

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 failure but a wide spectrum of injury to the kidneys (NICE 2013). 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) (AKIN 2015). 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 and treatment employed. Patients require an individualised dietetic approach with close monitoring and review (Kopple et al, 2013).

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). Up to 42% of patients with AKI present with signs of severe malnutrition on admission (Cano et al 2009). Furthermore, other studies suggest this prevalence increases to as high as 73% in critically ill patients (Oh et al 2015). It is therefore important that patients with AKI at risk of malnutrition are correctly identified. Where appropriate, patients should be referred to the dietitian so that nutritional support and/or dietary electrolyte manipulation can be individually tailored to the patient's nutritional needs.

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. It is recognised that patients with AKI represent a heterogeneous group rarely presenting with an isolated disease process but often in association with sepsis and multi-organ failure (Lewington et al 2011).

2. Identifying patients with AKI at risk of malnutrition

Nutrition support guidelines published by the National Institute of Clinical Excellence (NICE 2006) state that:

“...all hospital inpatients on admission and all outpatients at their first clinic appointment should be screened for malnutrition. Screening should be repeated weekly for inpatients and when there is clinical concern for outpatients.”

NICE recommend the use of the Malnutrition Universal Screening Tool (MUST) in order to screen for malnutrition in hospital. 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). There is currently no validated screening tool to identify malnutrition in people with kidney problems. 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 2017). In clinical practice, independently of the tool used (either MUST, RNST, iNUT or locally developed tools), 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 of AKI (NICE 2013) to describe the cause of AKI the following description is often used in clinical practice: pre-renal, intra-renal and post-renal AKI (Rahman et al 2012). Table 1 summarised this description and their possible relevance from a nutritional point of view.

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; 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.	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.

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 1990). A number of different factors will influence the patient's nutritional needs. For those patients identified as being at high risk of malnutrition, 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 (Lewington et al 2011).

a. 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

Despite a lack of universal definition of how to identify the degree of catabolism, assessing the presence of a catabolic state helps to guide the dietitian in their nutritional assessment. 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

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 with 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

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. 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. Although hypercatabolism cannot be reversed by simply increasing protein intake, appropriate nutrition support may reduce nitrogen losses (Patel et al 2017).

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.

The use of novel substrates in critically ill patients in intensive care units (ICU) remains contentious (Kreymanna et al 2006, Canadian Clinical Guidelines Committee 2015). It is not known whether immune modulating enteral formulae confer any benefit to patients with AKI.

Where the enteral route cannot be used for feeding, parenteral nutrition support should be considered.

Renal Replacement Therapy (RRT)

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 (Lewington et al 2011).

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 in anuric patients where tight fluid balance is required. Nutrient-dense and low electrolyte feeds can prove helpful. 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 in 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 2015).

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. It is also important to account for calorie provision from other medications, primarily propofol, used as a sedative in the critical care setting). 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.

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 an 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). Protein provision should be increased to compensate for these losses and the patient's catabolic rate (Brown & Compher 2010). To achieve these high protein intakes in enterally fed patients without providing excessive amounts of energy, high protein feeds or the addition of protein-rich supplements to feeds can 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).

b. Nutritional Requirements

Table 2. Summary table of nutritional requirements in AKI based on current guidelines. 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*	At least 1.0*	1.0 -1.5 (Cano et al 2006) 1.5-2.0 (Gervasio 2011; Patel et al 2017)	Up to 1.7 in hypercatabolism (KIDIGO 2012) 1.5-2.5 /kg/day in critically ill patients with AKI (McClave 2016, Patel et al 2017)
Energy	Not affected by AKI. Tailored to individual requirements and clinical state Some authors suggest 20-30kcal/kg/day even in the critical settings (KIDGO 2012). Others instead promote 25-30kcal/kg/day patients in ICU with AKI (Cano 2009, Brown 2010, Gervasio 2011; McClave 2016).					
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. Will vary depending on disease state and type of treatment.					
Micronutrients	Requirements are not well documented. Lipid soluble vitamin levels and anti-oxidant status are low. CRRT has negative effect on balance of some vitamins and trace elements. Whether micronutrient supplementation improves outcomes is unknown (Oh et al 2015).					
All recommendations for kg/body weight (BW) refer to actual body weight, however special consideration to avoid overfeeding should be taken into account in obese individual.						

*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.

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 (PEN and 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).

Critical care specific equations may be used for those on ICU to avoid risks of overfeeding (Frankenfield et al 2004). 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.

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). Recommendation for protein requirements vary in the literature. It is essential that attention is given to individual patient condition, when considering the protein requirements as suggested in table 1. For stable patients with stage 1 AKI 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 with stage 2 and 3 AKI 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).

Electrolytes

AKI may be associated with significant electrolyte changes such as elevated potassium 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. However, where the dietary restriction of phosphate may restrict 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. Avoidance of hyperkalaemia takes greater clinical priority, however not all patients with AKI and hyperkalaemia require dietary potassium restriction. Health care professionals should consider the cause of hyperkalaemia; take into account metabolic acidosis, other non-dietary related causes as well as considering dietary intake and provision via enteral and parenteral solutions if applicable.

If and when kidney function recovers serum potassium and phosphate levels normalise any restrictions can be lifted. In the polyuric phase oral or intravenous supplementation may be required.

Fluid

Fluid provision should be advised by the medical team 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 indicators 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 2015) 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 because of the danger of secondary oxalosis in kidney failure. 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 0.97µmol (Berger et al 2004) and some authors suggest 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 2015). Current guidelines lack specific recommendations on specific micronutrient doses and duration.

Monitoring and review

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.

Conclusion

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, treatment and good 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.

4. Dietetic audit measures in AKI

- All patients should be screened for risk of malnutrition within 24 hours of admission (NICE 2006)

Renal Association guidelines for AKI (2011) have suggested the following audit measures specifically for AKI stage 3:

- Proportion of patients with AKI receiving renal replacement therapy reviewed by dietitian within 24 hours
- Proportion of patients with AKI receiving renal replacement therapy prescribed the recommended nutritional support.

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