

Nutritional considerations in adult patients with acute kidney injury

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Disclaimer

To the best of our knowledge, the contents of this publication are in line with National Institute for Health and Care Excellence guidance relating to the management and treatment of acute kidney injury.

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1. Introduction

Acute Kidney Injury (AKI) encompasses not only kidney disease but a wide spectrum of injury to the kidneys (NICE 2019). It is characterised by a rapid reduction in kidney function resulting in a failure to maintain fluid, electrolyte and acid-base homeostasis (Segaran et al, 2015). The term AKI encompasses a spectrum of injury from moderate to severe deterioration of kidney function where patients may require renal replacement therapy (RRT). The clinical status and treatment of patients with AKI can vary greatly. As a result, nutritional requirements of a patient can vary significantly depending on the degree of AKI, underlying clinical condition, metabolic state, inflammation and treatment employed. Patients require an individualised dietetic approach with close monitoring and review, especially if they require RRT (Kopple et al, 2013).

To date there are no randomised controlled trials on nutrition and AKI, which remains an under researched area. However, given that malnutrition is a strong predictor of mortality in patients with AKI, it seems judicious to optimise nutritional status is key (Li et al, 2012; Meyer et al, 2020; Fiaccadori et al, 2021).

The aim of this document is to provide an overview of nutritional considerations with patients affected by AKI. It is not intended to replace the role of health care professionals. In view of the lack of systematic reviews and high quality studies in the current literature, the recommendations provided are limited to current guidelines and the expert opinions of the authors of this document.

Malnutrition, specifically protein energy wasting (PEW), is an important predictor of in-hospital mortality in patients with AKI, independent of complications and co-morbidities (Fiaccadori et al, 1990; Meyer et al, 2020). Up to 42% of patients with AKI present with signs of severe malnutrition on admission (Cano et al, 2009; Sabatino et al, 2017). Between 24 and 60% of all hospitalised patients with AKI present with a degree of malnutrition (Meyer et al, 2020). In the critical care setting this prevalence increases to as high as 73% (Oh et al, 2019). Moreover, patients with malnutrition who survive to hospital discharge have poor outcomes (Meyer et al 2020). It is therefore important that patients with AKI at risk of malnutrition are identified and where appropriate, referred to the dietitian so that nutritional support and/or dietary electrolyte manipulation can be individually tailored to the patient.

Nutritional support for patients with AKI must take into account not only the specific metabolic disturbances associated with the kidney injury but also the underlying disease process, the potential nutritional losses and gains during RRT, fluid retention (including gut oedema) and retention of uraemic toxins and inflammatory markers (Mercado et al, 2019; Ostermann et al, 2019). It is recognised that patients with AKI represent a heterogeneous group rarely presenting with an isolated disease process. AKI is often present when there is sepsis and multi-organ failure (Kanagasundaram et al, 2019).

2. Identifying patients with AKI at risk of malnutrition

There is currently no validated screening tool to identify malnutrition in people with acute kidney injury. However, early publications of tools in development, include work on the Renal Nutrition Screening Tool, RNST (Xia et al, 2016) and the inpatient nutrition screening tool “iNUT” (Jackson et al, 2019). The Malnutrition Universal Screening Tool (MUST) is fairly specific and correlates well with other nutritional markers but lacks sensitivity when used with people with kidney impairment because weight and weight changes may be masked by fluid changes (Lawson et al, 2012). In clinical practice, independently of the tool used health care professionals caring for patients with AKI should use their clinical judgment to refer the patients to the dietitian if concerned about their nutritional status and/or intake.

Screening tools are often not appropriate in the critical care setting. Instead, the specialist critical care dietitian should undertake a nutritional assessment to determine patients’ nutritional risk and thereby those who will benefit the most from nutrition support (Segaran & Bear 2015).

In addition, to the stages (Stage 1, 2 and 3) of AKI (NICE 2019) the following descriptions are often used in clinical practice: pre-renal, intra-renal and post -renal AKI (Rahman et al, 2012). Table 1 summarises these descriptions and their possible nutritional relevance.

Table 1. AKI terminology and nutritional relevance

Cause	Description	Nutritional relevance
Pre-renal AKI	Pre-renal injuries refer to anything which obstructs the flow of blood to the kidneys. Causes include reduced cardiac output; hypovolaemia; vascular emboli; heart failure; liver failure or drugs which affect the blood supply. Pre-renal injuries are often reversed within a couple of days once normal blood flow is restored. However, if the cause of the pre-renal injury is not reversed or treated effectively it may lead to an intra-renal injury.	There is typically no change in the nutritional requirements of patients with a pre-renal AKI.
Intra-renal AKI	Intra-renal injuries refer to anything which damages the cells of the kidney. Causes include nephrotoxic agents; infections or systemic diseases, e.g. lupus or sarcoidosis; acute tubular necrosis; glomerulonephritis.	This is a more severe form of injury and is likely to alter the nutritional requirements of the patient.
Post-renal AKI	Post-renal injuries refer to anything which obstructs the flow of urine from the kidneys. This often results in hydronephrosis (“water on the kidneys”). Causes include kidney stones; enlarged prostate; cervical cancer or tumours. Post-renal injuries are often reversed within a couple of days once the urinary obstruction is cleared.	There is typically no change in the nutritional requirements of patients with a post-renal AKI.

Patients with pre-existing chronic kidney disease (CKD) may present with AKI. Acute illness in addition to underlying CKD is more likely to impact on and alter nutritional needs. These patients have an increased risk of worsening of their nutritional status when admitted with AKI and should be monitored closely (Meyer et al, 2020). Their nutritional needs will need to take into account the acute illness as well as the CKD.

3. Principles of nutrition support in patients with AKI

The general aim of nutrition support is to maintain nutritional status whilst limiting the complications of AKI. This includes preventing or minimising PEW, preserving lean body mass, avoiding further metabolic derangements, improving wound healing, supporting immune function and thus reducing risk of mortality (Fiaccadori et al, 2021; Meyer et al 2020).

A number of different factors will influence the patient's nutritional needs (Fiaccadori et al, 2021). For those patients identified as being at high risk of malnutrition, an individualised assessment undertaken by a dietitian is recommended (Kanagasundaram et al, 2019).

3.1 Dietetic classification of AKI

From a nutritional point of view patients can be divided into two groups:

- Patients with AKI in the non-catabolic state
- Patients with AKI in the catabolic state

Assessing the presence of a catabolic state is important to guide the dietitian in their nutritional assessment. There is no universal definition of how to identify the degree of catabolism, however, the following factors should be considered. This includes considering both objective measurements of inflammatory markers as well the underlying condition, clinical status, and clinical observation. Typically, patients with an intra-renal injury present in a catabolic state.

AKI in the non-catabolic state

Typically, patients with pre-renal or post-renal injuries present in a non-catabolic state. Common causes of AKI in a non-catabolic state include dehydration, certain medications and urinary obstruction. Generally, these patients are stable and where required any renal replacement therapy (RRT) will usually be provided by conventional haemodialysis (HD). Although they could have any stage of AKI, these are likely to be predominantly patients with stage 1 and 2 AKI. Oral diet alone, or the addition of nutritionally-dense supplementary sip feeds will frequently be sufficient to meet the patients' needs; if not artificial nutrition support should be implemented. Typically, patients with pre-renal or post-renal injuries present in a non-catabolic state.

AKI in the catabolic state

Typically, patients with an intra-renal injury present in a catabolic state. Causes of AKI in a catabolic state include sepsis, acidosis and trauma. Patients often have multi-organ failure and are likely to be managed on an intensive care unit. Although they could have any stages of AKI, these are likely to be predominantly patients with stage 2 and 3 AKI. AKI is seen in approximately 50% patients on ICU (Hostle et al, 2015), of whom 5-10% will require Continuous renal replacement therapy (CRRT) (Tandukar and Palevsky 2019).

PEW is a frequent finding in this group of patients and is associated with poorer patient outcomes in terms of length of hospital stay, complications and mortality rates (Fiaccadori et al, 1999). Protein turnover rates are increased and negative nitrogen balance results. Nitrogen requirements are increased above normal and are influenced by the mode of RRT undertaken. Nutrition can only improve protein and energy balance and possibly protein synthesis but cannot suppress critical illness-induced catabolism (Ostermann et al, 2019).

Patients will usually require artificial nutrition support, particularly if intubated and sedated. Wherever possible, this should be provided via the enteral route. Standard formulae can be used, but nutritionally-dense feeds with or without reduced electrolyte content are useful where the control of fluid balance and/or serum phosphate and potassium levels proves difficult. However, caution may need to be applied in patients on ICU, as nutritionally dense feeds may be less well tolerated.

3.2 The impact of Renal Replacement Therapy (RRT) on nutritional requirements

For patients receiving RRT as a treatment in AKI, nutritional requirements will be affected. All patients with AKI requiring RRT should be referred to the dietitian (Kanagasundaram et al, 2019).

Intermittent haemodialysis (IHD) may be used with stable patients. Fluid removal may be limited and therefore it can be challenging to fully meet nutritional requirements within fluid allowances. Nutrient-dense and low electrolyte feed can be useful. Alternatively, discussion with the medical team to adapt dialysis prescriptions may be necessary to allow a greater volume of feed to be delivered.

Sustained low-efficiency diafiltration (SLED-F) may be used with some patients. Preliminary data showed that unadjusted losses of free amino acids and 18 trace elements in IHD and SLED-F appear to be similar, despite theoretical concerns that the extended hybrid treatment might confer increased risk of micronutrient loss (Oh et al, 2019).

Continuous renal replacement therapy (CRRT), unlike IHD, can be used with patients who have cardio-vascular instability, and therefore is the method of choice for critically ill patients. Care is needed to examine in detail the types and volumes of replacement solutions and dialysis solutions (if applicable) that are used, since these may contain 'hidden' calories. Each millilitre of propofol provides 1.1 kcal, and usually taken into consideration when patient is receiving at least 10 ml/h. Where lactate-containing solutions are being used, the energy derived from its metabolism should be

taken into account. Each millimole of lactate provides 0.32kcal (Casaer et al, 2008). However, in practice it is often difficult to calculate the exact net energy gains. In newer replacement solutions, bicarbonate has replaced lactate as a buffer and consequently there will be no calorie gain. It is also important to account for calorie provision from other medications, primarily propofol, used as a sedative in the critical care setting.

Acute peritoneal dialysis (PD). The use of acute peritoneal dialysis has increased in some centres in the UK following the COVID-19 pandemic. The glucose from the dialysate will inevitably contribute as a source of energy. The following factors affect glucose absorption: dwell time, dialysate glucose concentration, duration of treatment with acute PD and presence of insulin resistance during the inflammatory response. There is very limited evidence, making it is very difficult to estimate the amount of glucose absorbed. We would suggest using a cautionary approach estimating that 35% (or using a range 25-45%) of glucose provided is absorbed. However, this may be not applicable to all patients. Calorie provision from Icodextrin should be excluded as this are negligible (Mafrici et al, 2020). Acknowledging the limited evidence base, protein requirements may be estimated to be 1.2-1.3g/kg/body weight based on the patient metabolic status and dietetic assessment (CCSG 2015).

Another potential source of CRRT derived energy is citrate (an anti-coagulant which sometimes is used instead of heparin solutions), providing 0.59kcal per millimole. However, the net energetic gain depends on the dose infused and the amount removed by CRRT (Oudemans et al, 2012; Balik et al, 2013).

CRRT also has a negative influence on nutrient balance. Extra-corporal losses of amino acids are significant with estimated amino acid losses between 10 and 15g a day (Davenport & Roberts 1989, Davies et al, 1991, Frankenfield et al, 1993, Patel et al, 2017). Total amino acid losses have been shown to be lower in IHD vs SLED-F vs CRRT (Oh et al 2019). In CRRT protein provision should be increased to compensate for these losses and catabolism (Brown & Compher 2010). To achieve these high protein intakes in enterally fed patients without providing excessive amounts of energy, high protein, moderate energy feeds or the addition of modular protein supplements an increase the nitrogen to calorie ratio.

CRRT allows feed volumes and electrolyte intake to be liberalised. Hyperphosphataemia is rapidly corrected, and since most current replacement solutions currently contain no phosphate, intravenous supplementation with phosphate will usually be required if hypophosphatemia is to be avoided (Bellomo & Boyce 1993).

3.3 Nutritional Requirements in AKI

Table 2. Nutritional requirements in AKI. These guidelines are based on low grade evidence and expert opinion, therefore their implementation in clinical practice requires an individualized and patient centred approach.

	Non-Catabolic (no RRT)	Non-Catabolic (on IHD)	Non-Catabolic (on CRRT)	Catabolic (no RRT)	Catabolic (on IHD)	Catabolic (on CRRT)
Protein (g/kg BW/day)	0.8-1.0 (KIDIGO 2012)	Minimum 1.1 (Naylor et al, 2013)	1.2-1.5*	1.0-1.3* Gradually increase to 1.3 (Singer et al, 2019; Fiaccadori et al, 2021)	1.0-1.5 (Cano et al, 2006) 1.5-2.0 (Gervasio 2011; Patel et al, 2017)	Up to 1.7 in hypercatabolism (KDIGO 2012, Singer et al, 2019) 1.5-2.5 /kg/day in critically ill patients with AKI (McClave et al, 2016; Patel et al, 2017)
Energy	Tailored to individual requirements and clinical state (Some authors suggest 20-30kcal/kg/day even in the critical settings, KIDGO 2012)					
Fluid	Fluid requirements require individual medical assessment. Standard fluid equations are unlikely to be helpful. Fluid balance and daily weights should be monitored closely.					
Electrolytes	Monitor and adjust intake as required. These will vary depending on disease state and type of treatment.					
Micronutrients	Requirements are not well documented. Lipid soluble vitamin levels and antioxidant status are low. CRRT has negative effect on balance of some water-soluble vitamins (such as thiamine, folate and vitamin C) and trace elements (copper, zinc, selenium). Whether micronutrient supplementation improves outcomes remain unknown (Oh et al, 2019).					
All recommendations for kg/body weight (BW) refer to actual body weight, however special considerations to avoid overfeeding and underfeeding should be taken into account in individual who are underweight or obese.						

*There is currently no evidence to support these protein requirements and values are provided based on the expert opinion of the authors. If protein provision is increased further close monitoring of kidney function, patient condition and nitrogen balance will be required. A target of 1.3 g/kg/day should be reached progressively in patients on ICU with AKI and should be adapted to the clinical conditions (Singer 2020).

Energy

AKI itself has no effect on the patient's energy requirements. Even in individuals with AKI and multi-organ failure, measured energy requirements are only 20%-30% above the estimated basal metabolic

rate values (KDIGO 2012). KDIGO guidelines for AKI (2012) suggest achieving a total energy intake of 20–30 kcal/kg/day in patients with any stage of AKI. Others suggest to 25-35kcal/kg/day, in the critically ill patient with AKI (Gervasio et al, 2011; McClave et al, 2016; Singer et al, 2019).

Critical care specific equations may be used for those on ICU to avoid risks of over and underfeeding (Frankenfield et al, 2004). However, predictive formulas may frequently lead to incorrect energy need estimation, as a result in the absence of indirect calorimetry close monitoring is needed (Sabatino et al, 2017). In those receiving CRRT, the energy contribution from solutions that use lactate as a substrate or citrate as an anticoagulant should be taken into account, as previously discussed.

In acutely unwell patients with AKI on ICU, hypocaloric feeding (not exceeding 70% of energy expenditure) should be administered for the first 3 days of ICU admission (ESPEN 2019). After day 3, caloric delivery can be increased up to 80-100% of measured EE (ESPEN 2019, Fiaccadori et al, 2021).

Protein

Protein requirements are influenced by the patient's clinical condition, severity of AKI, presence or absence of a catabolic state and any RRT they may receive (Table 2). Recommendations for protein requirements vary in the literature. It is essential that attention is given to individual patient condition and treatment goals, when considering the protein requirements as suggested in Table 1. For stable patients who are not receiving RRT, protein turnover is not increased; a protein intake of 0.8–1.0 g/kg/day has been suggested (Gervasio et al, 2011; KDIGO 2012). In contrast, patients receiving RRT will require a higher protein intake, with a suggested range from 1.5-2.5g/kg/day (Cano et al, 2006; Gervasio et al, 2011; McClave et al, 2016; Patel et al, 2017). It is important to remember that these recommendations are based on small sample sizes (n = 7-50), the majority of who were fed via parenteral nutrition (Brown et al, 2010; Patel et al, 2017).

Concerns have previously been raised regarding the safety protein intakes greater than 2.5g/kg/day, with fears the administered nitrogen may be used as an energy source or could even have a pro-inflammatory effect (Casaer et al, 2008). In practice, in patients on CRRT who are metabolically stressed 1.7g/kg/BW is a more realistic target (KDIGO 2012; Patel et al, 2017; Fiaccadori et al, 2021). There is no indication to reduce protein administration in fear of increased urea. This increase may be controlled by dialysis if it occurs (Singer et al, 2020).

Electrolytes

AKI may be associated with significant electrolyte changes such as elevated potassium and phosphate levels. Electrolyte intake should be individualised according to blood biochemistry, patient clinical condition as well as dietary provision. Frequent monitoring of blood biochemistry is essential. Patients requiring dietary electrolyte manipulation should be referred to the dietitian regardless of their risk of malnutrition. Low potassium and low phosphate diets can be implemented where serum levels are high.

Avoidance of hyperkalaemia is a clinical priority in AKI, however not all patients with AKI and hyperkalaemia require dietary potassium restriction. Health care professionals should consider the cause of hyperkalaemia; the presence of metabolic acidosis and other non-dietary related causes as well as the contribution of potassium from dietary intake and provision via enteral and parenteral solutions where applicable. Recently, there has been an increased use of gastrointestinal cation-exchange resins to support in the emergency management of acute life threatening hyperkalaemia (Meyer et al, 2020; Selby et al. 2020). If and when kidney function recovers and serum potassium and phosphate levels normalise any restrictions can be lifted. If and when kidney function recovers,

serum potassium and phosphate levels normalise and any restrictions can be lifted. In the polyuric phase if low electrolyte levels occur, oral or intravenous supplementation may be required.

The dietary restriction of phosphate may limit an individual's food choice. Allowing more liberal phosphate consumption to help promote food intake is a better approach. In the short-term, maintaining a good nutritional intake is of greater priority than achieving phosphate control. persistent hypophosphatemia in critically ill patients has been correlated with increased mortality (Meyer et al, 2020). Hypophosphatemia may be an effect of the RRT employed, particularly in CRRT in which case the treatment is likely to be pharmacological. The nutritional intake of patients with hypophosphatemia, particularly in those who are not undergoing RRT, should be scrutinised to ensure that a deficiency in protein intake is not present.

Fluid

The medical team should advise on fluid provision taking into account clinical condition, stage of AKI and current treatment. Equations to estimate fluid requirements may be not useful in AKI. Instead a multidisciplinary team approach is needed to establish the optimal fluid intake for the patient as well as the volume within which nutrition support can be given if required. Daily weights, strict fluid balance and medical assessment are key tools to assess optimal fluid requirements. In oliguric and fluid overloaded patients, fluid intake may be restricted. Limiting sodium intake will help control thirst and aid adherence with fluid restriction. During recovery patients may become polyuric. An increased fluid intake (adequate to cover the large urine volumes and insensible losses) must then be maintained.

Micronutrients

Requirements for micronutrients are not well defined. Caution should be taken when interpreting serum micronutrient levels; many patients with AKI present in the acute phase response, which will itself alter blood levels (Gervasio et al, 2011). Plasma levels of vitamins A, D, E (Druml et al, 1998) and vitamin C (Story et al 1999) together with selenium and zinc (Story et al, 1999; Berger et al, 2004; Oh WC et al, 2019) are lower in AKI than in normal subjects. However, this may be related to inflammatory processes, RRT or medication rather than true nutritional deficiencies. Micronutrient losses on CRRT may also be significant. Documented daily ultrafiltrate losses include 100mg vitamin C (Bellomo & Boyce 1993), 290g folate (Fortin et al, 1999) and 4mg thiamine (Berger et al, 2004), with some authors suggesting supplementation (Gervasio et al, 2011). Megadoses of vitamin C (>250mg/day) are potentially toxic due to the risk of secondary oxalosis in kidney failure, although ensuring adequate ascorbic acid status may confer some benefit (Honore et al, 2020). Vitamin A toxicity has been reported in patients with AKI receiving parenteral nutrition; some authors suggest monitoring Vitamin A levels (Gervasio et al, 2011).

Trace elements circulate mainly bound to protein and therefore are generally unaffected by CRRT but may be affected by the acute phase response. Selenium is affected by CRRT with reported daily losses of 0.97 μ mol (Berger et al, 2004) some authors suggest a need for its supplementation (Gervasio et al, 2011).

Since the provision of micronutrients from commonly used parenteral sources and enteral feeds may be insufficient to replace some of these losses to meet requirements, there is some suggestion that selective supplementation should be considered (Gervasio et al, 2011). However, demonstrating deficiency of micronutrients in patients with AKI does not equate to demonstrating a clinical benefit from supplementation; it is not known whether micronutrient supplementation to compensate for

RRT losses improves outcomes (Oh et al, 2019; Meyer et al, 2020). Current guidelines lack specific recommendations on specific micronutrient doses and duration.

3.4 AKI and COVID-19

COVID-19 associated AKI is common in patients hospitalised with Covid-19. It is associated with high mortality and is an independent risk factor for all-cause in-hospital death (Cockwell et al, 2020; Nadin et al, 2020). In an American study, of 3993 hospitalized patients with COVID-19, AKI occurred in 1835 (46%) patients (19% of which required dialysis). Of all patients with AKI, only 30% survived with recovery of kidney function by the time of discharge (Chan et al, 2021). Based on data collected from ICNARC 26.8% of patients admitted to intensive care with Covid-19 required some form of renal support (admission date up to 31st August 2020). Note this number was lower in the second surge 14.5% since September 1st (data up to the 12th Feb 2021).

Based on current experience with COVID-19, a large proportion of patients are likely to develop AK (Bear and Terblanche 2020). Patients with COVID-19 are at risk of malnutrition due to various factors such as prolonged immobilization, catabolic changes and reduced food intake. Insulin resistance and a hypercatabolic state are common in COVID-19 and contribute to hyperglycaemia (Nadin et al, 2020). However, no dedicated studies on nutritional management in patients with COVID-19 AKI exist. Therefore, in patients with COVID-19, the existing recommendations for the nutritional management of critically ill patients with AKI should be followed (Minelli et al, 2020) whilst taking an individualised and patient centred approach (see Table 2) (Bear and Terblanche 2020).

4. Monitoring and Conclusion

Where patients require dietary input it is important that they receive regular dietetic review throughout the course of treatment and during recovery. Energy, protein and fluid requirements will require review as kidney function changes. Where kidney function recovers and electrolyte and fluid balance normalise, patients should be supported to relax restrictions and return to a normal balanced diet.

Nutritional management plays an important role in the care of patients with AKI. Since different disease types and AKI stages can affect patients in a variety of ways, nutrition is best tailored to individual needs, taking account AKI stage, clinical condition and treatment whilst using clinical judgement. Data is lacking on the optimal intake of a variety of nutrients. There is some guidance on energy and protein requirements based on the limited evidence available. Individualised assessment enables the nutritional prescription to be matched to the various needs of patients. It is recommended that this be undertaken by a dietitian.

5. Dietetic audit measures in AKI

All patients should be screened for risk of malnutrition within 24 hours of admission (NICE 2006)
The Renal Association guidelines for AKI (Kanagasundaram et al, 2019) recommend that patients with AKI receiving renal replacement therapy should be referred to a dietitian for individual assessment:

- Proportion of patients undergoing dietetic review by the calendar day after initiation of renal support (applies even if no longer RRT-dependent)
- Proportion of patients meeting at least 80% of their estimated energy and protein requirements by the 2nd calendar day after initiation of renal support (applies even if no longer RRT-dependent).

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