



Guidelines

Nutrition support of children with chronic liver disease: A rapid review and consensus statements from Australasian society of parenteral and enteral nutrition (AuSPEN) Paediatric liver group



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ARTICLE INFO

Article history:

Received 19 September 2025

Accepted 12 May 2026

Available online 1 June 2026

Keywords:

Chronic liver disease

Paediatric nutrition

Liver transplant

Consensus

Rapid review

ABSTRACT

Chronic liver disease (CLD) in children is associated with high risk of malnutrition, micronutrient deficiencies, growth failure, and poor post-transplant outcomes. Although international 2019 guidelines exist, variability in practice across Australia and New Zealand (ANZ) highlights the need for locally relevant, updated nutritional guidance. This study aimed to conduct a rapid review of recent evidence (2018–2025) on the nutritional management of paediatric CLD and develop updated consensus statements to inform a best practice nutrition pathway for ANZ. A rapid review was conducted in accordance with PRISMA guidelines. Evidence tables were developed and appraised using National Health Medical Research Council (NHMRC) guidelines. Consensus statements were derived from the evidence tables, and voted on by all AuSPEN group members. Consensus was reached at 80% agreement. Of 1260 records screened, 67 full-text articles were included in the review, resulting in 19 evidence-informed consensus statements. Key areas included: proactive use of enteral and parenteral nutrition; cautious use of medium chain triglyceride (MCT) in context of potential essential fatty acid (EFA) deficiency; use of mid upper arm circumference (MUAC) and subjective global nutritional assessment (SGNA) for assessment; monitoring of sarcopenia; targeted support during post-transplant recovery and transition to adult care. Emerging topics included sarcopenia measurement and rehabilitation, and advancements in optimal nutrition management. This rapid review highlights both alignment with and advances upon 2019 international guidelines. The consensus statements provide a framework for best practice nutritional care of children with CLD across ANZ.

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1. Introduction

Chronic liver disease (CLD) is defined as the progressive destruction and regeneration of liver parenchyma leading to fibrosis and cirrhosis, which has been present for at least 6 months [1,2]. The most common cause of CLD in infancy is extra-hepatic biliary atresia (EHBA), occurring in approximately 1 in 8000 live births in Australia and New Zealand (ANZ) [3] which is significantly higher than estimated in Europe and North America (1 in 14 – 21,000 live births) [4]. In the New Zealand (NZ) Māori population, incidence is even higher (1 in 5285 live births) [3]. While we acknowledge that nutrition management of liver disease in children is tailored to whether the presenting disease is acute, chronic, or caused by an inborn error of metabolism, the main focus of this paper is chronic liver disease. However, chronic steatotic liver disease (including Metabolic Dysfunction-Associated Steatotic Liver Disease) will not be covered.

In children with CLD, malnutrition and sarcopenia (a surrogate marker for malnutrition) are common complications with known negative effects on morbidity, mortality and transplant outcomes [5–8]. Prevention of malnutrition in children with chronic liver disease (CLD) remains a constant challenge; however, it is well accepted that improving nutritional status may reduce morbidity, enhance life expectancy and improve quality of life [9]. To achieve optimal outcomes patients require proactive nutrition management by a multidisciplinary team (MDT) of specialist clinicians [9,10].

Malnutrition in CLD is multifactorial with contributing factors being nutrient malabsorption (due to reduced intestinal bile salts), disordered substrate metabolism, endocrine dysfunction (altered growth hormone and insulin like growth hormone) and increased nutrient requirements (hypermetabolic state) exacerbated by decreased nutrient intake (due to anorexia, nausea, vomiting, reflux and early satiety) [2]. CLD complications such as ascites (due to reduced albumin production), variceal bleeds (if portal hypertension) and organomegaly (with associated reflux and early satiety) may further impact nutritional intake. In end stage liver disease (ESLD), impaired liver glycogen storage may result in hypoglycaemia, especially in infants even when fasted for relatively short periods.

Individualized nutritional management is particularly critical in end stage liver disease, including children requiring liver transplantation. EHBA accounts for 54% of liver transplants (LTs) in ANZ in children under two years of age [11]. Other reasons for LT include metabolic diseases (15%) the most common of which is alpha-1-antitrypsin deficiency, followed by Alagille Syndrome and hepatoblastoma. ANZ has a 95% ten year patient survival rate in children receiving LTs, comparable with the rest of the world [11]. LTs are conducted in three specialist tertiary paediatric hospitals in Australia and one in New Zealand, servicing expansive geographical areas. Patients may have to fly for 5 h or 4000 km to reach their transplant centre. Therefore many hospitals share the care of these patients pre- and post- transplant, making consistency of care of paramount importance.

In 2019, Mouzaki et al. for the North American Society Paediatric Gastroenterology Hepatology And Nutrition (NASPGHAN) and the European Society Paediatric Gastroenterology Hepatology And Nutrition (ESPGHAN) committees published a comprehensive summary of evidence guiding clinical practice in nutrition support of children with CLD [12]; however, more recent evidence is yet to be synthesized to inform clinical practice. The AuSPEN paediatric liver group is a team of experienced clinicians working in tertiary specialist centres across Australia and NZ and includes two gastroenterologists and 12 dietitians. Despite the availability of the 2019 guidelines, there remains variability in practice of the nutritional management of paediatric CLD, necessitating locally relevant, and consistent updated national guidelines. Therefore, this rapid review aimed to review recent evidence (2018 to 2025) on the nutritional

assessment and management of CLD in young people aged 0–18 years. Findings from the rapid review were used to develop consensus statements using an expert working group to inform a best practice pathway, enabling consistency in clinical nutritional practice across Australia and NZ to achieve the best nutritional outcomes.

2. Materials and methods

This rapid review was conducted in line with the PRISMA Guidelines [13,14] and registered via Prospero (<https://www.crd.york.ac.uk/PROSPERO/view/CRD42023482415>). The rapid review approach was chosen to generate evidence in an accelerated and resource efficient manner to support timely clinical decision-making in the nutritional assessment and management of paediatric CLD, without compromising quality. As such, this rapid review was not designed to be as comprehensive as a systematic review and no risk of bias assessment was undertaken.

Four databases were searched; PubMed, Scopus, CINAHL, and Embase from January 2018 to March 2025, using the same keywords as Mouzaki et al. [12]. Three search sets were used in combination including terms relating to (1) liver disease, (2) nutrition, and (3) young people. The search was restricted to the English language and human studies. Studies were eligible for inclusion in the review if they reported on the nutritional assessment or treatment of paediatric CLD (ages 0–18 years). All study types were considered for inclusion in the review, with case studies and studies with insufficient methodological detail excluded. See Appendix 1 for further information regarding study eligibility.

Studies identified by the search strategy were imported to Covidence and titles and abstracts screened by two reviewers (DB, ML) for inclusion in the review. All studies selected for inclusion were placed in one or more of the following 6 categories; (i) anthropometry, (ii) biochemical parameters, (iii) nutrient requirements, (iv) practical nutrition management, (v) post transplant nutrition, and (vi) transition to adult services.

Each of the 6 categories was addressed by a subgroup comprising of 2 to 4 AuSPEN Paediatric Liver group dietitians (SA, AA, SA-J, SB, DB, TC, HC, KH, SK, EL, ML, KM). The paediatric gastroenterologists (AR, KMcG) and researcher (KP) provided advice, performed literature searches and reviewed throughout. Each subgroup reviewed full texts and collated an evidence table using the NHMRC Hierarchy of Evidence guidelines to assess level of evidence (LoE) [15]. The hierarchy ranges from high level of evidence (I) to low level of evidence (VII). Each subgroup presented the new evidence table to the main group for discussion around best practice recommendations for that category, with a focus on the highest quality of evidence. Each subgroup then formulated consensus statements and practice points, that were distributed by email to the main group for voting and comments in an iterative process. Generally 2 to 3 rounds of independent anonymous voting were necessary to refine statements, with the group leader (DB) collating responses. Consensus was reached at 80% agreement by all main group members. Conflict was resolved in main group meetings, with deference given to opinions from clinicians working in transplant centres (6 dietitians and 2 gastroenterologists).

3. Results

Of the 1260 papers screened by title and abstract, 166 were selected for full text review with 67 included in data extraction. Of the papers used to inform the consensus statements, few were considered of high quality and LoE ($n = 5$ systematic reviews including RCTs and observational studies, LoE I–IV; $n = 1$ RCT, LoE II). Of the remaining papers, 34 were cross-sectional and cohort studies (LoE IV), 13 were consensus and clinical guidelines (LoE IV–V),

Table 1

Summary of Consensus Statements: Nutrition Support in Paediatric Chronic Liver Disease from the AuSPEN Paediatric Liver Group.

	Consensus Statement	Agreement
1	In children with CLD, individualised patient care in partnership with caregivers, by a specialist multidisciplinary team (consisting of at least a gastroenterologist/ hepatologist, dietitian, nurse and social worker / psychologist) is essential.	100%
2	In children with CLD, early identification of malnutrition and micronutrient deficiencies followed by rapid, intensive intervention is crucial to optimise status and improve quality of life.	100%
3	Malnutrition is a common complication of end-stage liver disease with negative effects on liver transplant outcomes. Optimising nutritional status prior to liver transplant may improve post transplant outcomes.	100%
4	Serial anthropometric measurements including MUAC, TSF, weight and length/height z-scores, should be taken in children with CLD. The frequency of measurements is dependent on nutritional status and can range from every week to 3 monthly.	100%
5	Biochemical markers of nutrition can be challenging to interpret in children with CLD. Serial measures may be used in assessment of disease progression, and should be used in conjunction with clinical symptoms and signs to assess nutritional status.	100%
6	Energy requirements are higher in children with CLD by at least 130–150% NRV for age.	100%
7	Protein requirements are higher in children with CLD by at least 130–150% NRV for age.	100%
8	Evidence for the supplementation of BCAA to improve growth parameters and enhance nitrogen balance and muscle mass remains inconclusive. However, in advanced liver disease (at risk of hepatic encephalopathy), a BCCA supplemented formula may be considered if available, once the provision of adequate protein and energy has been ensured for positive nitrogen balance.	93%
9	Children with CLD are at increased risk of hypoglycaemia and carbohydrate intake should be regular, becoming more frequent if required as disease progresses.	100%
10	Once fat malabsorption is evident, aim for 30–50% of fat from MCT.	100%
11	Providing >80% of total fat as MCT increases the risk of essential fatty acids deficiency and should be avoided.	100%
12	To avoid EFA deficiency, greater than 10% total energy should be provided as PUFA with a minimum of 3% Linoleic acid and 0.7–1.0% alpha Linolenic acid. Monitor for biochemical and clinical signs of EFA deficiency and supplement as indicated.	100%
13	Fat-soluble vitamin (A, D, E, K) deficiencies are common in children with CLD. Early and regular biochemical monitoring and clinical surveillance are essential, with proactive supplementation instituted at the first sign of deficiency.	100%
14	Due to malabsorption, children with CLD require monitoring of key micronutrients, including water-soluble vitamins (such as B-complex vitamins and vitamin C), and essential minerals (such as zinc, selenium, iron, magnesium, and calcium). Supplementation should be based on deficiency risk or confirmed low levels. If supplementation of multiple vitamins and minerals is needed, a standard paediatric multivitamin at twice the usual dose for age is recommended, with additional targeted supplementation as necessary.	93%
15	A combination of feeding methods (oral, enteral nutrition, parenteral nutrition) together with specialised formulas may be required to proactively support growth in children with CLD.	100%
16	In end-stage liver disease, the initiation of supplementary parenteral nutrition is recommended when enteral nutrition is insufficient to maintain nutrition and growth	100%
17	Targeted nutrition support after liver transplant is required to facilitate progress to an age appropriate diet with the aim of promoting positive feeding experiences and managing oral aversion.	100%
18	Children who have undergone a liver transplant should be routinely screened for overnutrition and metabolic syndrome related complications.	100%
19	Transition from paediatric to adult care requires early planning and active coordination between paediatric and adult multidisciplinary teams.	93%

CLD= chronic liver disease; MUAC= mid upper arm circumference; NRV = nutrient reference value; BCAA = branch chain amino acids; MCT= medium chain triglycerides; PUFA = polyunsaturated fatty acids; ESLD = end stage liver disease; TSF = triceps skinfold thickness.

and 14 narrative reviews. Therefore, the grade of certainty of the overall body of evidence since 2018 was deemed to be low, according to the NHMRC LoE. Due to limited high quality study designs in CLD nutrition, the following statements are the consensus expert opinions of our clinician group after reviewing the current evidence. The 6 subgroups prepared evidence tables for presentation to the main group, and 19 consensus statements were generated. Please refer to Appendix 3 for PRISMA diagram, and Table 1 for Summary of Consensus statements.

4. Key statements

1. In children with CLD, individualised patient care in partnership with caregivers, by a specialist multidisciplinary team (consisting of at least a gastroenterologist/ hepatologist, dietitian, nurse and social worker / psychologist) is essential [100% Consensus]
2. In children with CLD, early identification of malnutrition and micronutrient deficiencies followed by rapid intensive intervention, is important to optimise nutritional status, minimise complications and improve quality of life [100% Consensus]
3. Malnutrition is a common complication of end-stage liver disease with negative effects on liver transplant outcomes. Optimising nutritional status prior to liver transplant may improve post transplant outcomes [100% Consensus]

5. Nutritional assessment

5.1. Anthropometry

Nutritional status in children with CLD can be assessed using static measurement of anthropometrics, body composition and

functional assessments. Mid upper arm circumference (MUAC) [16,17] and triceps skinfold thickness (TSF) [16,18] have been shown to be more accurate than weight or body mass index (BMI), in children with ascites or organomegaly, [16,19,20] and may indicate disease severity. MUAC reflects muscle, fat and bone while TSF reflects fat mass [12]. A MUAC z score of <-2 indicates moderate to severe malnutrition and criteria for malnutrition have been established for children aged 6 months to 17 years [21,22]. Dual energy X-ray absorptiometry (DEXA), and bioelectrical impedance analysis (BIA) become unreliable in the presence of ascites [23]. Serial measurements are crucial for monitoring nutritional status, and should be more frequent in severe disease [12].

The Subjective Global Nutrition Assessment (SGNA) has recently been validated [6] in the paediatric CLD population and evaluates anthropometry, weight change, food intake, body composition and function, allowing assessment of malnutrition severity. SGNA supports timely detection of malnutrition and proactive management. Pawaria et al. [6] found that SGNA severity predicts hospital admission, length of stay, and overall transplant-free survival in children with CLD.

Sarcopenia, characterised by loss of skeletal muscle strength, mass and function [24], is increasingly recognized as a nutritional marker in paediatric CLD and is linked to poorer transplant outcomes, lower one year survival, longer hospital stays [24–26], infections, and bone fractures [5,27]. Sarcopenia is prevalent in 24–70% of paediatric patients with CLD and was identified as a statistically significant risk factor for post-transplant outcomes such as longer intensive care unit (ICU) stay, longer duration of intubation, repeat operation, serious infection, longer hospital stay, death or long term growth failure [28]. Sarcopenia correlates with disease severity, but is independent of the Pediatric

End-Stage Liver disease (PELD) score, age, gender, weight, height or BMI.

Total psoas muscle surface area (tPMSA) via computed tomography (CT) was the most frequent method for assessing muscle mass and has recently had normative values defined for children 1–16 years [29–31]. No normative data exists for infants under 12 months. tPMSA is a more accurate measure of muscle mass than MUAC, though MUAC is easier to obtain [17]. Shpoliansky et al. [32] found that bilateral thigh thickness (BATT) via ultrasound, correlated with CT tPMA, but not with anthropometrics. Boster et al. [33] studied 127 children (5–18yrs old) with genetic CLD and found that low estimated skeletal muscle via DEXA, correlated with low bone density, but not severity of liver disease. In clinical settings, CT scans for psoas muscle, BIA, DEXA and total body potassium are impractical. For children 6–18 years, validated strength tests like hand grip and sit to stand can assess muscle function [34]. In younger children (< 3 years), validated developmental motor scales like Peabody and Alberta may be used [35].

In paediatric patients, a modified frailty index using handgrip strength, 6 min walk, TSF, a quality of life (PedsQL) and a physical activity questionnaire has been found to correlate to CLD severity; however further studies are required to determine usefulness as an outcome predictor [36]. Hager et al. [37] found a positive effect from exercise interventions on muscle strength and physical performance prior to transplant, but this was not reflected in muscle mass.

In summary, a nutritional status assessment using anthropometry (MUAC, TSF, weight, and height), together with a validated sarcopenia assessment (once determined) would be most useful. Completion of a paediatric SGNA to determine risk of malnutrition is recommended. However, given time constraints in clinical practice in Australia and NZ at present, it is most expedient to serially measure MUAC, TSF, weight and height whilst understanding their limitations in CLD.

5.2. Consensus statement

1. Serial anthropometric measurements including MUAC, TSF, weight and length/height z-scores, should be taken in children with CLD. The frequency of measurements is dependent on nutritional status and can range from every week to 3 monthly [100% consensus]

Practice Point: In end stage liver disease (ESLD), weight may be used to monitor fluid shifts, and an estimated dry weight based on linear growth may be more useful.

5.3. Nutritional assessment - biochemical parameters

Biochemical markers of nutritional status such as albumin, pre-albumin, transferrin and retinol binding protein are affected by inflammation, hydration status and the liver disease process reducing their reliability [12], but Machino et al. [38] found in 29 patients with EHBA, that serum albumin taken prior to Kasai portenterostomy (KP), significantly predicts 10 year native liver survival ($p < 0.01$).

Due to malabsorption, children with CLD require monitoring of key micronutrients, including fat-soluble vitamins (A, D, E, K), water-soluble vitamins (such as B-complex vitamins and vitamin C), and essential minerals (such as zinc, selenium, iron, magnesium, and calcium). Serial measures are more useful in detecting trends in disease progression, but should be interpreted alongside clinical signs and symptoms. In EHBA, the biochemical and clinical signs of fat soluble vitamin (FSV) deficiency are seen early, if not supplemented [39]. Please refer to Appendix 4 for clinical

signs and symptoms of FSV deficiencies and Appendix 5 for suggested nutritional biomarker monitoring. We recommend monitoring biomarkers at least three monthly, and more frequently as indicated. In particular, FSV and international normative ratio (INR) levels should be monitored to guide adjustment of supplement doses to prevent deficiency or toxicity [12].

Children with ESLD, are at high risk of bone disease. Astolfi et al. [40] found in 105 children (0–18yrs) undergoing LT, that 21% sustained a pathological bone fracture (PF) before or after LT. A PF was associated with low weight and height z-scores and delayed bone age. Despite Vitamin D and calcium supplementation to normalise serum levels, PF risk remained the same, leading to the conclusion that osteometabolism is complicated and multifactorial. Low bone mineral density (BMD) has been correlated to low weight z-scores before LT in 23 children [41], and to low serum calcium and hyperbilinaemia in 50 cholestatic children [42]. Therefore in CLD, serum levels of calcium and parathyroid hormone (PTH), magnesium and phosphate should be monitored as clinically indicated or every 3–6 months. If biomarkers or clinical signs indicate poor bone health, a DEXA is recommended [12].

5.4. Consensus statement

1. Biochemical markers of nutrition can be challenging to interpret in children with CLD. Serial measures may be used in assessment of disease progression, and should be used in conjunction with clinical symptoms and signs to assess nutritional status [100% consensus]

6. Nutrient requirements in CLD

Nutrient requirements in children with CLD are elevated due to nutrient malabsorption, disordered substrate metabolism, endocrine dysfunction and a hypermetabolic state. See Appendix 6 for Summary of Nutrient Requirements in CLD.

6.1. Energy and protein

Energy and protein requirements are typically 130–150% NRV for age and may further increase as disease progresses [12]. In end stage liver disease (ESLD), protein restriction under 2 g/kg/day should be avoided except when severe encephalopathy is evident and only then for 2–3 days [43] when medical treatments have failed [44].

Meeting the recommended intake for protein should provide sufficient branch chain amino acids (BCAAs); however, altered metabolism and increased energy demands may mean that BCAA requirements are not fully met without targeted supplementation or high quality protein sources. Limited data suggest that BCAA supplementation may have a favourable effect on weight, fat mass, fat free mass and serum albumin levels in infants and children with ESLD [45]. However, variability in dose, duration of supplementation, disease severity and lack of standardised outcome assessment tools limit the ability to draw definitive conclusions about its overall benefit [45]. Prophylactic BCAA supplementation appears to be safe and may improve survival in cirrhotic patients [45,46]. BCAAs have also recently been suggested as potential third line therapy for the inpatient management of hepatic encephalopathy when lactulose and antibiotics are not adequate; however, future research is needed [44].

6.2. Carbohydrate

Patients with ESLD are at higher risk of hypoglycaemia, due to impaired glycogen storage and release. Carbohydrate intake should contribute 40–60% of total energy supplied and be spread evenly throughout the day [9,12].

6.3. Fat

In children with cholestasis, interrupted or absent bile flow reduces the capacity to digest and absorb long chain triglycerides (LCTs). In contrast, MCTs do not require emulsification with bile due to their partial water solubility, allowing them to diffuse easily into intestinal cells for direct absorption into the portal venous system bound to albumin. Once fat malabsorption is evident, existing guidelines recommend that 30–50% of total energy from fat be from MCT, starting at 30% of total energy from fat, and increasing as required to support growth and minimise malabsorption up to 50% total fat calories [12].

A recent literature review highlighted the limited high-quality evidence supporting the benefit of MCT supplementation on growth and clinical outcomes in children with CLD [47]. Furthermore, a retrospective cohort study [48] in infants with biliary atresia post-KP found no significant association between the amount of MCT given (expressed as a percentage of total fat or in g/kg/day) on growth, nutritional status or clinical outcomes. Further research is needed to clarify the benefits of MCT supplementation and to define the optimal percentage of MCT to aim for in the diet of children with CLD. Current practice across centres in Australia and New Zealand is to change to a 50% of fat as MCT enriched formula once cholestasis is evident and continue to maximise nutritional intake and monitor growth closely in the lead up to LT [See Appendix 7]. Further research is needed to support the development of consensus statements regarding breastfeeding and transition to MCT-enriched formula, given the current limited evidence base.

6.4. Essential fatty acids (EFAs)

As MCTs are not a source of essential fatty acids (EFAs), it is important to maintain a proportion of LCTs in the diet. Providing > 80% of total energy from fat as MCT may increase the risk of EFA deficiency and should be avoided [9,12,47,48]. However, these recommendations are based on very limited evidence (four studies involving a total of 10 children) [47].

Essential fatty acids are nutrients at risk in children with CLD due to malabsorption and altered fat metabolism. Mancell et al. recently reported a correlation between EFA deficiency and diets high in MCT [48]. Current recommendations suggest at least 10 percent of energy should come from long chain polyunsaturated fatty acids (PUFA) with a minimum of 3% linoleic acid and 0.7–1.0% alpha linolenic acid [9,12]. Clinical signs of EFA deficiency can be misinterpreted as fat soluble vitamin (FSV) deficiencies and may be present before reflected in biochemical measures. The ability to test biochemical levels of EFAs, in a timely manner, is limited at many sites across Australia and NZ so it is recommended to suspect EFA deficiencies if FSVs are low.

6.5. Fat soluble vitamins

Fat soluble vitamin levels should be routinely monitored and supplemented as indicated. Intramuscular injections may be required as disease (and malabsorption) progresses to ensure adequate levels are maintained [9,12]. Da Rocha et al. [49] found that daily supplementation of 6000 IU oral cholecalciferol for at least 60 days in 31 Vitamin D deficient children with CLD resulted in restoration of Vitamin D to within range in 29% of patients. This was associated with less severe liver disease. In those whose levels did not restore to within normal range, an increase in median Vitamin D level and reduced PTH levels was noted [49].

6.6. Water soluble vitamins and minerals

Studies investigating the requirement for water soluble vitamins (WSVs) and minerals in children with CLD are limited. Consensus

opinion suggests they should be routinely monitored in malnourished patients with CLD and supplementation should be based on deficiency risk or confirmed low levels. If supplementation of multiple vitamins and minerals is needed, a standard paediatric multivitamin at twice the usual dose for age is recommended, with additional targeted supplementation as necessary [9].

7. Fluid

Fluid requirements in children with CLD are considered normal for age unless restriction is required if oedema or ascites are present.

8. Consensus statements

1. Energy requirements are higher for children with CLD by at least 130–150% NRV for age [100% consensus]
2. Protein requirements are higher for children with CLD by at least 130–150% NRV for age [100% consensus]
3. Evidence for the supplementation of BCAA to improve growth parameters and enhance nitrogen balance and muscle mass remains inconclusive. However, in advanced liver disease (at risk of hepatic encephalopathy), a BCCA supplemented formula may be considered if available, once the provision of adequate protein and energy has been ensured for positive nitrogen balance [93% consensus]
4. Children with CLD are at increased risk of hypoglycaemia and carbohydrate intake should be regular, becoming more frequent if required as disease progresses [100% consensus]
5. Once fat malabsorption is evident, aim for 30–50% of fat from MCT [100% consensus]
6. Providing greater than 80% of total fat as MCT increases the risk of EFA deficiency and should be avoided [100% consensus]
7. To avoid EFA deficiency, greater than 10% total energy should be provided as PUFA with a minimum of 3% Linoleic acid and 0.7–1.0% alpha Linolenic acid. Monitor for biochemical and clinical signs of EFA deficiency and supplement as indicated [100% consensus]
8. Fat-soluble vitamin (A, D, E, K) deficiencies are common in children with CLD. Early and regular biochemical monitoring and clinical surveillance are essential, with proactive supplementation instituted at the first sign of deficiency [100% consensus]
9. Due to malabsorption, children with CLD require monitoring of key micronutrients, including water-soluble vitamins (such as B-complex vitamins and vitamin C), and essential minerals (such as zinc, selenium, iron, magnesium, and calcium). Supplementation should be based on deficiency risk or confirmed low levels. If supplementation of multiple vitamins and minerals is needed, a standard paediatric multivitamin at twice the usual dose for age is recommended, with additional targeted supplementation as necessary [93% consensus]

9. Practical nutrition management

To optimise nutritional status in CLD and minimise adverse effects of progressive liver disease on growth and neurodevelopment, literature supports early aggressive nutrition intervention using all combinations of feeding methods ranging from oral supplementation to enteral and parenteral nutrition [12,50,51,52]. Appendix 7 outlines a suggested pathway for feeding in CLD.

We encourage the preservation of breast feeding and the use of expressed breastmilk (EBM) if clinically feasible, as breast milk is beneficial, safe and may enhance growth [53]. Tessier et al. studied 447 infants with EHBA, fed either human milk (50.5%) or formula alone (variety used, not all containing MCT). There was a significant decrease in total bilirubin ($p = 0.01$) at 3 months post

KP when using human milk, and greater weight gain and MUAC scores (both $p < 0.001$) [53]. No significant difference in clearance of jaundice was found between the 2 groups. Thus, human milk should not be discouraged in this population and decisions for individuals should be based on clinical situation. For infants with CLD, oral skill development and complementary food introduction when developmentally appropriate (around 4–6 months), should be encouraged to avoid development of oral aversion, even though oral intake may not contribute significant nutrition [12]. Referral to a multidisciplinary feeding team should be considered if feeding concerns arise.

In infants, we recommend changing to an MCT enriched formula (50% of total fat as MCT) once cholestasis is evident. Increases in feed concentration should occur slowly to monitor for side effects (i.e. vomiting, bloating, diarrhoea) and provide time for tolerance to be achieved [47,52]. In children with CLD, EFA sources such as walnut, soy, canola oil, fish oils, and egg yolks, should be introduced and regularly included in the diet to mitigate the risk of deficiency [12]. In certain cases in the older paediatric population, consider supplementation with walnut oil [54].

In cases where oral intake is inadequate in meeting nutritional goals, or if there is a suboptimal trend in anthropometrics over 2–4 weeks, insertion of a nasogastric (NG) feeding tube is recommended [12]. For patients who cannot tolerate NG feeds or are at risk of aspiration, trans-pyloric feeding should be considered to help improve tolerance [55]. If tube feeding otherwise known as enteral nutrition (EN) is not tolerated nor effective (ie. fails to meet ≥ 60 –70% of estimated energy requirements for >5 –7 days), or contra-indicated (high risk of oesophageal variceal haemorrhage), supplementary parenteral nutrition (PN) has been demonstrated to be a safe and effective method to resolve malnutrition before liver transplant [19,56,57,58]. Ueno et al. demonstrated in 14 patients awaiting LT, that PN resulted in greater mean rate of weight gain with minimal complications observed, where EN was ineffective or not indicated [58]. Limitations of PN include hospitalisation, fluid and sodium imbalance, risk of line infection and further compromised liver function [59]. Further higher quality studies are needed to guide use of supplementary PN regarding optimal commencement, duration and patient selection. Parenteral supplementation of fat-soluble vitamins may be necessary if vitamin levels remain low despite optimal oral supplementation and EN support [42]. Clinical consideration should be given to the addition of trace elements in PN, in particular the potential for copper toxicity.

9.1. Managing clinical symptoms: nutritional considerations

The clinical symptoms of worsening CLD [ascites, organomegaly, reflux, vomiting, anorexia, dysgeusia, cholestasis, portal hypertension, bone disease, hypoglycaemia, encephalopathy] will present challenges in meeting patients' nutritional requirements [60]. If ascites and organomegaly are present, with associated reflux, nutritional support with both oral and enteral feeding, involving smaller, more frequent meals/feeds and continuous overnight tube feeds, may be beneficial. In cases of vomiting, consider increasing feed concentration to reduce volume, and slowing the feed rate and feeding over a longer period of time. In ESLD, our AuSPEN expert group notes that the majority of children require EN, sometimes with PN support to meet daily nutrition requirements prior to transplant. However, there is no published data to support this assertion.

Children with ESLD are at higher risk of hepatic encephalopathy (HE), particularly when porto-systemic shunting is present. Malnutrition and sarcopenia are significant contributors to the occurrence of encephalopathy. Serum ammonia and signs and symptoms (including day-night sleep reversal, irritability and slurred speech) should be monitored. If HE is present, manage medically as per

local guidelines [44]. Nutritional management of HE should prioritize adequate protein intake, including use of ammonia-lowering agents as appropriate, and correction of micronutrient deficiencies (e.g. zinc, vitamin D). In refractory HE protein restriction to 1–2 g/kg/day may be considered for a few days [12].

Patients are also at higher risk of hypoglycaemia, due to impaired glycogen storage and release, and appropriate monitoring is essential, especially in infants. If hypoglycaemia is persistent, patients may require continuous feeding via NG or NJ tube [12]. Special care should be taken when patients with ESLD who are at high risk of hypoglycaemia are fasting for procedures. They should not be fasted for >2 –3 h without an alternative carbohydrate source such as intravenous dextrose (10% minimum) [61]. Puri et al. suggest aiming for a glucose infusion rate (GIR) of 10mg/kg/min or 15g/kg/day [61]. Oral rehydration solution alone provides insufficient carbohydrate to prevent hypoglycemia and needs the addition of a glucose polymer [12,62].

Salt restriction may be recommended in older children and adolescents with CLD in the management of ascites secondary to cirrhosis and portal hypertension, due to impaired renal sodium excretion and a resultant positive sodium balance. However, in malnourished children, strict sodium restriction may reduce dietary palatability and subsequently compromise overall energy and protein intake [62]. In clinical practice, it is more common to restrict fluid in this setting, than to restrict salt [12,62].

9.2. Consensus statements

1. A combination of feeding methods (oral, enteral, PN) together with specialised formulas may be necessary to proactively support growth in children with CLD [100% consensus]
2. In end-stage liver disease, the initiation of supplementary parenteral nutrition is recommended when enteral nutrition is insufficient to maintain nutrition and growth [100% consensus].

10. Post transplant nutrition

There is limited paediatric literature available to inform the nutritional management of post liver transplant patients.

10.1. Early post transplant: critical care

Under guidance from hepatologists, surgeons and critical care specialists, feeding should commence within the first 24–72 h post LT if the child is clinically stable [63]. Physiologically, there are no indications for the use of MCT for nutrition support post liver transplant in the absence of biliary complications or chyle leaks. Appendix 8 outlines a pathway for feeding in the early post operative period post LT.

Electrolyte disturbances may occur due to immunosuppressant and other medications (e.g. diuretics, antifungals), and should be monitored and corrected accordingly. For example, immunosuppressive therapy with tacrolimus can lead to hyperkalemia or hypomagnesaemia [12]. Magnesium supplementation may cause diarrhoea. If tacrolimus has been prescribed, dietary interactions must be considered. Grapefruit, blood orange and pomegranate are to be avoided due to effects on CYP3A metabolism. In some centres, fasting pre and post-tacrolimus dosing may be recommended by the transplant team as food can affect therapeutic levels.

10.2. Ward level care

The majority of infants will have been enterally fed prior to transplant, and rapid weaning is indicated unless clinical concerns (e.g. poor hydration, graft rejection, sepsis, severe malnutrition or

oral aversion) are present. Pre-existing oromotor delays may impact the resumption of post operative oral intake. Early involvement of a multidisciplinary team (dietitian, speech therapist, occupational therapist) to proactively manage feeding difficulties and oral aversion is recommended.

10.3. Home care

Post LT patients should be encouraged to eat a wide variety of nutritious, age-appropriate foods, including being offered foods they may have disliked pre-transplant. Avoidant restrictive food intake disorder (ARFID) and selective eating patterns are rare but can develop after LT, and may be under-reported in paediatric patients [64].

A study by Liu et al. in 2024 found that parents of children who had undergone liver or kidney transplant believed that the quantity of food consumed implied adequate nutrition, without awareness of nutritional quality [61]. This highlights the need for structured post LT education for families, around food group balance. Most post LT children achieve weight catch up within a year [65,66]; however, height catch up may take 5 years [67]. A multicentre study of 892 children who survived beyond the first year post LT found that transplant recipients are shorter than expected based on mid-parental heights [68]. Ee et al. using total body potassium measurements, demonstrated that weight recovery in 32 long term post LT patients (>5 years) was due to increased fat mass, not body cell mass [69].

The persistence of sarcopenia post LT is associated with poorer growth and recurrent hospital admissions [70]. A 2024 study of 108 paediatric LT patients found a sarcopenia incidence (via CT) of 45%, and a higher rate of transplant rejection in the sarcopenic group [71]. Ooi et al. [72] postulate that immunosuppressive therapies (corticosteroids, calcineurin inhibitors) may contribute to alterations in skeletal muscle composition. Individualised physical rehabilitation programmes are recommended as part of routine post LT care. Supervised exercise rehabilitation programmes may help reverse sarcopenia and support linear growth [73,74].

In addition, patients should be screened for overnutrition and metabolic syndrome, which are associated with increased risk of graft loss, overall morbidity, and mortality [12,75–77]. A 2018 study of 21 children in New Zealand found that 52% of patients were either overweight or obese (median) 8 years post transplant [78]. Tessitore et al. note that data is scarce on prevalence of post-transplant metabolic syndrome (PTMS) in children, with its associated glucose intolerance and hypertension [9].

There is a higher risk of developing new food allergies after LT compared with other transplant groups and the general population [79]. A meta-analysis by Bergamo et al. found this occurred in up to 15% of LT patients, particularly those on Tacrolimus [80].

10.4. Consensus statements

1. Targeted nutrition support after liver transplant is required to facilitate progress to an age appropriate diet with the aim of promoting positive feeding experiences and managing oral aversion [100% consensus]
2. Children who have undergone a liver transplant should be routinely screened for overnutrition and metabolic syndrome related complications [100% consensus]

11. Transition of care

The transition from paediatric to adult care poses an increased risk of medical complications and morbidity. It requires targeted partnership between multi disciplinary paediatric and adult teams to move from family centred to patient centred care [81,82]. The

essential elements for a successful transition were identified with an international Delphi study [83]. They identified that planning should begin in early adolescence, self management strategies should be developed with the patient and family, the views and preferences of the patient should be considered, that they should be seen alone for part of the consultation, and should be introduced to the adult clinician before care transfer [81].

In 2023, Joshi et al. published UK Guidelines on Transition and management of childhood liver disease into adulthood [84]. Joshi et al. highlighted that barriers to a successful transition were the young person's lack of knowledge of their condition and limited experience in self-care. The guidelines recommend psychological assessment and support, bone health screening every 2–3 years, and that readiness for transfer not be based solely on age [84]. The impact of mental health conditions should be considered as this affects adherence to medications, appointment attendance and self-management of their condition and local mental health pathways should be planned as part of the transition process [82]. In ANZ setting, there are additional challenges due to geographical isolation and access to specialized services, which may disrupt the continuity of care. Given the higher EHBA incidence in the NZ Maori population, consideration should be given to a culturally safe transition pathway.

11.1. Consensus statement

1. The transition from paediatric care to adult, requires early planning and active coordination between paediatric and adult multidisciplinary teams [93% consensus]

12. Discussion

This rapid review has reviewed recent evidence regarding nutritional management of children with CLD, and developed 19 consensus statements. The NASPGHAN/ESPGHAN position paper on nutritional management of CLD 2019 [12] provides an excellent clinical guide for paediatric patients with CLD, and our findings are broadly consistent with NASPGHAN/ESPGHAN's core recommendations. In the last 6 years, research has focussed on assessment of sarcopenia, malnutrition and bone disease, effectiveness of MCT and BCAA supplementation, safety of PN in ESLD, and transition guidelines. Further studies are needed to determine a validated sarcopenia or frailty assessment tool, bone disease prevention strategies, the optimal nutritional formula and nutrition management. Investigations into the impact of interventions such as exercise programs to reduce sarcopenia, addition of BCAAs and MCT, increased PN use and close monitoring of bone disease, FSVs and EFAs, and sarcopenia will help elucidate optimal CLD management.

Although we followed a systematic process for this rapid review, this review was designed to prioritise key information in a timely manner to inform clinical decision making. Additionally, formal risk-of-bias or GRADE assessments were not performed due to the heterogenous nature of included studies and the consensus based approach. Studies included in this review were frequently retrospective and cross-sectional, included small sample sizes, and involve medically complicated populations, which impacted study design and limit generalisability. Therefore the strength and quality of evidence to inform best practice nutrition management of CLD is low and limited, with few RCTs and meta-analyses, and findings of the review should be interpreted in light of this. Whilst ideally, large prospective studies are desired to guide clinical management, in practice heterogeneity in research can be a limitation delaying the ability to make recommendations, slowing translation. A further limitation is that consensus was derived from expert opinion

rather than a formal, iterative process such as the Delphi methodology, which may have introduced confirmation bias. However, despite these limitations, consensus based recommendations based on available evidence and clinical expertise can advance clinical practice in a timely manner.

In particular, a best practice pathway and clinician toolkit developed from consensus statements may enhance consistency of nutritional care between rural and urban centres in ANZ. An on-line toolkit may improve clinician skill and confidence in managing children with CLD, and improve awareness of the proactive often intensive nutritional management required.

12.1. Future research priorities

Despite some new research in recent years, notably in MCT use, safety of PN, functional assessment of sarcopenia and link with clinical outcomes, Mouzaki et al.'s list of topics for future research remains current:

1. Determine utility of functional assessments of nutritional status on guiding the approach to nutrition support, and assessing the impact on interventions.
2. Determine the optimal nutritional support across the spectrum of CLD severity.
3. Determine the nutritional risk of transplant recipients, and optimal approach to monitoring and intervening [12].

However, our AuSPEN Liver group also suggests that research priority should be given to the optimal use of MCT, usefulness of breastfeeding, proactive strategies for nutritional rehabilitation using enteral feeding and PN, and prevalence and management of PTMS in children.

13. Conclusion

The optimal nutritional management of children with CLD involves early identification, proactive intervention and intensive monitoring of nutritional parameters by a skilled multidisciplinary team, to minimise malnutrition and nutrient deficiencies. The rapid review and consensus statements assist in informing a best practice pathway that aims to improve quality and consistency of nutritional management across Australia and New Zealand, as well as identifying future research areas.

Funding

No funding to declare.

Declaration of competing interest

The authors declare no conflict of interest

Acknowledgments

The authors would like to thank Dr Scott Nightingale, Paediatric Gastroenterologist at the John Hunter Children's Hospital for his review of the paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at [doi:10.1016/j.dld.2026.05.006](https://doi.org/10.1016/j.dld.2026.05.006).

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