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Review

Nutritional Management for Infants and Children Pre and Post-Liver Transplant

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Abstract

Background: Infants and children undergoing liver transplant require ongoing nutritional evaluation throughout the pre and post-transplant period. The pathophysiologic causes of chronic liver disease and acute liver failure are varied, and each present different and unique nutritional challenges.

Methods: A review of the literature and Seattle Children's established guidelines for nutritional management of pediatric liver transplant patients was conducted.

Results: We present guidelines of care to optimize nutrition status of infants and children in the pre and post-transplant phases.

Conclusion: While malnutrition is common in the child with liver failure, it is also treatable when approached with a multidisciplinary focus on identification of clinical symptoms.

Keywords
Pediatrics; liver transplant; nutrition

1. Introduction

Liver disease in the infant or child involves irreversible changes in the hepatic structure leading to symptoms including, but not limited to, liver cirrhosis, cholestasis and jaundice, hepatosplenomegaly and ascites, pruritus with disruptions of sleep leading to decreased quality of life, and malnutrition [1]. The degree of malnutrition in the infant or child with liver disease plays a significant role in their health and stability pre-transplant, ability to heal and recover immediately post-transplant, and thrive in the months and years after liver transplant. Malnutrition in chronic liver disease leads to decreased neurocognitive development and growth, and is a predictor of suboptimal outcomes post-transplant, leading to increased morbidity and mortality [2].

A multidisciplinary approach is required to manage the prevention and treatment of malnutrition in the pediatric patient with liver disease requiring transplantation. Optimizing growth and nutrition status before transplant helps to minimize post-transplant complications such as stunting, delayed healing, laboratory abnormalities and disordered eating [1].The causes of liver disease in pediatrics and indications for transplant, nutritional care in the pre and post-transplant settings, and long-term management to avoid complications of poor growth and malnutrition is described here. This article serves as a concise reference for liver transplant teams. We reviewed up to date literature that focused on pre-transplant nutrition and/or post-transplant nutrition.

2. Indications for Liver Transplant in Childhood

The indications for referring and ultimately listing a child for liver transplantation can be condensed to four basic groups, namely acute liver failure, chronic liver failure, unresectable tumor in the liver and liver-based inborn errors of metabolism. In these metabolic conditions, the enzyme defect results in injury to other organs away from the liver. Further injury can be prevented by liver transplantation because the deficient metabolic pathway is located within the liver.

Acute liver failure is defined as loss of liver functions over a period of days to a few weeks, having previously had no evidence of liver disease. This loss of function may manifest clinically as jaundice, bleeding and encephalopathy. Causes include toxicity from medications (most commonly acetaminophen overdose), herbals and other environmental chemicals, viral infections, autoimmune liver disease and, particularly in children, liver failure of unknown cause [3].

Chronic liver disease results from a wide range of congenital and acquired conditions that gradually erode the function of the liver, most often due to progressive fibrosis resulting eventually in cirrhosis. At this stage, portal hypertension with all the potential complications such as encephalopathy, variceal hemorrhage and ascites may occur. Additionally, synthetic function of the liver may fail with worsening jaundice and coagulopathy. Therefore, there are two basic indications for liver transplantation in children with chronic liver disease: i) liver failure, where the

function of the liver deteriorates to a point where death will occur if the liver is not transplanted, and ii) where complications of the disease make the quality of life unacceptable such as growth impairment, gastrointestinal bleeding or severe and uncontrollable itching.

In children, the most common cause of chronic liver failure is biliary atresia, a condition that affects infants in the first two months of life and results in complete obliteration of the biliary system [4, 5]. The precise cause or causes of biliary atresia are not known. An operation originally described by the Japanese surgeon Morio Kasai, a hepatoportoenterostomy, can, if done early enough and by the appropriately skilled team, alleviate or prevent progression of the fibrosis. However, in many infants the operation does not result in good bile flow and cirrhosis ensues. Other causes of chronic liver disease in children include genetic defects such as Alagille syndrome and the various types of progressive familial cholestasis. Autoimmune diseases such as autoimmune hepatitis and primary sclerosing cholangitis primarily affect older children and adolescents. Although liver tumors are far less common than in adults, both malignant and benign tumors of the liver may necessitate transplantations. Unlike in adults where hepatocellular carcinoma associated with chronic liver fibrosis accounts for the majority of malignant tumors going to liver transplant, in children hepatoblastoma is by far the most common indication. Many hepatoblastomas are able to be fully removed with liver resection but a proportion involve too much of the liver to allow for anything other than total hepatectomy and transplant. It is imperative when considering transplantation that there is no extrahepatic metastatic spread or complete clearance of metastases prior to transplantation. There are a variety of other rare tumors that can be managed with transplantation of the liver but detailed discussion is beyond the scope of this chapter.

Lastly, there is a group of conditions where single metabolic pathways in the liver are defective due to genetic mutations [6]. Some result in direct liver injury, such as alpha-1 antitrypsin deficiency and Wilson disease, and evaluation of these patients is based on the considerations for acute or chronic liver disease described above. Other liver-based metabolic defects, however, do not directly damage the liver but lead to injury to other tissues and organs. Liver transplantation has been shown to prevent further injury, although it will not reverse damage already established. For example, neurological injury can occur in urea cycle disorders due to accumulation of ammonia, in organic acidemias, in Maple Syrup Urine Disease (MSUD) because of acidosis and metabolic decompensation and in Crigler-Najjar because of bilirubin encephalopathy. See Table 1 for examples of metabolic liver diseases.

The nutritional assessment and interventions will clearly vary depending on the primary diagnosis and careful monitoring is needed for all infants and children considered for liver transplantation. In addition, both dietitians and medical teams should have the knowledge and expertise to manage such patients optimally, recognizing the importance of nutrition to transplant outcomes.

Table 1 Examples of metabolic liver diseases in children [6].

Liver Organ Damage	**Other Organ Damage**
Alpha 1 Antitrypsin Deficiency	Familial Hypercholesterolemia
Cystic Fibrosis	Maple Syrup Urine Disease
Erythropoietic protoporphyria	Methylmalonic and Propionic Acidemia
Glycogen Storage Disease	Primary Hyperoxaluria type 1
Progressive Familial Intrahepatic Cholestasis	Urea Cycle Disorders
Tyrosinemia	Crigler-Najjar type 1
Wilson Disease	—

3. Pre-Transplant Nutrition Care: Preventing and Resolving Malnutrition in a Multidisciplinary Model

Infants, children and adolescents with end stage chronic liver disease are the patients at greatest risk of malnutrition. The cause is multifactorial, including poor liver synthetic function, decreased appetite, portal hypertensive enteropathy leading to malabsorption, increased metabolic demands, encephalopathy and GI bleeding. Malnutrition is a particular risk to the youngest patients (age less than 2 years), those with cholestasis, and those with significant sequelae of hepatic dysfunction such as ascites and varices (Figure 1A). Malnutrition is associated with poor post-operative outcomes after transplantation, including increased mortality [7] and increased hospital length of stay [8]. Earlier papers focused on pre- & post-transplant survival, but as progress is made in caring for these patients, current research now describes increased morbidity associated with malnutrition and not just mortality [2]. It is important for early identification of malnutrition in infants and children to provide timely intervention, thus optimizing outcomes. Transplant teams are encouraged to optimize linear growth in children pre transplant as evidence indicates this is predictive of post-transplant catch up within the first 2 years [9]. Each patient undergoes a multidisciplinary evaluation, including hepatologist, surgeon, nurse coordinator, pharmacist, social worker, and dietitian; all are stakeholders in optimizing transplant outcomes.

While understanding the disease progression for each diagnosis is important, ultimately nutrition screening by the team transplant dietitian contains the following same principles [1]. Nutritional status is determined with nutrition focused physical assessments, including anthropometrics such as mid-upper arm circumference (MUAC) and triceps skinfold (TSF), as well as biochemical indices. The dietitian has the most comprehensive knowledge of dietary interventions, dietary products available to facilitate the interventions, feeding methods and ongoing monitoring of responses to nutritional intervention. The transplant dietitian leads the nutrition plan of care to preserve or improve growth potential and minimize post-transplant complications regardless of the cause for ESLD.

3.1 Guidelines for Nutrition Support

Maintaining adequate nutrition orally may be challenging for infants and children with organomegaly, ascites, fatigue and malabsorption with progressive liver disease. Enteral nutrition infused continuously via nasogastric (NG) tube is beneficial if recurrent emesis is related to hepatosplenomegaly resulting in decreased ability for the stomach to expand to hold a bolus. For infants, breast milk is preferred if available for all the documented benefits [10]. Formula or fortified breast milk is concentrated to meet calorie goals to a maximum of 30 kcal/oz. A formula high in medium chain triglyceride (MCT) content is preferred (30-70% fat kcal as MCT) in infants and children with cholestasis as short to medium chain fats are less dependent on bile acids for absorption. Please see Table 2 for energy and protein recommendations. At Seattle Children's, our standard is to start at 120kcal/kg. Some malnourished infants need >140-150 kcal/kg [2, 8]. Calorie provision is increased as indicated by growth parameters and anthropometrics. If feeding via a nasogastric tube, providing opportunities for oral stimulation to maintain skills and prevent oral aversion is essential in preparation for post-transplant transition back to oral autonomy.

Table 2 Estimated enteral energy and protein needs in the setting of cholestatic liver disease (general starting guidelines per our institution, with evidence indicating at least 1.3-1.5 x RDA for energy needs) [1, 8].

Age	Energy-Nourished	Energy-Malnourished	Protein
0-6 months	120-140 kcal/kg	>140 kcal/kg	3-4 g/kg
6-12 months	100-120 kcal/kg	120-140 kcal/kg	3-3.5 g/kg
1-2 years	80-100 kcal/kg	100-120 kcal/kg	2.5-3 g/kg
Older children/ teens	BMR x 1.2-1.5	BMR x 1.5-2	2-2.5 g/kg

RDA = Recommended dietary allowance; Kcal/kg = kilocalories per kilogram; g/kg = grams per kilogram; BMR = Basal metabolic rate

Parenteral nutrition (PN) is effective for improving nutrition status while on the transplant waiting list, and even in resolving malnutrition in some patients prior transplant [2, 11, 12]. PN is not favored as a treatment approach at all transplant centers for multiple reasons, such as the cost versus enteral feeds, risk of complications from central line access, complexity of PN orders resulting in opportunity for prescription errors, and PN component shortages requiring rationing or suboptimal substitutions. However, PN has been utilized successfully by transplant centers familiar with outpatient PN management, and this has been the experience at Seattle Children's for infants and toddlers with severe malnutrition not thriving on optimized enteral feeds. Fewer calories are required intravenously and fluids needed to meet goals are generally less than enteral formulas [8]. Thus, parenteral nutrition can also be beneficial in the setting of fluid restriction for ascites management. Refer to Table 3 below for energy, protein and fluid recommendations for this cohort to meet 100% estimated nutrition needs. If a patient requires parenteral nutrition, NG

feeds are discontinued while the NG tube may remain in place for medication administration, but feeds are still offered by mouth to maintain oral skills as discussed above. PN is often maximally concentrated, providing goal energy and protein without compromising fluid status. When at full maintenance, TPN typically provides a glucose infusion rate of 12-14 mg/kg/min over 24 hours, 2.5-3 g/kg protein, and 1 g/kg standard soy based lipid emulsion. Some programs are beginning to use SMOFlipid, containing soy, MCT, olive oil and fish oil, if additional lipid calories are desired, but currently this preparation is not licensed for Pediatric use in the United States.

In older children and teens, we start with the lower range of estimated enteral energy needs since metabolic demand for linear growth decreases with age. Patients with acute liver failure with encephalopathy may be intubated and sedated, altering metabolic demands. Indirect calorimetry studies may be useful if there is concern for under or over-feeding. Protein needs are also individualized in the context of impaired renal function and/or encephalopathy. Caution is advised in under dosing protein to prevent or treat hyperammonemia as this could contribute to catabolism and an overall rise in ammonia from endogenous protein catabolism. We recommend no less than 1.5-2 g/kg for infants and young children and 1 g/kg for older children and teens.

Table 3 Parenteral nutrition energy, protein and fluid goals for malnourished infants with ESLD (General guidelines per our institution).

Age	Energy	Protein	Fluid
0-6 months	90-100 kcal/kg	3-4 g/kg	75-90 mL/kg
6-12 months	75-90 kcal/kg	3-3.5 g/kg	65-75 mL/kg
1-2 years	75 kcal/kg	2-3 g/kg	65-75 mL/kg

3.2 Micronutrients

In patients with cholestasis, fat-soluble vitamin and zinc deficiencies are common. Vitamin E deficiency, which may lead to irreversible neuropathy, stunting, and neurodevelopmental disabilities, necessitates aggressive supplementation with appropriate doses of a water-miscible (Tocopherol polyethylene glycol succinate – TPGS) preparation. Standard vitamin E preparations are so poorly absorbed in the absence of bile flow, that this may be a difficult issue to manage given that many insurance providers refuse to pay for these special forms of vitamin E despite their therapeutic necessity. Trace elements are individually dosed in parenteral nutrition solutions to prevent copper and manganese toxicity. Heavy metals are usually excreted in the bile, and without bile flow accumulate and may be deposited in the basal ganglia resulting in movement disorders [2]. Routine supplementation of chromium is avoided at Seattle Children's given lack of evidence for deficiency with supportive evidence for toxicity in pediatric patients on long term PN [13]. Sodium is not limited in enteral nutrition for infants to ensure adequate nutrition for brain development anticipating losses from potential causes such as emesis or diuretics. However, restriction may be required for children and teens to decrease fluid retention. Potassium restriction may be required pre-transplant if treating ascites with a potassium sparing diuretic, such as spironolactone. However, if a potassium wasting diuretic (loop diuretic or thiazide) is also

being given, serum potassium levels tend to normalize. General recommendations for supplements are outlined in Table 4.

Table 4 Micronutrient recommendations for infants and children with ESLD (general starting guidelines per our institution [1, 2, 13].

Fat soluble vitamins with minerals (MVW, AquADEKs)	Provide 0.5 mL/day, up to 1 mL BID Limit due to risk of Vitamin A toxicity and individually dose other vitamins as needed
Cholecalciferol	Cholecalciferol 5000-10,000 units/day (may increase to 10,000 units BID). Individual dosing of Vitamin D in parenteral nutrition is not available in the US.
Vitamin E using a water miscible preparation (TPGS)	100 units/day, may increase to 100 units BID
Vitamin K (Phytonadione, Menadione):	Enteral: 5 mg/day (may increase to 10 mg/day), IV dosing: <1 year 1 mg, >1 year 1 mg x age in years to max of 10 mg
Trace elements for parenteral nutrition	Individually dose trace elements: Zinc and Selenium start at standard doses Half dose copper and remove manganese Omit chromium
Sodium	Enteral or oral feeds: Not limited for infants. For older children and teens a sodium limit of 2 g/day may be needed. Parenteral nutrition: Limit to 2 mEq/kg
Potassium	Limit or supplement depending on type of diuretic and serum potassium levels.

mL/kg = milliliters per kilogram; mg/day = milligrams per day; mEq/day = milliequivalents per day

4. Post-Transplant Nutrition Support

Nutritional management of infants and children following liver transplant remains critical, with the ultimate goal of resolving or preventing malnutrition to promote wound healing, decrease infection risk, and decrease hospital length of stay. The nutrition plan varies in intensity depending on the phase: immediate post-operatively to time of initial discharge, then stable state for long term follow up.

4.1 Immediate Post-Operative Period

Several factors influence the chosen modality for nutrition support. Chiefly, nutrition status at time of transplant is paramount in estimating nutrition needs. Oral or enteral fed patients nourished at time of transplant may go without nutrition support up to 5 days post operatively. Older children and adolescents often resume adequate oral intake without nutrition support in the peri-operative period as opposed to infants and toddlers who may have developed oral

aversion. Patients on parenteral nutrition pre-transplant will resume parenteral nutrition post-transplant (typically post-op-day 1 when hemodynamically stable), regardless of nutrition status until enteral feeding tolerance is established. Moderate to severely malnourished patients, regardless of nutrition support pre-transplant, will receive parenteral nutrition until enteral feeding tolerance is established. The type of biliary reconstruction is a predictor of timing for initiation of oral or enteral feeds [14]. This is helpful in estimating need and duration for parenteral nutrition (see Table 5)

Table 5 Anticipated days NPO for infants and children status post liver transplant by anastomosis type (our institutional experience, adapted from Texas Children's Hospital Post Liver Transplant Nutrition Guidelines).

Duct to Duct	~2-3 days
Existing Roux-en-Y	~3-5 days
New Roux-en-Y	~5-7 days

Surgical and medical interventions guide nutrition interventions. Frequent need for NPO status (e.g. return to operating room, radiology and ultrasound scans, extubation readiness trials) resulting in <80% of goal nutrition is an indication to extend parenteral nutrition support. Wound issues such as presence of wound vacuum, non-healing wounds or infection, require nutrition assessment every 5-7 days to determine adequacy of nutrition support. It is important to provide additional protein at 1.5-2 x RDA with liberal calorie provision using the upper range of estimated energy needs [15]. Acute kidney injury may occur post liver transplant [16], requiring fluid restriction and influencing ability to meet nutrition goals, especially with feeding intolerance of concentrated formula. Patients requiring continuous renal replacement therapy while awaiting for return of renal function tolerate higher volumes as the circuit can remove fluid to the desired balance. Typically patients will require 0.2 g/kg extra protein. Additional B vitamins are recommended when on renal replacement therapies when unable to take enteral supplements. For parenteral nutrition, vitamins are dosed according to recommendations in Table 6.

Table 6 Parenteral nutrition vitamins for patients on continuous renal replacement therapy post liver transplant [15].

Infants < 6 months	Full dose MVI, additional ascorbic acid 50 mg/day and thiamine 5 mg/day
Infants >6 months	Half dose MVI, additional ascorbic acid 50 mg/day, folic acid 0.5 mg/day and thiamine 5 mg/day
1-11 year old	Half dose MVI, additional ascorbic acid 50 mg/day, folic acid 0.5 mg/day and thiamine 10 mg/day
>11 year old	Half dose MVI, additional ascorbic acid 50 mg/day, folic acid 1 mg/day and thiamine 10 mg/day

4.2 Parenteral Nutrition

Parenteral nutrition is initiated when patients are hemodynamically stable, typically post op day 1 or 2. The initial goal is to prevent catabolism and provide a source of substrates for liver recovery, including glucose, protein and phosphorus [14]. Start with typical day one parenteral nutrition order, as the transplanted liver is new to the patient, and progress per standard advancements to goal. See Table 7 for macronutrient goal guidelines.

Table 7 Parenteral nutrition guidelines post liver transplant (per our institutional guidelines, adapted from ASPEN Table 29-3 p342) [14].

Energy- use lower range for intubated/sedated patients, consider higher range for surgical complications, infection, etc	Infants: 75-90 kcal/kg Older children BMR x 1.2-1.4
Protein- upper range for dialysis or surgical complications	Infants and toddlers 2.5-3.5 g/kg Older children 1.5-2.5
Lipid	Limit to 1 g/kg standard lipids or 2 g/kg for SMOFlipid
Vitamins/Minerals/Trace Elements	Standard TPN MVI for age unless on dialysis- see above Continue with half dose copper and delete manganese even if conjugated bilirubin normalizes, as total body levels may remain elevated. Add additional ascorbic acid and increase zinc if has additional wound healing issues
Fluid	Volume allotted for parenteral nutrition typically provides ~60-80% maintenance initially as drips and medication volumes are included in total fluids.

4.3 Enteral Nutrition

Patients transition to an age/developmentally appropriate diet when ready. Well nourished children not receiving nutrition support pre-transplant often resume a regular diet for age within a few days post-transplant, without need for tube feeding or parenteral nutrition. If tube feeding is needed, a standard formula preparation for age is appropriate for initiation of feeds with appropriate graft function. If graft function is delayed resulting in continued cholestasis, consider resuming pre-transplant formula for the high MCT content. Formula may be concentrated up to 30 kcal/oz for infants if needed for fluid restriction. Infants commonly have hyperkalemia due to Tacrolimus, therefor may require a low potassium formula until serum potassium stabilizes. Breast milk is preferred if available as potassium content is low. Children often receive 30 kcal/oz ready to feed formula. Teenagers tolerate higher calorie concentrations as needed, such as 45 kcal/oz. See Table 8 below for additional enteral recommendations. Bedside swallow evaluations are completed for infants and children at Seattle Children's that appear unsafe for oral feeds or have developed oral aversion. The goal is for all patients to transition off nutrition support post liver transplant.

Table 8 Enteral nutrition recommendations post liver transplant (guidelines per our institution).

Energy- use lower range for well-nourished at time of transplant, upper range to resolve malnutrition	Infants: 100-110 kcal/kg, 110-120 kcal/kg if malnourished. Older children BMR x 1.5-1.8
Protein	Same for enteral or parenteral
Cholecalciferol	Give standard dose for age post-transplant
Multivitamin	Not indicated if well nourished and receiving goal feeds.
Special consideration: Biliary drain	Loss of zinc, copper, and sodium bicarbonate via biliary fluids may require supplementation. Consider adding a multivitamin containing zinc and copper, and supplementing sodium bicarbonate.

4.4 Long-Term Nutrition Management

Infants and children are exposed to several potential complications post-liver transplant. The following is a list of nutritionally relevant issues and strategies to address them.

- **Food Safety**: Families are educated on preventing food borne illness, including proper formula mixing and/or breast milk storage, food preparation and storage, and foods to avoid due to high risk for infection [14]. Patients are advised to avoid grapefruit and grapefruit containing beverages due to drug nutrient interaction with immunosuppressant impairing drug metabolism and leads to high drug levels.
- **Hyperglycemia/Post-Transplant diabetes**: Most common in older adolescents and patients with Cystic Fibrosis. If blood sugars are consistently elevated, instruct on a 'no concentrated sweets' (i.e. no added sugar, avoidance of candy, desserts, syrups, regular soda, etc) diet. Advance to carbohydrate controlled meal plans and insulin therapy if necessary.
- **Hyperkalemia**: commonly seen with use of calcineurin inhibitors such as Tacrolimus. Restrict potassium in the diet if serum levels consistently exceed 5-5.5 mEq/L. A potassium lowering medication may be needed, especially if dietary restriction is preventing adequate nutrition intake for weight gain and growth.
- **Hypomagnesemia**: again related to the immunosuppressive medications. Magnesium supplementation is typically required to maintain serum levels, however oral magnesium is poorly absorbed and may cause loose stools. Allowing less than optimal magnesium levels may be appropriate if patients are experiencing uncomfortable side effects of magnesium supplementation.
- **Hyperlipidemia**: Steroids and immunosuppressants, particularly mTOR inhibitors (Sirolimus, Everolimus) may increase cholesterol low density lipoprotein (LDL) and high density lipoprotein (HDL). Little is known of the relevance of hyperlipidemia in adolescents and young adults who are long-term survivors of solid organ transplants [17]. Atherosclerosis is

a concern in adult transplant recipients, therefore long-term consequences in pediatrics warrant concern. Test Total cholesterol, Triglycerides, HDL and LDL levels annually.

- **Rejection**: During periods of acute rejection, high doses of corticosteroids are frequently used. A low sodium and/or carbohydrate-counted diet may be indicated. High dose steroids increase protein catabolism, therefore ensure adequate protein intake during treatment of rejection.
- **Bone disorders**: Bone mass may be reduced pre-transplant due to vitamin D deficiency and immobility or inactivity. High dose steroids in the post-transplant period may decrease bone formation, reduce bone strength, and predispose to pathologic fractures. Ensure adequate calcium, vitamin D and phosphorus. Supplementation may be indicated to achieve these goals and to maintain serum levels [14].
- **Food Allergies**: immunosuppressive medications are associated with de novo food allergies, which is estimated to occur in 10-17% of young children [18]. Clinical signs/symptoms include rash and diarrhea. Radioallergosorbent (RAST) testing and eosinophilic infiltration on GI biopsy are both quite sensitive to determining presence of allergies, but neither are specific to type. The Registered Dietitian educates family on appropriate diet modifications once an allergen has been identified.
- **Overweight/Obesity**: Dyslipidemia and impaired glucose tolerance are risks associated with overweight and obesity in pediatric patients. Catch up weight gain and accelerated weight velocity is expected post-liver transplant, however linear growth lags behind in trajectory, thus these patients are prone to overweight/obesity [19]. Complicating matters is the use of corticosteroids to treat rejection, leading to insulin resistance and metabolic syndrome like symptoms. Weight trajectory may normalize about 1 year post transplant, but not for all patients. Timely nutrition intervention should be provided by the dietitian and reiterated by team members, including education on healthy food choices, limiting sugary beverages, and ensuring structured meal and snack times.
- **Transplant for metabolic conditions**: Protein metabolism may not normalize immediately post-transplant. Monitoring of amino acid levels may be relevant and require carnitine and arginine supplementation to help normalize the amino acid profile as the graft becomes fully functional.

4.5 Monitoring Over Time

Immediately post discharge, patients are seen twice weekly in Transplant clinic. Depending on complexity of care, the RD will see post-discharge patients at least once, if not twice weekly at the Transplant clinic appointments at this stage. Parenteral nutrition is discontinued before discharge with the exception of those receiving an isolated liver transplant for parenteral nutrition associate liver disease secondary to short gut syndrome. Weekly to monthly appointments continue as needed for patients receiving enteral nutrition support or on specialty formula (i.e. low potassium). Once on normal diet and growing appropriately, the dietitian will remain available to see the patient as needed, or upon request from the family or transplant team, for growth and feeding concerns as they arise (Figure 1B).

Figure 1 Pre and Post liver transplant pictures of a patient with diagnosis of Alpha-1 Antitrypsin Deficiency. A. Age 6 months prior to liver transplant. Note that a central line in place for parenteral nutrition initiated due to severe malnutrition. B. Aged 2 years a developmentally appropriate, well-nourished toddler. (Images supplied by parents and with full permission to publish).

5. Conclusions

The pathophysiologic causes of chronic liver disease and acute liver failure are varied, and each present different and unique nutritional challenges in both the pre and post-transplant period. While malnutrition is common in the child with liver failure, it is also treatable when approached with a multidisciplinary focus on identification of clinical symptoms. With aggressive efforts to correct nutritional deficiencies pre-transplant, patients are afforded a more favorable healing period, followed by improved post-transplant nutrition allowing for continued linear growth and improved quality of life.

Author Contributions

Melissa Mortensen – paper conception, writing and editing text; Christine Lundberg – paper conception and editing text; Simon Horslen - paper conception, writing and editing text.

Competing Interests

The authors have declared that no competing interests exist.

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