients (5–30%). In particular, a third of children who undergo living-related left lateral segment liver transplants develop biliary complications [35]. Bile leaks occur in the early post-operative period from the biliary anastomosis, the cut surface of a partial graft, from unrecognised segmental ducts or following removal of T-tube [36]. Patients present with fever or mild graft dysfunction, and, if undiagnosed, progress to biliary peritonitis. Endoscopic or percutaneous cholangiography and stenting lead to resolution and avoid the need for surgical reconstruction in the majority of cases. Anastomotic biliary strictures occur in 10% of patients, usually within 12 months of transplant and present with cholangitis or obstructive jaundice, but may be asymptomatic. Non-anastomotic biliary strictures are relatively rare. HAT is the cause of 25% of all biliary complications and should be excluded in all cases. Besides HAT, intrahepatic ischemic cholangiopathy can follow prolonged cold ischemia, transplantation with ABO incompatible blood groups and LT from non-heart-beating donors. Though some patients can be managed conservatively, the majority need retransplantation. Complications of the Roux loop hepatico-jejunostomy occur in approximately 5% of cases and include bile leak or stricture. Surgical revision is usually required though early cases may respond to percutaneous transhepatic dilatation.

6.5. Bowel perforation

Bowel perforation is an uncommon (6%) complication following LT. Contributory factors include previous operations, steroid therapy, viral infection, malnutrition and lymphoproliferative disease. The incidence is higher (15%) in children who have undergone transplantation for biliary atresia after Kasai portoenterostomy [37].

6.6. Infection

Infectious complications are important causes of morbidity and mortality in the first 3 months post-transplant, when immunosuppression is at its highest. Children with chronic liver problems considered for LT should undergo all available vaccinations before listing to prevent infection after surgery. Risk factors for infection include calcineurin inhibitors (tacrolimus and cyclosporin), steroids, poor graft function, prolonged intensive care, ventilator dependence, gut perforation, retransplantation and the use of anti-lymphocyte antibodies to treat severe rejection [38]. Bacterial infections are common during the first two weeks post-LT, while later problems are community acquired or opportunistic infections [39]. Gram-positive organisms from venous lines remain an important cause of sepsis in the first post-operative week, while Gram-negative sepsis is less common with the use of prophylactic antibiotics during and after surgery. Increasing problems, however, are being encountered with antibiotic-resistant organisms such as Klebsiella and Enterococcus. The presence of Gram-negative organisms and Candida species in the peritoneal fluid post-operatively suggests bowel perforation or biliary leak. Risk factors for fungal sepsis include graft dysfunction, HAT, bile leak, bowel perforation, reintubation and ALF (where it affects up to 40% of patients). Most fungal infections are due to Candida species but Aspergillus, Mucormycosis, Coccidioidomycosis and Cryptococcus may also occur and are associated with high mortality. Fungal sepsis should be suspected in any transplant patient with fever and high white blood count whilst receiving broad-spectrum antibiotics. Fluconazole is well tolerated as prophylaxis and therapy, but lipoosomal amphotericin is the mainstay of treatment. Itraconazole is effective for invasive aspergillosis and voriconazole is used for Candida species resistant to fluconazole. Herpes Simplex and Zoster, CMV, EBV and Adenovirus are all potential causes of early and late infections, often associated with over-immunosuppression. Age governs the clinical expression of infection with CMV and Epstein–Barr virus (EBV). Young patients, more likely to be seronegative for these viruses pre-LT, are susceptible to primary infections post-LT, which are particularly severe in the context of immunosuppression. Seventy percent of children develop primary CMV infection post-LT with a mortality of 7% [40]. Over-immunosuppressed patients, in particular those receiving anti-lymphocyte antibodies for severe rejection, have a particularly high risk of developing CMV disease [41].
Gancyclovir, however, in association to reduced immunosuppression, has dramatically improved the prognosis of this life-threatening condition. Primary EBV infection post-LT is common. Its most severe consequence is the development of EBV-driven post-transplant lymphoproliferative disease (PTLD). The mean time to presentation of PTLD after surgery is 264 days and affected patients are usually those who have received higher levels of immunosuppression [42]. Rarely adenovirus infection causes fulminant hepatitis or necrotizing pneumonitis in the early post-LT period, with 45% mortality [43]. Parvovirus B19 infection affects 1–2% solid-organ recipients during the first-year post-transplant. The most common symptom is anemia, but leukopenia and thrombocytopenia are also described. Intravenous immunoglobulin produces rapid improvement in most cases [44].

6.7. Immunosuppression

A major problem after LT is the toxicity of anti-rejection drugs. Children require proportionally more immunosuppression than adults, particularly during the first year post-LT, and, so far, they face life-long treatment, so that the complications of liver disease are swapped with those of immunosuppression. Tacrolimus and cyclosporin have similar side effects, but the latter is associated with more hypertension, hirsutism, and gingival hyperplasia, whereas tacrolimus is associated with more neurotoxicity, pruritus, and insomnia. Neurotoxicity is exacerbated by low serum magnesium levels.

6.8. Renal complications

Renal insufficiency immediately post-LT is less common in children than in adults, who usually have a higher incidence of renal dysfunction pre-LT. Some children, however, have a degree of renal impairment related to their underlying disease, e.g. tyrosinaemia, congenital polycystic disease, Alagille syndrome. Hepatorenal syndrome in association with severe liver dysfunction is reversed by successful liver transplantation. Acute tubular necrosis, particularly of ischaemic origin, is responsible for almost half of acute renal failure cases post-LT. Aminoglycosides and volume depletion account for most of the remainder. Long-term nephrotoxicity is almost exclusively secondary to the use of calcineurin inhibitors, which induce vasoconstriction of the renal vasculature. Children suffer a progressive decrease in glomerular filtration rate with a fall of 20–50% in over half of the cases 2–4 years post-LT. Both tacrolimus and cyclosporin have similar nephrotoxic effects. Unlike cyclosporin, however, liver graft function affects the plasma profile of tacrolimus. With poor allograft function the levels of tacrolimus metabolites accumulate in the plasma, increasing the nephrotoxic risk [45]. At present, there are no non-nephrotoxic drugs that can replace cyclosporin or tacrolimus in the early phase post-LT, but a reduction in dose and toxicity is allowed by new agents, including anti-IL2-receptor monoclonal antibodies, mycophenolate mofetil and rapamycin. The latter two, in selected individuals, may completely replace cyclosporin and tacrolimus, providing nephrotoxicity-free immunosuppression.

6.9. Hypertension

Fifty to 80% of adult patients develop significant systemic hypertension following liver transplantation [46]. The number of paediatric patients with chronic hypertension is lower but this complication represents an important long-term management problem that may contribute to renal impairment. Hypertension often occurs within a few weeks of starting calcineurin inhibitors and steroids. The first-line treatment includes dietary sodium restriction and a reduction in steroid and calcineurin inhibitor levels, followed by the use of calcium-blocking agents. In a study, 87% of transplanted children required anti-hypertensive therapy during their initial hospital stay and 50% required it after discharge [47].

6.10. De novo autoimmune hepatitis

In 1998 de novo autoimmune hepatitis after LT was described for the first time in children [48]. This condition affects patients transplanted for disorders other than autoimmune hepatitis. Several papers [49] have since confirmed the occurrence of this condition in both children and adults transplanted for non-autoimmune disorders. Early diagnosis is crucial as treatment with prednisolone and azathioprine at the doses used for classical autoimmune hepatitis, if initiated promptly, is graft and life saving. The pathogenesis is unknown, but may be related to an increase in circulating autoaggressive T lymphocytes due to the use of calcineurin inhibitors [49].

6.11. Haematological complications

Thrombocytopenia after liver transplantation occurs frequently. It may be due to several causes, including hypersplenism, bleeding, disseminated intravascular coagulation, septicaemia, or the intrahepatic deposition of platelets [50]. Severe thrombocytopenia may also develop with a sudden onset at a later stage after transplantation. In such cases, drug-induced thrombocytopenia or an immunologically mediated destruction of platelets such as that seen in idiopathic thrombocytopenia (ITP) should be considered. Tacrolimus-related thrombotic thrombocytopenic purpura has been reported [51]. The diagnosis of ITP is based on sudden onset, increase in number of megakaryocytes in the bone marrow and elevation of platelet-associated IgG (PAIgG). In general, acute ITP in childhood is considered
to be associated with a viral infection. Two cases of virus-associated ITP after liver transplantation, one due to varicella zoster virus and the other to human parvovirus B19, have been reported [52,53]. In case of severe thrombocytopenia, treatment with intravenous γ-globulin may induce a prompt increase in the platelet count [54]. Acute ITP is usually a benign disease, where mortality and haemorrhagic complications are relatively rare with most children achieving spontaneous remission [55].

Immune-mediated haemolytic anemia in the transplant setting may be alloimmune or autoimmune. Alloimmune haemolytic anemia tends to occur within the first few weeks following transplant in an ABO-compatible, but non-identical, transplant recipient. Should transfusions be required in the early post-transplant period, donor ABO blood group should be used to avoid the passenger lymphocyte syndrome, which can cause severe haemolysis.

Autoimmune haemolytic anemia (AIHA) is a rare cause for haemolytic anemia after transplant and can be mediated by warm-reacting IgG antibodies or cold-reacting IgM antibodies that also bind complement (cold AIHA). Altered T cell immunity appears to play a role, as observed in bone-marrow transplant and solid-organ transplant patients on calcineurin inhibitors. Tacrolimus in particular has been associated with AIHA in liver transplant recipients [56]. Often, the toxicity associated with calcineurin inhibitors is dose dependent and may resolve by decreasing the drug dose, or switching to another drug [57].

Most cases of de novo cold AIHA in children resolve spontaneously and no treatment is necessary. A number of therapeutic strategies have been utilised for the treatment of severe, newly diagnosed, cold AIHA, including plasmapheresis and anti-CD20 monoclonal antibody [58,59].

6.12. Post-LT malignancies

Tumour occurrence on immunosuppression remains a major concern after LT [60]. Several mechanisms have been proposed for the development of post-LT malignancies, including the inability of a depressed immune system to destroy malignant cells, direct DNA damage from drugs such as azathioprine and calcineurin inhibitors and the oncogenic potential of viral infections including EBV (PTLD), Herpes and Papilloma viruses (carcinomas of skin, lips, vulva and perineum), and CMV (Kaposi’s sarcoma). Over 50% of tumours are PTLD. Exposure to sunlight is a risk factor for skin cancers and should be avoided.

6.13. Lymphoproliferative disorders

PTLD affects 5–15% of children post-LT. Most PTLDs are associated with EBV infection [61]. Primary EBV infection post-LT occurs in 90% of PTLDs in children, most of whom are EBV negative pre-LT, primary EBV infection being a greater risk factor for the development of PTLD than reactivation [62]. Another risk factor is the intensity of immunosuppression. Most PTLDs are non-Hodgkin’s lymphomas (93% compared to 65% in the non-transplant population). The majority are B-cell, but T cell tumours account for 14%, while null cell for <1%. The initial treatment is reduction of immunosuppression. Acyclovir and ganciclovir are often used, but there is no evidence that either is effective. Recently, rituximab (anti-CD20 monoclonal antibody) and human leucocyte antigen-matched EBV-specific T-cell therapy have been used with success [63,64]. As reduction of immunosuppression leads to rejection, it is often difficult to balance the treatments of PTLD and rejection. Under these circumstances and when PTLD is overtly malignant, chemotherapy is required [65].

6.14. Late mortality

Causes of late mortality in children are usually graft-related and include infections, PTLD, chronic rejection and non-adherence. The latter is less common with tacrolimus-based treatment [66].
7. Survival and quality of life

Both patient and graft survivals after paediatric LT have improved progressively over the years (Fig. 2). Most post-LT deaths, usually related to infection, occur early. Quality of life on the other hand is subjective and difficult to measure, especially in children, though it has been reported from poor to superior in various studies [67–69]. Quality of life depends on good graft function and absence of complications requiring hospital admission.

8. Conclusion

The initial obstacles to survival, particularly organ preservation, surgical technique and immunosuppression have been successfully addressed, but the psychological, social and health problems produced by successful LT in children are only beginning to be recognised. Their life expectancy and future problems are still unknown. Many will have progressive liver damage probably requiring re-transplantation [70]. The focus of research for paediatric LT should be induction of tolerance, avoiding long-term immunosuppression and its toxicity.

References


