Overview of nutritional considerations in the treatment of adult patients with acute kidney injury in hospital

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This document is based on extracts from the British Dietetic Association Parenteral and Enteral Nutrition (PEN) “Pocket Guide for Clinical Nutrition” (2011) authored by George Hartley, Dietetics Manager, Newcastle Upon Tyne Hospitals NHS Foundation Trust. The document will be updated following the planned review of the PEN guide in 2016.

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) is characterised by a rapid reduction in kidney function resulting in a failure to maintain fluid, electrolyte and acid-base homeostasis (Renal Association 2011). 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, and following this their nutritional status and nutritional requirements, can vary greatly.

The aim of this document is to provide an overview of the nutritional considerations in the treatment of patients affected by AKI. It is not intended to replace the role of health care professionals. The recommendations described are limited to expert opinion (KDIGO 2012; Renal Association 2011) in view of the lack of systematic and high quality studies in the current literature.

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 (ESPEN 2009). Therefore it is important that patients with AKI at risk of malnutrition are correctly screened. 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 (Renal Association 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 renal 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. In clinical practice, independently of the tool used (either MUST 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).

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, avoiding further metabolic derangements and thus reducing risk of mortality (Fiaccadori et al 1990). The patient’s nutritional requirements will be influenced by a number of different factors. 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 (Renal Association 2011).

a. Dietetic classification of AKI

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

- AKI in the non-catabolic state
- AKI in the catabolic state

This classification helps to guide the dietitian in their nutritional assessment, especially during the first consultation with patient.

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). 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.

AKI in the catabolic state

The causes of AKI in a catabolic state include sepsis and trauma. Patients often have multi-organ failure and will be managed on an intensive care unit. They present complex therapeutic challenges. PEW is a frequent finding 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 also influenced by the mode of RRT undertaken. Although hypercatabolism cannot be reversed by simply increasing protein intake, appropriate nutrition support will reduce nitrogen losses (Murphy et al 1993).
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 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 those receiving RRT as a treatment in AKI, nutritional requirements will be affected. All patients with AKI requiring RRT should be referred to the dietitian (Renal Association 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.

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. 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 new energy gains. In newer replacement solutions, bicarbonate has replaced lactate as a buffer and consequently there will be no calorie gain. The use of dextrose based dialysis solution is now largely outdated, however if used gains in energy will need to be taken into account (Bellomo et al 1991).

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 approximately 10% of infused amino acids being lost during both haemofiltration (Frankenfield et al 1993, Davenport & Roberts 1989) and haemodiafiltration (Davies et al 1991). Protein intakes 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

Summary table of nutritional requirements in AKI

<table>
<thead>
<tr>
<th>Protein (g/kg/day)</th>
<th>Non-catabolic (no RRT)</th>
<th>Non-catabolic (on IHD)</th>
<th>Catabolic (on IHD)</th>
<th>Catabolic (on CRRT)</th>
</tr>
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<td></td>
<td>0.8-1.0 (KIDGO 2012)</td>
<td>Minimum 1.1 (Naylor et al 2013)</td>
<td>1.5 (Cano et al 2006)</td>
<td>Up to a maximum of 1.7 in hypercatabolism (Renal Association 2011, Cano et al 2006, Cano et al 2009)</td>
</tr>
</tbody>
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Energy

- Not affected directly by AKI itself
- Tailored to individual requirements and clinical state (presence or absence of catabolism).
- Standard formulae to estimate requirements can be used
- 20-35kcal/kg/day range suggested by KIDGO (2012) & RA (2011)

Fluid

- For individual assessment, fluid intake as advised by medical team.
- Standard fluid recommendations 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. Fat 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 WC et al 2015).

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). Standard formulae can be used to estimate energy requirements (PEN 2012). Alternatively, KDIGO guidelines for AKI (2012) suggest achieving a total energy intake of 20–30 kcal/kg/day in patients with any stage of AKI. Renal Association guidelines (2011) suggest a slightly higher aim of 25-35kcal/kg/day. Individual assessment of energy requirements should be made by the dietitian.

Critical care specific equations should be used for those on intensive care units (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.
Protein
Protein requirements are influenced by the patient’s clinical condition and any RRT they may receive.

For non-catabolic patients who are not receiving RRT, protein turnover is not increased. KDIGO guidelines suggest avoiding restriction of protein intake with the aim of preventing or delaying initiation of RRT and suggest protein intakes of 0.8–1.0 g/kg/day.

For those individuals receiving IHD, protein intakes should be increased to a minimum of 1.1g/kg/day (Naylor et al 2013). Higher intakes of up to 1.5g/kg/day have been recommended to meet needs in catabolic patients (Cano et al 2006).

Optimal protein requirements in CRRT and hypercatabolism are unknown but it is suggested that they may be increased up to a maximum of 1.7 g/kg/day (Renal Association 2011, Cano et al 2006, Cano et al 2009). There have been concerns over the safety protein intakes higher than 2.5g/kg/day, as the administered nitrogen may be used as an energy source or could even have a pro-inflammatory effect (Casaer et al 2008).

Electrolytes
In addition to malnutrition, AKI may be associated with significant electrolyte changes such as elevated potassium levels. Electrolyte intake should be individualised according to blood biochemistry. Frequent monitoring of blood biochemistry is essential to guide provision. 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 renal function recovers serum potassium and phosphate levels normalise any restrictions can be lifted.

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. 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). Trace elements circulate mainly bound to protein and therefore are generally unaffected by CRRT. Selenium is an exception with reported daily losses of 0.97µmol (Berger et al 2004). However, demonstrating deficiency of micronutrients in patients with AKI does not equate to demonstrating a clinical benefit from supplementation (Oh WC et al 2015). Studies that assess supplementation of micronutrients and clinical outcomes in AKI are lacking.

Since the provision of micronutrients from commonly used parental sources and enteral feeds may be insufficient to cover these losses and meet requirements, there is some suggestion that selective supplementation should be considered (Renal Association 2011). It is not known whether micronutrient supplementation to compensate for RRT losses improved outcomes (Oh WC et al 2015). Current guidelines lack specific recommendations on specific micronutrient doses and duration. Megadoses of vitamin C are potentially toxic because of the danger of secondary oxalosis in renal failure. Cases of severe oxalosis have been reported in individuals with AKI receiving daily vitamin C supplement of 500mg intravenously (Friedman et al 1983) or 500mg orally (Mashour et al 2000). More research is needed in this field.

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 renal function changes. Where renal 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 clinical condition and treatment. 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. This document should serve to inform of the nutritional considerations in the management of AKI. 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.

KDIGO (2012) give the following research recommendations:

- The risk-benefit ratio of diets with low, medium, and high protein contents in different stages of AKI should be addressed in RCTs
- Given gastrointestinal tract dysfunction in AKI, the possible benefit of enteral vs. parenteral feeding in AKI patients should be further evaluated in prospective RCTs.
5. References


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6. **Acknowledgements**

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