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Nutritional management of children with acute kidney injury—clinical practice recommendations from the Pediatric Renal Nutrition Taskforce

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Abstract

The nutritional management of children with acute kidney injury (AKI) is complex. The dynamic nature of AKI necessitates frequent nutritional assessments and adjustments in management. Dietitians providing medical nutrition therapies to this patient population must consider the interaction of medical treatments and AKI status to effectively support both the nutrition status of patients with AKI as well as limit adverse metabolic derangements associated with inappropriately prescribed nutrition support. The Pediatric Renal Nutrition Taskforce (PRNT), an international team of pediatric renal dietitians and pediatric nephrologists, has developed clinical practice recommendations (CPR) for the nutritional management of children with AKI. We address the need for intensive collaboration between dietitians and physicians so that nutritional management is optimized in line with AKI medical treatments. We focus on key challenges faced by dietitians regarding nutrition assessment. Furthermore, we address how nutrition support should be provided to children with AKI while taking into account the effect of various medical treatment modalities of AKI on nutritional needs. Given the poor quality of evidence available, a Delphi survey was conducted to seek consensus from international experts. Statements with a low grade or those that are opinion-based must be carefully considered and adapted to individual patient needs, based on the clinical judgment of the treating physician and dietitian. Research recommendations are provided. CPRs will be regularly audited and updated by the PRNT.

Keywords Acute kidney injury · Nutrition · Continuous kidney replacement therapy · Pediatric Renal Nutrition Taskforce · Clinical practice recommendations

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Introduction

Children with acute kidney injury (AKI) are at high risk for malnutrition and nutritional deterioration due to the high impact that electrolytes, minerals, protein, and fluid from nutritional sources have on the metabolic anomalies seen in AKI. As such, the nutritional assessment and nutritional management of AKI are as dynamic as its disease process. Appropriate nutritional therapy of AKI in children can limit metabolic and fluid derangements while continuing to provide optimal nutrition that supports healing. However, nutritional management in children with AKI lacks standardization, which is especially challenging for healthcare professionals given AKI's phenotypic variation and the paucity of nutrition-based research.

The Pediatric Renal Nutrition Taskforce (PRNT), an international team of pediatric renal dietitians and pediatric nephrologists, has developed clinical practice recommendations (CPRs) addressing nutritional assessment and nutritional therapy in children with AKI. This CPR is based on an extensive review of the literature and, with the acceptance of there being limited evidence in the nutritional management of pediatric AKI, expert opinion employing adaptation of other areas of the literature (e.g., pediatric chronic kidney disease, pediatric critical care, adult acute kidney disease).

Methods

Previous PRNT publications have described in detail the guideline development process, including working group composition and task distribution [1]. Abbreviations that are frequently used in the manuscript are listed in Table 1.

Developing the PICO questions

Clinical practice recommendations provide precise actionable guidance on choosing between alternative approaches in specific clinical situations. We developed clinical questions to be addressed by each statement and framed them in a searchable format, with the specification of the patient group (P) to whom the statement would apply, the intervention (I) being considered, the comparator (C) (which may be "no action" or an alternative intervention), and the outcomes (O) affected by the intervention. Our PICO terms were as follows:

Population: Infants, 37 or more weeks gestation, to children 18 years of age, with AKI
Intervention: Nutritional support or assessment in children with AKI, with or without kidney replacement therapy (KRT)
Comparator: Children without AKI or no comparator

Table 1 List of abbreviations used in the manuscript with their full forms

Abbreviation	Full form
AKI	Acute kidney injury
PRNT	Pediatric Renal Nutrition Taskforce
CPR	Clinical practice recommendation
PICO	Patient group, intervention, comparator, outcome
KRT	Kidney replacement therapy
PICU	Pediatric intensive care unit
SDI	Suggested dietary intake
CKD2-5D	Chronic kidney disease, stages 2 through 5, including dialysis
ICU	Intensive care unit
CKRT	Continuous kidney replacement therapy
PYMS	Paediatric Yorkhill Malnutrition Score
STAMP	Screening tool for the assessment of malnutrition in paediatrics
PNST	Paediatric nutrition screening tool
PN	Parenteral nutrition
GI	Gastrointestinal
REE	Resting energy expenditure
MREE	Measured resting energy expenditure
IC	Indirect calorimetry
CHO	Carbohydrate
KDOQI	Kidney disease outcomes quality initiative
UL	Upper level

Outcomes: Optimization of nutrition to limit catabolism, limit metabolic complications, and maintenance or improvement, when necessary, of nutritional status

Literature search

An electronic search using PubMed and an inclusive academic library search (including MEDLINE, Cochrane, and EMBASE databases) was performed using the search terms and strategy detailed in Supplementary Table 1. Limits were preset to only include manuscripts published in the English language between 1980 and August 2022. Given the paucity of studies in this field, all publications, including meta-analyses, prospective observational studies (irrespective of patient numbers), and retrospective observational studies and case series, have been included.

Framing advice

The first PRNT-published CPR has outlined the development process and purpose of the recommendations [1]. Statements have been graded using the American Academy of Pediatrics grading matrix (Supplementary Table 2) and were submitted to a Delphi procedure, as previously described [1], to validate expert opinion.

Clinical practice recommendations and rationale

Throughout this CPR, a critically ill child is defined as one requiring treatment in the pediatric intensive care unit (PICU) or comparable hospital unit (e.g., cardiac intensive care unit). Both critically ill and non-critically ill children may be receiving dialysis. AKI is defined based on laboratory and clinical criteria and was assumed to be based on an AKI guideline definition, which the PNRT recognizes may be modified over time [2]. Furthermore, given the evidence limitations, extrapolation and use of the suggested dietary intake (SDI) in children with chronic kidney disease stages 2 through 5, including dialysis (CKD2–5D) for a more pragmatic and international approach as described by Shaw and colleagues was used when referring to energy and protein CPRs [3].

1. Collaboration

   1.1 Ensure close collaboration between the healthcare professionals providing medical management and those providing the nutritional prescription for the optimal overall care of children with AKI. (level X; strong recommendation)

Rationale: Close collaboration between healthcare professionals is essential for the nutritional management of children with AKI in order to properly optimize nutrition provision and to determine the correct nutrition prescription and goals that align with the medical management and necessary treatment modalities. This is especially important in this patient population since major changes in clinical status or management occur frequently and may directly affect the nutritional prescription. Yet, especially in the intensive care unit (ICU) setting, there may be a variety of providers making medical decisions and other providers modifying the nutritional prescription. Medical decisions that may affect the nutritional prescription include stopping or starting dialysis, changing the dialysis prescription, and stopping or starting diuretics. Changes in clinical status may include volume overload or volume depletion, abnormalities in gut motility or absorption, and laboratory evidence or symptoms of uremia. Highly effective communication has the potential to improve patient safety [4]. The medical treatment of AKI is dynamic and nutritional therapy should reflect the dynamic nature of the medical treatment. Collaboration between medical and nutrition support teams spans the entirety of our recommendations and is thus a defining theme we will discuss throughout.

2. Nutritional assessment

   2.1 Utilize a validated pediatric nutrition risk screening tool for the assessment of nutritional risk within 48 h of AKI diagnosis. (level B, moderate recommendation)
   2.2 Refer any patient found to be at nutritional risk to a dietitian for nutritional assessment. (level B; moderate recommendation)
   2.3 Repeat nutritional assessments in accordance with the severity of nutritional risk, severity and duration of AKI, and changes in KRT. (level D; weak recommendation)

Rationale: The prevalence of malnutrition in hospitalized children ranges between 6 and 32%, depending on the country [5]. Children with the most severe AKI, requiring continuous kidney replacement therapy (CKRT), have reported malnutrition rates between 30 and 55% [6–9]. Fluid and electrolyte restrictions limit the selection of commonly consumed foods by children and impact nutritional status. Furthermore, it has been reported that critically ill children with AKI experience high rates of nutritional debt within the first 5 days of PICU admission [10]. In hospitalized children, malnutrition can increase adverse outcomes such as mortality, length of stay [11], readmission rates [12, 13], and infection rates [14], and it disproportionally affects children

with severe medical conditions [11]. Due to the epidemiological shift of AKI in the last 20 years, there is likely to be a substantial overlap between children experiencing AKI and those having severe medical conditions such as multiorgan system failure, septicemia, respiratory failure, and congenital cardiac anomalies. A study on nutrition screening in hospitalized adults reported that high-risk patients identified via nutrition screening tools exhibited a higher incidence of AKI when compared to patients with normal nutritional status [15]. Therefore, we expect that children with AKI are at high risk for malnutrition and should have appropriate nutritional screening as detailed below.

Nutrition screening is an important first step that serves to notify a dietitian that a patient may be at nutritional risk and that further assessment of nutritional status is warranted. Only recently has there been the evidence to allow more informed decisions on the appropriate use of validated and reliable nutrition screening tools in hospitalized children. Four tools (Paediatric Yorkhill Malnutrition Score, PYMS; Screening Tool for the Assessment of Malnutrition in Paediatrics, STAMP; STRONGkids; and Paediatric Nutrition Screening Tool, PNST) have the most evidence to support their use with moderate validity [16–18]. However, it is important to note that none is specific to children experiencing AKI. Furthermore, the heavy reliance on anthropometric measurements of weight or proportionality of weight to height should be used with caution in children with AKI because of the potential for false positives or negatives in the case of patients with dehydration or fluid overload, respectively. Additionally, PYMS, STAMP, and STRONGkids include screening for high-risk diseases with nutritional impact and specify kidney disease, although not necessarily AKI. Yet, there is no reason not to utilize these screening tools in children with AKI.

Most groups recommend a 48 h screening period after admission to allow for timely nutrition evaluation and intervention if needed [18]. Timely intervention would allow nutrition teams to decrease the nutritional debt or limit the amount of time children with AKI are not meeting their optimal nutritional needs. This could also allow for the implementation of preventative strategies that support optimizing fluid balance and avoiding or minimizing electrolyte derangements. There is no consensus on the frequency of reassessment for hospitalized children other than via weekly re-screening of nutritional risk. However, given the dynamic nature of AKI and its treatment modalities, the reassessment of nutritional risk and efficacy of nutritional interventions should reflect the frequency with which the medical management of AKI changes. Furthermore, given the higher risk for malnutrition in younger children [11] and critically ill children [19], it is also expected that age and the presence of critical illness will also dictate the frequency at which children are reassessed for nutritional risk. Therefore, strong multidisciplinary collaboration is necessary to provide optimal nutritional management, which may require daily reassessment in some patients, especially those in the ICU.

2.4 Obtain accurate anthropometric measurements as soon as feasible and throughout hospitalization. (level A; strong recommendation)

- 2.4.1 Estimate euvolemic weight using accurate trended weight measurements in conjunction with other clinical assessment measures such as fluid balance, blood pressure, physical examination, and available biometric tools (e.g., bioelectrical impedance analysis, mid-upper arm circumference, non-invasive blood volume monitoring). (level D; weak recommendation)
- 2.4.2 Measure height or recumbent length for children under 2 years of age. For those unable to stand for accurate measurement of height, use recumbent length or a surrogate missed measurement of height. (level A; strong recommendation)
- 2.4.3 Measure head circumference for children up to 2 years of age or up to 3 years of age when appropriate centile charts are available. (level A; strong recommendation)
- 2.4.4 Assess for muscle wasting by physical assessment and use of biometric tools where available. (level D; weak recommendation)

Rationale: Accurate evaluation of a hospitalized child's nutritional status hinges on the appropriate evaluation of anthropometrics and body composition. As hospitalization and critical illness promote muscle protein breakdown in excess of muscle protein synthesis, children with AKI will experience negative alterations in fat mass, muscle mass, and functional capacity, thereby worsening morbidities and promoting long-term metabolic abnormalities in pediatric AKI survivors. Measurement of anthropometrics would ideally occur upon hospital admission, but are imperative once a child is diagnosed with AKI. It is highly likely that hypervolemia will mask muscle loss or true weight loss experienced by these patients. The most basic anthropometrics required for a full nutrition assessment to evaluate for malnutrition (acute or chronic) are weight and length/height [and head circumference for children less than 3 years of age] as per standard practice even outside of a diagnosis of AKI. Optimal nutritional management will attempt to limit or rectify malnutrition and malnutrition risk. Anthropometrics are a vital component for this evaluation. In an ideal setting, measurement of functional capacity and strength of a child with AKI to aid in the assessment of malnutrition would be performed. Unfortunately, anthropometrics are obtained at low frequency and accuracy in hospitalized children, worsening with critical illness [20, 21]. Children with AKI are most at risk for

infrequent and inaccurate anthropometrics; the most frequent reasons reported for inability to obtain anthropometrics include hemodynamic instability and extracorporeal therapies. Furthermore, children with AKI frequently have fluid overload, which interferes with an evaluation of nutritional status when weight is used alone. Thus, the use of surrogate measures, including for height, may be necessary, as has been recommended previously in children with CKD [22]. Individualized nutritional prescription relies on monitoring changes in nutritional status, and if changes are not accurately quantified, then children may not receive optimal nutritional prescriptions. Thus, evaluation of a child's nutritional status and body composition (namely muscle mass and volume status) should be performed using a variety of metrics.

Assessment parameters such as net fluid balances, bioelectrical impedance analysis (BIA), ultrasound, musculoskeletal physical assessment, non-invasive blood monitoring in acute hemodialysis (HD), and mid-upper arm circumference (MUAC) may provide alternative means to support the assessment of nutritional status in the absence of accurate anthropometric measurements. Table 2 provides examples of a variety of body composition (muscle, fat, and water) techniques that may be available for comprehensive nutritional status evaluation in the setting of shifting fluid status to support individualized therapeutic nutrition interventions; these are consistent with CKD assessment recommendations [22]. A physical assessment of muscle wasting can predict the longer length of stays independently of anthropometrics or other nutritionally-related risk factors of malnutrition in hospitalized children [39]. MUAC has been revitalized in the pediatric malnutrition literature and may hold promise to aid the assessment of malnutrition [32, 40, 41]; it is a quick and easy anthropometric tool, especially when weight and height measurements are not available in immobilized patients [42]. Interestingly, MUAC has been reported to be less affected by fluid status changes than weight, especially in the setting of hypovolemia [43, 44]. Volume status can be estimated via physical assessment and net fluid balance. However, for institutions with more resource availability, the utilization of biometric tools such as ultrasound or BIA may improve the assessment of volume status and muscle mass changes. BIA has been utilized to evaluate a variety of clinical parameters including both fluid and nutritional status in critically ill children [19, 45]. Ultrasound has also been utilized in a variety of ways. Some studies have assessed fluid status through point-of-care lung ultrasound testing [46] or inferior vena cava diameter [31], while others have investigated muscle thickness changes in critically ill children [47, 48] to monitor nutritional status in the absence of other anthropometrics. Adaptations and advances in technology will be necessary to allow for improvements in assessment methods of nutritional status and the efficacy of individualized nutritional therapies.

The standard nutritional practice recommends nutritional reassessments of hospitalized children weekly, at minimum. However, there are no definitive recommendations on the frequency of anthropometric evaluation by nutrition support societies [49]. Anthropometric and body composition evaluation are key components to aid in the monitoring and evaluation of the appropriateness of nutritional therapies. Therefore, the frequency of anthropometric measures should continue to reflect the dynamic changes in AKI, its corresponding medical management, and the acuity and age of the child. A weight measurement of smaller children and infants should occur more frequently. A weight measurement of children where fluid overload has masked euvolemic weights may be considered more frequently to refine nutrition prescriptions. Measures reflecting anthropometrics or body composition changes that may change more slowly may be done at lower frequencies.

3. Oral and enteral feeding

   3.1 Oral feeding, including breastfeeding, is the preferred method of providing nutrition. (level X; strong recommendation)

   3.2 In critically ill children, consider early initiation (within 48 h of admission) of supplemental or exclusive enteral tube feeding when oral feeding does not meet nutritional requirements, especially when nutritional intake is likely to remain suboptimal. (level C; weak recommendation)

   3.3 Use whole protein (polymeric) formulas unless otherwise indicated, such as in the case of gastrointestinal dysfunction. (level C; weak recommendation)

   3.4 Consider the use of protein and energy-dense formulas to achieve nutritional goals within the limits of the fluid allowance and gastrointestinal tolerance; adjust formula density gradually to maximize tolerance. (level C; moderate recommendation).

4. Parenteral nutrition

   4.1 For children with malnutrition or risk for nutrition deterioration, when oral or enteral nutrition cannot provide all nutritional requirements, initiate supplemental parenteral nutrition. (PN) (level X, moderate recommendation)

   4.2 For children without malnutrition or risk for nutrition deterioration, when oral or enteral nutrition cannot provide all nutritional requirements, PN may be withheld for up to 1 week provided micronutrients are delivered. (level B; moderate recommendation)

   4.3 For all children, regardless of nutritional status, receiving KRT that causes significant nutrient losses, initiation of PN before 1 week should be considered when oral or enteral nutrition cannot

Table 2 Multifocal body composition and body compartment assessment techniques available for hospitalized children

Method	Body Compartment	Advantage	Limitation
BIA [23, 24]	Fat mass, fat free mass estimated from total body water	Quick, portable, inexpensive, safe, reproducible, non-invasive, does not require highly trained personnel, Increasing evidence in utilization of fluid status assessment [25, 26]	Sensitive to hydration, individual level prediction errors, limitations to prediction equations (e.g., sex, age, ethnicity), limited standardization of reportable measures
CT [23, 27]	Adipose tissue, skeletal muscle, bone, organs	Accurate, L3 cross sectional area good correlation with total body muscle volume	Radiation exposure, costly, requires expertise, body size limitation, availability of site and disease state
MRI [23, 27]	Adipose tissue, skeletal muscle, visceral organs	Accurate	Costly, requires expertise, not portable
B-Mode ultrasound [23, 27–29]	Localized muscle thickness and fatty infiltration Localized skin thickness – multisite equations for FM estimations Fluid overload – lung scoring [30], inferior vena cava diameter [31]	Portable, safe, minimal training, reproducible, beneficial for abdominal adiposity discrimination between subcutaneous and visceral May differentiate volume-dependent hypertension from volume-independent hypertension in dialysis	Muscle measurement more prone to error due to compressibility, site selection, transducer position and hydration, secondary processing required for fatty infiltration of muscle, 2 dimensional Fluid assessment not reliable, not correlated with extracellular fluid volume
Anthropometry	Weight/Length – fat estimation BMI – fat estimation SFT – fat estimation Circumferences: MUAC – total body muscle estimation [32, 33] Leg – site specific muscle estimation Arm – site specific muscle estimation	Quick, portable, easy, inexpensive, non-invasive, safe SFT performs well in CKD MUAC with good correlation to BMI z scores ≤ –2 [32]	Insensitive, altered by hydration, best used serially, dependent on accurate weight and height measurements Difficulty attaining accurate weight and height measures in ICU SFT with operator variability, performs poorly in dialysis [34]
Physical assessment [14, 35, 36]	Subjective surrogate measures for change in fat, muscle, and edema	Reliable, clinically validated	Requires additional measures to confirm assessment
% Fluid overload [37]	Relative TBW %FO = ((Fluid in – Fluid out)/ ICU admission wt) × 100 Weight modified (CKRT) %FO = ((CKRT initiation wt – ICU Admission wt)/ ICU admission wt) × 100 %FO = ((CKRT initiation wt –hospital wt)/ hospital admission wt) × 100	Quick, easy to perform, validated	Reliant on precise and accurate I/O measurement and calculations, does not account for insensible losses, requires accurate weight measurements, fluid accumulation not site specific
Non-Invasive Blood Volume Monitoring [38]	relative blood volume; hematocrit dilution changes during hemodialysis	Validated method to achieving euvolemic weights while limiting dialysis-associated morbidities	Specific to hemodialysis, requires specialized equipment

BIA, bioelectrical impedance analysis; *CT*, computed tomography; *MRI*, magnetic resonance imaging; *L3*, lumbar vertebra 3; *BMI*, body mass index; *SFT*, skin fold thickness; *CKD*, chronic kidney disease; *ICU*, intensive care unit; *MUAC*, mid-upper arm circumference; *FO*, fluid overload; *CKRT*, continuous kidney replacement therapy; *wt*, weight; *I/O*, ins and outs; *TBW*, total body water

Adapted from multiple sources within table

provide all nutritional requirements. (level D; weak recommendation)

4.3.1 Pay careful attention when transitioning from PN to enteral feeding in children receiving CKRT to ensure the provision of optimal nutrition within the fluid allowance. (level C; moderate recommendation)

Rationale: Feeding through the gastrointestinal (GI) tract remains important to decrease morbidity in hospitalized children. Critically ill children who receive enteral feeding early in their management, especially feeding associated with higher protein intakes (> 60% of their prescribed goal during their PICU stay), exhibit lower mortality rates [50].

Breastmilk contains the optimal energy and nutrient composition to promote health and age-appropriate growth in infants and toddlers [51, 52], and the PRNT supports breast feeding or giving expressed breastmilk whenever possible [3]. The use of energy-dense formula is a key nutrition intervention, often applied in children with AKI, especially when fluid restriction is necessary for optimal medical management. Energy-dense oral nutrition supplements should be offered to children not meeting nutritional needs with an oral diet alone. In a randomized controlled trial of children with faltering growth, energy-dense oral nutrition supplementation has been shown to result in significantly higher energy intake compared to lower-density oral nutrition supplements [53]. Furthermore, the use of energy-dense formula in critically ill adults has been shown to meet nutritional needs sooner due to the ability to achieve goal energy needs in less time, allowing for meeting nutritional goals despite procedural disruptions [54]. The introduction of energy-dense enteral tube feeding should occur gradually to promote GI tolerance [3, 55]. There is insufficient evidence to support the selection of partially hydrolyzed or extensively hydrolyzed protein formulas over whole protein (polymeric) formulas unless the latter are contraindicated or poorly tolerated [49]. However, there are also reports that children with AKI exhibit higher rates of GI intolerance [56]. Additionally, patients with sepsis-related AKI may frequently experience GI intolerance [57].

When oral feeding fails to meet nutritional requirements and enteral tube feeding is necessary, there is not enough evidence to recommend the optimal tube placement site, but the gastric route is preferred for patients in the PICU as compared to the post-pyloric route [3, 55]. There is no evidence to support the use of continuous over intermittent feeding in critically ill children [49]. Despite the reported benefits of enteral feeding, it may be a barrier to the provision of adequate protein in children with high protein: calorie ratio needs, such as those receiving CKRT [58]. However, enteral feeding, even at trophic levels, promotes gut barrier function, thereby limiting potential infectious complications [59–61].

In agreement with current evidence on initiation of PN when sufficient nutrition via the enteral route cannot be achieved, in the absence of malnutrition or KRT, PN initiation may be delayed up to 1 week, provided micronutrients are delivered prior to that time. Current evidence indicates that early (< 24 h) PN initiation is associated with worse outcomes (longer length of stay and increased infection rates) [59]. In early PN initiation, amino acids (ranging from 0.75 to 1.15 g/kg per day) were associated with increased infections and longer dependency on mechanical ventilation [60]. The evidence supporting the avoidance of early PN initiation is based on a single randomized clinical trial in critically ill children where PN was initiated if enteral goals did not provide more than 80% of their caloric target. Admittedly, this is a relatively high goal within the first week of admission to a PICU given the metabolic changes that occur in such patients. Evidence supporting a more pragmatic timeframe to achieve therapeutic nutrition goals is limited to the critical care literature in children, where achieving approximately 60% of energy and protein targets within 7 days of a PICU admission via enteral or enteral with supplemental PN is associated with lower mortality, but not ventilator-free days or infections [62]. It is hypothesized that withholding early PN support during the acute phase of critical illness maintains autophagy, an essential survival mechanism, which decreases the risk of organ failure and cell death; autophagy also has an important role in innate immunity [63]. In children with AKI, where there are higher rates of malnutrition, GI disturbance, and increased dialysis nutrient-related losses, PN may need to be considered earlier than 1 week given their higher overall risk for nutrition-related disturbances.

5. Energy requirements

5.1 non-critically ill children, the initial prescription for energy intake should approximate the SDI based on euvolemic weight, not measured weight. (level B; moderate recommendation)

5.2 In the acute phase of critical illness, energy requirements should not exceed the resting energy expenditure (REE). (level C; weak recommendation)

5.3 In the stable phase and recovery phase of critical illness, the energy prescription must account for energy debt, physical activity, rehabilitation, and growth. (level X; moderate recommendation)

5.4 Modify the energy prescription to account for dialysis-related net gain or loss of energy. (level C; weak recommendation)

5.5 In critical illness, consider an increased percentage of energy intake from fat to reflect changes in beta-oxidation when parenterally fed. (level C; weak recommendation)

Rationale: For non-critically ill children with AKI, the SDI for energy can guide the initial prescription of dietary intake [3]. During critical illness, the body reacts to physiological stress in three phases characterized by different metabolic responses [64]. In the acute phase, where vital organ support is required, muscle protein synthesis and REE decrease. The body reacts with an inflammatory cascade aiming to survive the critical illness by supplying blood, energy, and substrates to the injured site [65]. The metabolic response is catabolism (muscle protein breakdown and lipolysis) for substrate delivery to the vital tissues. Hyperglycaemia occurs due to increased gluconeogenesis and peripheral insulin resistance [66]. Catabolism and muscle protein breakdown is not reversed with increased provision of nutrients during this phase despite previous ideas to the contrary [67]. In the stable phase, there is maintenance or weaning of vital organ support, but not all aspects of the stress response are resolved [64]. There is still protein wasting despite the small increase of anabolic hormones such as growth hormone and insulin-like growth factor-1. Muscle wasting is exacerbated by immobilization and medication [68]. During the recovery phase, there is no longer a need for vital organ support, the child is mobilized, and the stress response is resolved. Hormone levels normalize and the body shifts from catabolism to anabolism (positive nitrogen balance, tissue repair, and catch-up growth) [64, 69]. During the stable and recovery phases, the aim of nutritional support is to restore lean body mass [64].

Little is known about the energy requirements of critically ill children with AKI. Based on observational cohort studies in critically ill children (not AKI exclusively), measured REE (MREE) by indirect calorimetry (IC) is the best diagnostic technique to determine the energy requirements during the acute phase. However, IC is not widely available nor feasible, so the Schofield weight-height or Schofield weight (see Table 3) or the World Health Organization equations without the addition of stress factors may be used to determine EE during the acute phase of illness [49, 55]. The Harris–Benedict equations and recommended dietary allowances should not be used to determine the energy requirement of critically ill children. No correlation has been found between MREE and nutritional status, initial diagnosis, or severity of the acute illness [49, 55, 72–75]. We suggest that IC be used to determine the EE in critically ill children with AKI. In the absence of IC, either the Schofield or WHO equations (without stress factors) may also be used [76]. REE can be increased when the child has a body temperature above 38 °C, or decreased when the child is deeply sedated. Energy intake should be increased gradually above the REE to ensure full recovery and catch-up during the stable and recovery phases. Furthermore, based on the prior discussion regarding avoidance of early PN initiation and the research available on appropriate timing to achieve target energy goals, it is likely that achieving greater than the REE would be difficult when following feeding modality guidance in critically ill children. To avoid over and underfeeding the critically ill child who is on CKRT, it may be important to account for the energy (calories) from citrate (3 kcal/g), lactate (3.62 kcal/g) and glucose from dialysis, hemofiltration or anticoagulation solutions [76]. The net caloric gain depends on the type and rate of fluids used as well as the CKRT dose [77]. Furthermore, acute peritoneal dialysis may also provide calories from glucose-containing dialysis solution; however, there are limited studies that address this issue [78, 79], and maintenance peritoneal dialysis nutrition practice is to avoid this calculation and adjust based on growth trends. In total, serial anthropometric evaluation works in concert to aid the assessment of energetic adequacy.

The optimal energy substrate in critically ill children with AKI is also not well known. However, in a cross-sectional study with 33 critically ill children (non-AKI) receiving PN and mechanical ventilation, fat was used preferentially for oxidation, and carbohydrate (CHO) was utilized poorly [80]. A higher CHO intake promotes lipogenesis and decreases lipid oxidation. In a prospective multicenter study on 42 adult patients with AKI receiving enteral and/or PN, CHO oxidation was significantly lower than both prescribed and administered CHO. This study showed that adults with AKI used much less CHO than expected, while oxidizing much

Table 3 Schofield [70] and WHO [71] equations for calculating REE

	Age	Boys	Girls
WHO	0–3 y	60.9 × (w kg) − 54	61.0 × (w kg) − 51
	3–10 y	22.7 × (w kg) − 495	22.5 × (w kg) + 486
	10–18 y	22.7 × (w kg) + 651	12.2 × (w kg) + 746
Schofield w	0–3 y	59.5 × (w kg) − 30	58.3 × (w kg) − 31
	3–10 y	22.7 × (w kg) + 505	20.3 × (w kg) + 486
	10–18 y	17.7 × (w kg) + 658	13.4 × (w kg) + 692
Schofield w/ l	0–3 y	0.167 × (w kg) + 1517.4 × (l m) − 616.6	16.252 × (w kg) + 1023.3 × (l m) − 413.5
	3–10 y	19.6 × (w kg) + 130.3 × (l m) + 414.9	16.25 × (w kg) + 161.8 × (l m) + 371.2
	10–18 y	16.97 × (w kg) + 137.2 × (l m) + 515.5	8.365 × (w kg) + 465 × (l m) + 200.0

WHO, World Health Organization; *REE*, resting energy expenditure; *w*, weight; *l*, length

more lipid [81]. An increased insulin resistance leads to hyperglycemia and increases lipolysis leading to increased beta-oxidation, indicating a potential shift in substrate utilization because of the inflammatory milieu from critical illness and AKI. The precise composition of CHO and lipid substrate delivery could prevent worsening metabolic derangements, and thus, in children with AKI, increased lipid over CHO administration may be more appropriate.

6. Protein requirements

6.1 For non-critically ill children, the initial prescription for protein intake should approximate the SDI based on euvolemic weight, not measured weight. (level B; moderate recommendation)

6.2 critically ill children, consider the potential need for increased protein intake above the SDI to limit negative protein balance. (level B; moderate recommendation)

6.3 In children with very elevated blood urea nitrogen levels, especially if progressively worsening, first ensure adequate energy intake; then, consider a temporary lowering of protein intake towards the lower end of the SDI. (level C; moderate recommendation)

6.3.1 Do not persistently compromise protein intake to lower urea nitrogen levels or postpone KRT initiation. (level X; strong recommendation)

6.4 For all children receiving dialysis, protein prescription needs to be further increased to account for dialysis losses, which are highest in CKRT. (level C; moderate recommendation)

Rationale: Baseline protein need is higher in children than in adults given the need for growth. Updated consensus recommendations support the use of a pragmatic and international approach to appropriate protein provision in pediatric CKD2–5D utilizing the SDI [3]. In the setting of limited evidence, for non-critically ill children, the SDI guide protein recommendations. Catabolism and negative nitrogen balances are frequently reported in children [7, 82, 83] and adults [84–87] with AKI. In the setting of AKI, the inflammatory milieu and changes to the ubiquitin–proteasome system pathway can increase muscle protein breakdown, which leads to higher urea nitrogen production and thus negative nitrogen balances [88]. Adequate protein provision aims to attenuate muscle protein breakdown, support tissue repair, and facilitate rehabilitation leading to better functional outcomes of survivors. It is important to consider how healthcare professionals aim to achieve optimal protein intake in children with AKI. Although enteral protein provision has been associated with lower ICU mortality [50, 62], enteral formulas may not contain the optimal protein concentration for critically ill children receiving CKRT; hence, they may require additional protein modular supplementation or even supplemental PN to achieve protein goals [58].

No data exists on the appropriate protein dosing for children receiving conservative medical management for AKI. However, restriction of protein which would cause worsening malnutrition to delay dialysis initiation is not recommended by pediatric nephrology healthcare professionals [3]. There is no data to support that protein provision inhibits AKI recovery. In the CKD literature, a randomized controlled trial comparing protein intakes showed that lower length and growth velocity were observed in children with lower protein intakes [89]. Moreover, it is well established that lower height attainment is associated with greater mortality in children with CKD [90]. We acknowledge that data from CKD are likely not fully comparable for those children with AKI. However, in AKI contributing factors to elevated blood urea nitrogen levels in the setting of catabolism, especially beyond the acute phase of critical illness, should be evaluated closely as muscle protein breakdown also acts as a source of urea generation. Any decreases in protein intake for extended periods of time need to be carefully evaluated for their utility and patient-centered goals of care.

Protein needs in acute dialytic therapies in AKI treatment are often extrapolated from chronic dialytic therapy. Studies of children receiving chronic peritoneal dialysis have shown protein losses may range from 0.1 to 0.28 g/kg/day, with the highest losses seen in smaller children [3, 91], while achievement of positive nitrogen balances required up to approximately 144% of the recommended daily allowance [92] in the setting of steady-state energy and protein metabolism. Through extrapolation of adult maintenance (3 times/week) HD studies, the Kidney Disease Outcomes Quality Initiative (KDOQI) workgroup recommended 0.1 g/kg/d to account for dialysis-related protein losses in children [93]. The PRNT recommendations for energy and protein for children with CKD2–5D updated these recommendations through the utilization of the SDI ranges when accounting for these losses [3].

Most of the data available regarding the nutritional management of patients with AKI addresses amino acid losses experienced in CKRT. These studies consistently reported losses equivalent to approximately 10–20% of amino acids provided via nutrition support [7, 82, 94]. Studies of children receiving CKRT demonstrate negative nitrogen (protein) balance. For example, in a study where 120–130% of MREE and 2 g protein/kg/day were provided, negative protein balances persisted [82]. Additionally, concentrations of serum amino acids appear to stabilize 5 days following the initiation of CKRT, save glutamic acid [94, 95]. Our knowledge of protein losses experienced in CKRT only includes the initial 5 days after CKRT initiation. Protein losses from KRT should be accounted for to prevent negative protein balances.

7. Micronutrient needs (vitamins, trace elements, and carnitine)

    7.1 In children who are conservatively managed, provide the recommended requirements of vitamins and trace elements for healthy children, with supplementation in the case of insufficient intake. (level D; weak recommendation)
    7.2 In children requiring dialysis, consider providing additional supplemental water-soluble vitamins, selenium, copper, zinc, and carnitine; either enterally or parenterally. (level D; weak recommendation)
    7.3 Avoid supplemental vitamin A in all children with AKI. (level B; strong recommendation)
    7.4 Evaluate for clinical signs and symptoms of deficiency or excess of vitamins, trace elements, and carnitine. (level C; weak recommendation)
    7.5 Do not routinely measure serum concentrations of vitamins, trace elements, and carnitine unless there are clinical signs or symptoms of deficiency/toxicity, or when the child is receiving treatment for deficiency or has known toxic concentrations. (level D; weak recommendation)

Rationale:

Vitamins Critically ill children are prone to vitamin deficiencies due to a hypermetabolic state, decreased intestinal absorption, insufficient intake, increased excretion, medication-related effects, and underlying metabolic disorders. Patients on dialysis are also at risk of deficiency due to the loss of water-soluble vitamins and trace elements in the dialysate [96], although there is limited data on their clearance by individual dialysis modality. There is also limited data on the assessment of vitamin B status in critically ill pediatric patients [97]. Based on one study, children on CKRT are at risk for decreased folate and thiamine levels [96].

The current level of evidence is too low to make recommendations for the routine measurement of vitamin and trace element concentrations in patients with AKI. Laboratory evaluation of most micronutrients should be used with caution. Inflammation can affect many serum micronutrient levels due to movement between plasma and the intracellular space as well as decreased sequestration and increased exposure to reactive oxygen species [98]. However, the presence of symptoms of nutrient deficiency or excess is important to identify, especially in children with underlying CKD who may have suboptimal nutrition or additional risk factors for pre-existing nutritional deficiencies [96]. In a retrospective review of 47 children on maintenance dialysis who received daily supplementation with pyridoxine and a commercial water-soluble vitamin preparation, the concentrations of several trace elements and vitamins were outside the reference range, with both deficiency and excesses noted [99]. Similar data in children with AKI is not available. Moreover, as may be expected in the clinical setting of AKI, it is not known if an acute and transient deficiency of particular vitamins and trace elements can lead to adverse outcomes. The clinical signs of deficiencies or excess of vitamins and trace elements are described in Table 4. If clinical symptoms of micronutrient deficiency or excess are noted, laboratory assessment is suggested to confirm clinical findings and intervene appropriately. Repletion dosing used in the general pediatric population is reasonable, though monitoring to document successful correction may be appropriate (Supplementary Table 3).

There is a paucity of research and high-quality evidence to guide micronutrient supplementation in AKI. The latter has led to large variations in practice around the world [99]. Requirements in children with AKI are compared against the recommended requirements for healthy children; however, caution should be exercised to avoid exceeding the upper levels (UL) when the intake of diet and supplement is combined. Children receiving the majority or all their energy requirements from adult renal formulas generally meet 100% of the recommended requirements for vitamins and trace elements and may not require vitamin supplementation [104]. Care should be taken not to exceed 100% of the recommended requirements for vitamin and trace element intake due to the potential for toxicity, particularly in the oligo-anuric dialysis patient [96]. Micronutrient losses may be cumulative, and the dietitian should keep the duration of AKI and specific KRT modality in mind when assessing micronutrient need.

Supplementing vitamin C in critically ill adults has a sound pathophysiological rationale and a positive safety profile. Patients on KRT likely need doses similar to those of critically ill patients not receiving KRT. Intravenous vitamin C may be necessary to achieve normal plasma concentrations during KRT. However, data on dose adjustment of vitamin C during intermittent or chronic KRT are sparse, and more pharmacokinetic and dose–response studies are required [107].

Supplementation doses should be individualized based on individual patient needs, risk profiles, and dialysis losses [105]. Assessment of serum vitamin B12 should be considered if folate supplementation is administered. High folic acid intake may mask signs of pernicious anemia and silent progression of neurologic disease; thus, both folate and vitamin B12 levels should be monitored if folate is being supplemented [105]. Monitoring of these levels may be considered at 2-week intervals if supplementation is initiated, as levels may normalize after 2 weeks of B12 supplementation.

The kidneys play an important role in the metabolism and excretion of vitamin A. Patients with impaired kidney function have high circulating levels of retinol, possibly due to a combination of decreased glomerular filtration of the

Table 4 Overview of vitamins and trace minerals at risk for deficiency or excess in the pediatric dialysis population

Nutrient	Symptoms of deficiency	Symptoms of excess	Dietary sources	Diagnostic tests
Vitamin A	Night blindness, xerophthalmia, keratomalacia, poor bone growth, impaired resistance to infection, follicular hyperkeratosis	Hyperostosis, hepatomegaly, hepatic fibrosis, alopecia, increased cerebrospinal, fluid pressure [100], hypercalcemia [101]	Fortified milk, liver, egg, cheese, yellow fruits and vegetables (carotenoid precursors)	Plasma retinol [97]
Vitamin E	Hemolytic anemia in premature infants; fat malabsorption causes deficiency; hyporeflexia and spinocerebellar and retinal degeneration	Bleeding, impaired leukocyte function	Sardines, green and leafy vegetables, vegetable oil, wheat germ, whole grains, butter, liver, egg yolk	Plasma alpha-tocopherol [97]
Thiamine (B1)	"Wernicke encephalopathy"- peripheral neuropathy, ophthalmoplegia, nystagmus, ataxia, edema	Unknown	Dairy, nuts, legumes, fruits, egg, unrefined grains, pork, vegetables	Thiamine pyrophosphate level, whole blood/red blood cell transketolase activation test
Pyridoxine (B6)	Facial seborrhea, glossitis, angular stomatitis, cheilosis, mental status changes	Neuropathy, photosensitivity	Fortified cereals	Pyridoxal 5'-phosphate 4-pyridoxic acid
Folate (B9)	Abdominal cramps, nausea, diarrhea, irritability, poor sleep, seizures, megaloblastic anemia	Masking of B12 deficiency symptoms in patients with pernicious anemia not receiving cyanocobalamin (B12)	Green vegetables, liver, grains, some fruits	Acute deficiency – low serum folate; chronic deficiency – low red blood cell folate
Vitamin C (ascorbic acid)	"Scurvy"- osmotic diarrhea, gingival bleeding, perifollicular hemorrhage, long bone changes (arthropathy)	Massive doses predispose to kidney stones; nausea, abdominal pain; rebound scurvy when massive doses stopped	Citrus fruits	White blood cell ascorbate concentration, radiographic evidence of long bone widening
Zinc	Clinical phenotype of"Acrodermatitis enteropathica" – anorexia, hypogeusia, growth retardation, diffuse skin lesions with impaired wound healing	Few toxic effects; may aggravate marginal copper deficiency	Meat, dairy, legumes, whole grains, oysters	Blood zinc level
Selenium	"Keshan disease"- progressive cardiomyopathy, cardiovascular disease with thyroid disease, also with myositis	Irritation of mucous membranes, pallor irritability, indigestion	Meats, seafood, nuts	Blood selenium level
Copper	Sideroblastic anemia, retarded growth, osteoporosis, neutropenia, decreased pigmentation	Few toxic effects; Wilson disease, liver dysfunction	Shellfish, meat, legumes, nuts, cheese	Serum copper, ceruloplasmin, superoxide dismutase activity
Chromium [102]	Glucose intolerance, weight loss and a metabolic encephalopathy-like confusion state	Genotoxic and carcinogenic	Meat and meat products, oils and fats, breads and cereals, fish, pulses and spices	Serum, plasma, urine chromium levels
Manganese [103]	Fleeting dermatitis, miliaria crystallina	Neurological disorder	Nuts, chocolate, cereal-based products, crustaceans and molluscs, pulses, fruits and fruit products	None
Carnitine	Hypoglycemia, dyslipidemia, muscle weakness, rhabdomyolysis, cardiomyopathy, arrhythmia, sudden death	Diarrhea	Meat, fish, dairy	Plasma, urine, skeletal muscle

Table adapted from multiple sources [96, 102–106]

retinol–retinol-binding protein complex, reduced conversion of retinol to retinoic acid, and an accumulation of retinol-binding protein [100]. Increased levels of retinol are reported in infants [108] and children with CKD and on maintenance dialysis despite lack of supplementation and are correlated with hypercalcemia [101]. Retinoic acid has been shown to reduce inflammation and fibrosis in experimental models of kidney injury; however, hypervitaminosis A has also been reported in AKI [109].

Trace elements Trace elements play a key antioxidant role in critical illness. Altered concentrations of trace elements during AKI are well described and are due to variable protein binding, redistribution from blood to tissues, acute losses, and removal by CKRT [110–112]. In the few studies that assessed effluent losses of trace elements in children with AKI, there was significant variation among studies, depending, in part, on the dialysis modality and its duration [94, 113, 114]. Trace element removal by CKRT, including zinc, copper, chromium, and selenium, was reported to be lower than the supplemental amounts received through PN, thus causing no deficiencies. However, two pediatric studies reported manganese excess but without evident clinical symptoms in the affected children [94, 113]. In the only prospective pediatric study, selenium balance was negative on day 2 and day 5 after initiation of CKRT [94]. Lower concentrations of selenium have been independently associated with an increased risk of death and hospitalization [115]. A prospective study in adults has shown that all trace elements studied were below the reference range throughout the 6-day study period, both in CKRT and non-CKRT patients [116]. Nevertheless, no study has provided high-quality evidence on the effect of supplementation, deficiencies, or excess of trace elements on the clinical outcome of critically ill pediatric or adult patients [86, 114, 117]. Repletion dosing as suggested in the general pediatric population is reasonable, though monitoring to document successful correction may be appropriate (Supplementary Table 3).

Carnitine The combination of a low protein intake together with the removal of carnitine by CKRT can lead to the depletion of levocarnitine (L-carnitine), which facilitates the transport of fatty acids across the inner mitochondrial membrane and is thus a critical co-factor for normal energy production in cardiac and skeletal muscle. Dialysis-related carnitine deficiency is common among children and adults on chronic HD due to the efficient removal of carnitine during each HD treatment, with an inverse relationship between muscle carnitine and duration on dialysis [118, 119]. Within a single dialysis session, clearance is 30 times greater than would be expected in a healthy individual. HD results in an abnormal acylcarnitine:free carnitine ratio [120]. In a pediatric cohort of 42 patients on CKRT, carnitine was even more rapidly depleted by CKRT compared with chronic HD, with losses approximating 80% of intake. Carnitine deficiency was associated with a longer duration of stay and increased mortality [106]. In a small pediatric study, CKRT patients who did not receive carnitine supplementation were carnitine deficient after 1 week on CKRT, while supplementation with intravenous carnitine was associated with repletion of plasma carnitine and improvement in myocardial strain [121]. Data are less conclusive in adult critically ill patients with AKI starting CKRT. One study showed significantly lower carnitine concentrations at 24 h but not beyond [116], while another study showed that carnitine losses continued during 6 days of follow-up [122].

8. Electrolyte monitoring

    8.1 Routinely monitor serum sodium, chloride, potassium, calcium, phosphorus, magnesium, and bicarbonate ("electrolytes") throughout the course of AKI. (ungraded)

    8.2 Adjust the frequency of electrolyte monitoring based on laboratory and clinical variables including: trends in electrolyte levels, changes in estimated glomerular filtration rate and urine output, use of or change in KRT prescription or modality, urinary and extrarenal losses of electrolytes and water, adjustments in delivery of electrolytes and fluid to the patient and medications. (ungraded)

9. Electrolyte targets

    9.1 Nutritional electrolyte delivery should be individualized and adjusted in close collaboration with the medical team based on ongoing and anticipated changes in clinical status, medications, and KRT prescription, generally aiming for the normal blood/serum ranges. (ungraded)

    9.2 Adjust nutritional and non-nutritional sodium and water delivery to optimize intravascular volume, with the goal to maintain adequate perfusion and prevent or correct volume overload or depletion. (ungraded)

        9.2.1 Chronically low or high serum sodium values should be corrected gradually to minimize the risk of neurological complications. (ungraded)

Rationale: Electrolyte abnormalities are common among patients with sustained AKI [123]. In some cases, KRT is necessary to manage life-threatening electrolyte and acid–base derangements, particularly severe hyperkalemia and acidosis. However, no studies have investigated the optimal frequency at which electrolytes should be monitored in AKI, as it is based on the clinical status of the patient, which can be very labile in children. A patient's clinical condition and biochemical parameters are usually the primary drivers of fluid and electrolyte management, not nutritional

parameters; hence, fluid and electrolyte delivery should be determined in conjunction with the clinical team.

Initial electrolyte monitoring may be required every 8 to 12 h, and occasionally more frequently, depending on the individual patient's clinical status. Once electrolytes have stabilized, the frequency of laboratory monitoring in the absence of other changes in patient-related variables, including urine output, may be less frequent, though subsequent increases in the frequency of monitoring may be needed due to changes in clinical status, especially initiating or stopping KRT (Supplementary Table 4).

Assessment of serum electrolyte values may be necessary prior to initiating intermittent HD. In patients requiring CKRT, daily electrolytes should be obtained so that the KRT prescription can be adjusted as necessary. Volume status may be a key driver of sodium balance and should be monitored on an ongoing basis. It is also important to consider medication changes when deciding upon appropriate electrolyte monitoring frequency, given that medications may influence electrolytes (Supplementary Table 5).

No studies have specifically investigated the electrolyte needs of children with AKI. It is not possible to provide a general prescription that would be appropriate for every patient given the extreme clinical heterogeneity in this population, including the level of kidney function and whether the patient is receiving KRT. The normal electrolyte requirements for healthy children may be used as an initial framework, but different clinical scenarios (e.g., extrarenal fluid and electrolyte losses), levels of kidney function, urine output, and different dialysis modalities will dictate ongoing adjustments (Table 5).

Serum potassium concentrations frequently increase in the setting of AKI due to decreased excretion, and thus intake is typically limited [124]. However, potassium losses in patients with diarrhea can be high, which may require replacement [124]. This may also occur in patients with certain tubular disorders if urine output is still present [125]. Conversely, patients with some disorders (e.g., rhabdomyolysis) or clinical situations (e.g., need for frequent red blood cell transfusions) may develop hyperkalemia and thus require limited intake and/or increased potassium removal via KRT [126]. Potassium is readily removed via KRT and the amount can be adjusted based on the potassium concentration of the dialysis or replacement fluid [127]. Changing the dialysate/replacement potassium concentration, stopping dialysis, and changing dialysis modality may dramatically affect potassium removal. Hence, it is critical that all healthcare providers communicate with each other regarding planned changes in the KRT prescription in order that nutritional potassium intake can be adjusted appropriately.

Most children with AKI develop a metabolic acidosis due to decreased acid excretion, which may be accentuated by certain clinical situations (e.g., diarrhea, lactic acidosis). Children not receiving KRT may require base supplementation [128, 129]. Conversely, gastric losses via emesis or

Table 5 Electrolyte requirements in healthy individuals and potential adjustments in patients with AKI

	Healthy children					Patients with AKI*				
	ASPEN		ESPEN			AKI without KRT	iHD	PD	CKRT	Special Considerations
Electrolyte	Infants/children	Adolescents and children > 50 kg	0–18 yr							
Sodium (mEq/kg/d)	2–5	1–2	1–3			↓	↓	↓	↓	Driven mainly by fluid balance
Potassium (mEq/kg/d)	2–4	1–2	1–3			↓	=	=	↑	Can be regulated by dialysis bags
			0–6 months	7–12 months	1–18 yr					
Calcium (mMol/kg/d)°	0.25–2	5–10 mMol/day	0.8–1.5	0.5	0.25–0.4	↑	↑	↑	↑	
Phosphorus (mMol/kg/d)	0.5–2	10–40 mMol/day	0.7–1.3	0.5	0.2–0.7	↓	= ↓	= ↓	↑	Can be regulated by dialysis bags
Magnesium (mMol/kg/d)°	0.15–0.25	5–15 mMol/day	0.1–0.2	0.15	0.1	=	=	=	↑	
Acetate/bicarbonate	As needed to maintain acid–base balance					↑	=	=	↓	

Intended not as a prescription, but rather as a description of what is usually needed in most patients with AKI; for conversion, 1 mMol = 2 mEq; *AKI*, acute kidney injury; *ASPEN*, American Society for Parenteral and Enteral Nutrition; *ESPEN*, European Society for Parenteral and Enteral Nutrition; *KRT*, kidney replacement therapy; *iHD*, intermittent hemodialysis; *PD*, peritoneal dialysis; *CKRT*, continuous kidney replacement therapy

nasogastric losses may cause a metabolic alkalosis. Acid–base balance impacts serum potassium (i.e., decreasing pH tends to increase serum potassium) and ionized calcium (i.e., decreasing pH tends to increase serum ionized calcium). These effects should be considered when correcting acid–base disorders in patients with potassium or calcium disorders (e.g., delay or avoid correcting acidosis with base in a patient with a very low serum potassium or ionized calcium). KRT provides a base to patients which tends to correct metabolic acidosis [130]. The amount of base provided will vary depending on the KRT prescription [131]. Physicians and dietitians should effectively communicate all changes in the KRT prescription that will influence base delivery, including the decision to stop KRT or changes to dialysis modality.

Hyperphosphatemia is common with AKI due to decreased phosphorus excretion, though levels may be low due to decreased intake or intracellular shifts [132, 133]. Decreased phosphorus intake is nevertheless frequently necessary. While KRT removes phosphate, the amount removed is generally limited with intermittent therapies. In contrast, CKRT may remove substantial amounts of phosphate [123, 130, 134, 135]. The amount of phosphate removed can be adjusted in some forms of CKRT [127, 135]. Physicians and dietitians should communicate regarding all changes to the KRT prescription that influence phosphate removal, including the decision to stop KRT or changes to dialysis modality.

In AKI, hypocalcemia may occur through a variety of mechanisms [133]. Hypocalcemia is also common in critically ill patients [132]. Though the amount varies based on dialysis modality and prescription, KRT generally results in a net delivery of calcium to patients. In some forms of CKRT, patients receive a continuous calcium infusion ("drip") that is adjusted to maintain a normal ionized calcium level. In these situations, the nutritional calcium prescription is not typically adjusted based on the serum calcium level. Hence, it is important that dietitians are aware if a patient is receiving a calcium drip.

Due to decreased excretion, magnesium levels classically increase in AKI, though many critically ill patients develop hypomagnesemia [136–138]. Hence, the nutritional delivery of magnesium is adjusted based on serum levels. KRT results in the net removal of magnesium, with the amount depending on the dialysis modality and prescription [123, 130]. Physicians and dietitians should communicate regarding all changes in the KRT prescription that will influence magnesium removal, including the decision to stop KRT or changes to dialysis modality.

There is no reason to target plasma electrolyte levels differently from the normal range values in healthy children. One exception is the rate at which serum sodium is corrected. Rapid correction of significantly low or high serum sodium values risks neurological complications, so correction should be done gradually [139, 140].

Sodium intake should be adjusted after consideration of both plasma sodium levels and fluid balance. Moreover, water intake has a dramatic effect on plasma sodium values and fluid balance. Hyponatremia in AKI is often due to volume excess, which should be treated by restricting fluid intake rather than by administering additional sodium. Fluid overload is recognized as an independent risk factor for mortality in children with AKI [141]. Sodium restriction is mandatory to mitigate or prevent fluid overload. Thirst is mainly an osmometric process; therefore, a reduction in sodium intake is required when a reduction of water intake is prescribed. Most patients with AKI not treated with KRT will require a restriction of sodium and water intake which, in turn, may limit the provision of optimal nutritional management. The need to restrict fluid and sodium intake may decrease or stop as urine output improves. KRT is very effective at removing sodium and water, often allowing a substantial liberalization of fluid intake. However, hemodynamic considerations may limit fluid removal with intermittent KRT. In contrast, CKRT typically does not require any restrictions of fluid intake since fluid can be removed continuously. However, this may change dramatically when a patient stops CKRT or changes to a different dialysis modality. The physician–dietitian team should communicate regarding the decision to stop KRT or any changes to dialysis modality.

A normal plasma sodium concentration is a reasonable target. However, in case of moderate or profound hyponatremia or hypernatremia, achieving target levels should be approached gradually to avoid serious complications like osmotic demyelinating syndrome (in case of overly rapid correction of hyponatremia) or cerebral edema (when hypernatremia is corrected too fast) [139, 140]. There are rare patients in whom a high-normal or mildly elevated sodium concentration is targeted due to cerebral edema [142].

Results of the Delphi survey

Thirty responses were received via an electronic Delphi survey, comprising 16 dietitians and 18 pediatric nephrologists across 22 countries. Delphi respondents are listed under Acknowledgements as "Participants in Delphi survey." The 37 clinical practice recommendation statements received an overall 88% consensus with a "strongly agree" or "agree" response and 11% with a "neutral" response. There were limited numbers of "disagree" with no respondents reporting "strongly disagree" for any statement. All statements met the stipulated 70% or higher level of consensus. Of note, three statements had a high number of "neutral" responses (4.2, 5.5, 7.2) where we suspect this being potentially due to a lack of experience with nutrition support in critically ill children. Taskforce members reviewed comments and agreed that these

statements did not require alteration as the GRADE reflected the evidence. Otherwise, minor grammatical adjustments were made to two statements to provide better clarification.

Summary of recommendations

A summary of recommendations is provided in Table 6.

Research recommendations

We recommend the following areas of study to provide future evidence-based recommendations for the nutritional management of children with AKI.

- Incorporation of dietitians within pediatric AKI-based studies and collaboratives in order to provide optimal

Table 6 Summary of recommendations

Category	Recommendation	Grade
Collaboration	1.1 Ensure close collaboration between the healthcare professionals medical management and those providing the nutritional prescription for the optimal overall care of children with acute kidney injury (AKI).	Level X; Strong recommendation
Nutrition Assessment	2.1 Utilize a validated pediatric nutrition risk screening tool for the assessment of nutritional risk within 48 hours of AKI diagnosis.	Level B; Moderate recommendation
	2.2 Refer any patient found to be at nutritional risk to a dietitian for nutritional assessment.	Level B; Moderate recommendation
	2.3 Repeat nutritional assessments in accordance with the severity of nutritional risk, severity and duration of AKI, and changes in kidney replacement therapy (KRT).	Level D; Weak recommendation
	2.4 Obtain accurate anthropometric measurements as soon as feasible and throughout hospitalization.	Level A, strong recommendation
	2.4.1 Estimate euvolemic weight using accurate trended weight measurements in conjunction with other clinical assessment measures such as fluid balance, blood pressure, physical examination, and available biometric tools (e.g., bioelectrical impedance analysis, mid-upper arm circumference, non-invasive blood volume monitoring).	Level D; weak recommendation
	2.4.2 Measure height or recumbent length for children under 2 years of age. For those unable to stand for accurate height measurement use recumbent length or a surrogate measurement of height.	Level A; strong recommendation
	2.4.3 Measure head circumference for children up to 2 years of age or up to 3 years of age when appropriate centile charts are available.	Level A; strong recommendation
	2.4.4 Assess for muscle wasting by physical assessment and use of biometric tools where available.	Level D; weak recommendation
Oral & Enteral Feeding	3.1 Oral feeding, including breastfeeding, is the preferred method of providing nutrition.	Level X; strong recommendation
	3.2 In critically ill children, consider early initiation (within 48 hours of admission) of supplemental or exclusive enteral tube feeding when oral feeding does not meet nutritional requirements, especially when nutritional intake is likely to remain suboptimal.	Level C; weak recommendation
	3.3 Use whole protein (polymeric) formulas unless otherwise indicated, such as in case of gastrointestinal dysfunction.	Level C; weak recommendation
	3.4 Consider the use of protein and energy dense formulas to achieve nutritional goals within the limits of the fluid allowance and gastrointestinal tolerance; adjust formula density gradually to maximize tolerance.	Level C; moderate recommendation
Parenteral Nutrition	4.1 For children with malnutrition or risk for nutrition deterioration, when oral or enteral nutrition cannot provide all nutritional requirements, initiate supplemental parenteral nutrition (PN).	Level X, moderate recommendation
	4.2 For children without malnutrition or risk for nutrition deterioration, when oral or enteral nutrition cannot provide all nutritional requirements, PN may be withheld for up to 1 week provided micronutrients are delivered.	Level B; moderate recommendation
	4.3 For all children, regardless of nutritional status, receiving KRT that causes significant nutrient losses, initiation of PN before 1 week should be considered when oral or enteral nutrition cannot provide all nutritional requirements.	Level D; weak recommendation
	4.3.1 Pay careful attention when transitioning from PN to enteral feeding in children receiving continuous kidney replacement therapy (CKRT) to ensure provision of optimal nutrition within the fluid allowance.	Level C; moderate recommendation

Table 6 (continued)

Category	Recommendation	Grade
Energy Requirements	5.1 For non-critically ill children, the initial prescription for energy intake should approximate the suggested dietary intake (SDI) based on euvolemic weight, not measured weight.	Level B; moderate recommendation
	5.2 In the acute phase of critical illness, energy requirements should not exceed the resting energy expenditure.	Level C; weak recommendation
	5.3 In the stable phase and recovery phase of critical illness, the energy prescription must account for energy debt, physical activity, rehabilitation and growth.	Level X; moderate recommendation
	5.4 Modify the energy prescription to account for dialysis-related net gain or loss of energy.	Level C; weak recommendation
	5.5 In critical illness, consider an increased percentage of energy intake from fat to reflect changes in beta-oxidation when parenterally fed.	Level C; weak recommendation
Protein Requirements	6.1 For non-critically ill children, the initial prescription for protein intake should approximate the SDI based on euvolemic weight, not measured weight.	Level B; moderate recommendation
	6.2 For critically ill children, consider the potential need for increased protein intake above the SDI to limit negative protein balance.	Level B; moderate recommendation
	6.3 In children with very elevated blood urea nitrogen levels, especially if progressively worsening, first ensure adequate energy intake; then consider a temporary lowering of protein intake towards the lower end of the SDI.	Level C; moderate recommendation
	6.3.1 Do not persistently compromise protein intake to lower urea nitrogen levels or postpone KRT initiation.	Level X; strong recommendation
	6.4 For all children receiving dialysis, protein prescription needs to be further increased to account for dialysis losses, which are highest in CKRT.	Level C; moderate recommendation
Micronutrient Needs	7.1 In children who are conservatively managed, provide the recommended requirements of vitamins and trace elements for healthy children, with supplementation in case of insufficient intake.	Level D; weak recommendation
	7.2 In children requiring dialysis, consider providing additional supplemental water-soluble vitamins, selenium, copper, zinc and carnitine; either enterally or parenterally.	Level D; weak recommendation
	7.3 Avoid supplemental vitamin A in all children with AKI.	Level B; strong recommendation
	7.4 Evaluate for clinical signs and symptoms of deficiency or excess of vitamins, trace elements and carnitine.	Level C; weak recommendation
	7.5 Do not routinely measure serum concentrations of vitamins, trace elements and carnitine unless there are clinical signs or symptoms of deficiency/toxicity, or when the child is receiving treatment for deficiency or has known toxic concentrations.	Level D; weak recommendation
Electrolyte Monitoring	8.1 Routinely monitor serum sodium, chloride, potassium, calcium, phosphorus, magnesium and bicarbonate ("electrolytes") throughout the course of AKI.	Ungraded*
	8.2 Adjust the frequency of electrolyte monitoring based on laboratory and clinical variables including: trends in electrolyte levels, changes in estimated glomerular filtration rate and urine output, use of or change in KRT prescription or modality, urinary and extrarenal losses of electrolytes and water, adjustments in delivery of electrolytes and fluid to the patient and medications.	Ungraded*
Electrolyte Targets	9.1 Nutritional electrolyte delivery should be individualized and adjusted in close collaboration with the medical team based on ongoing and anticipated changes in clinical status, medications and KRT prescription, generally aiming for the normal blood/serum ranges.	Ungraded*
	9.2 Adjust nutritional and non-nutritional sodium and water delivery to optimize intravascular volume, with the goal to maintain adequate perfusion and prevent or correct volume overload or depletion.	Ungraded*
	9.2.1 Chronically low or high serum sodium values should be corrected gradually to minimize the risk of neurologic complications.	Ungraded*

*A patient's clinical condition and biochemical parameters are usually the primary drivers of fluid and electrolyte management, not nutritional parameters; hence, fluid and electrolyte delivery should be determined in conjunction with the clinical team

nutrition-based metrics that are relevant and meaningful to the growth/development of children and nutrition practice.
- Studies evaluating energy expenditure utilizing indirect calorimetry and assessing the impact of non-nutritive calorie exposure.
- Protein balance studies investigating protein balance in all modalities of acute AKI therapies.
- Studies investigating micronutrient balances in all acute AKI therapies.
- Studies evaluating the effect of nutrition interventions on patient outcomes in all AKI treatment modalities.

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Declarations

Conflict of interest The authors declare no competing interests.

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