Nutrition Support Therapy in Acute Kidney Injury: Distinguishing Dogma From Good Practice

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Introduction

Acute kidney injury (AKI) is a frequently observed complication in critically ill patients. Its presentation may range from the early risk of renal dysfunction to complete renal failure. Morbidity and mortality in the AKI patient increase with the decline of renal function. Appropriate nutrition therapy is essential in the medical management of the AKI patient. Assessment of nutritional requirements should take into account the patient’s underlying complication, comorbid medical conditions, and severity of the renal dysfunction. Various stages of AKI determine the direction of nutrition therapy. Additionally, understanding the macro- and micronutrient modifications and electrolyte and vitamin alterations that should be implemented are vital for better patient outcomes.

AKI Classification

In the past, “acute renal failure” was defined in a multitude of ways based on differing clinical and laboratory features. Failure to define and classify the various stages of acute renal failure led the Acute Dialysis Quality Initiative (ADQI) group to collaborate, define, and identify a staging and outcomes system for acute renal failure [2,3]. Foremost, the term “acute renal failure” was replaced by “acute kidney injury,” a more encompassing term. The ADQI consensus definition of AKI is denoted by the acronym RIFLE, which refers to three stages of increasing severity classes—risk, injury, and failure—and two outcome classes—loss and end-stage renal disease. The three severity classes are based on combined criteria of serum creatinine concentration or glomerular filtration rate and urine output, with the worst of each criterion used. The two clinical outcome criteria—loss and end-stage kidney disease—are based on the relation to duration of loss of kidney function [2,3].

Although the RIFLE criteria took the first step toward defining and classifying AKI, emerging data imply that smaller changes in serum creatinine than those initially considered might be associated with adverse outcomes; subsequently, the Acute Kidney Injury Network modified the RIFLE criteria so that patients meeting the definition for AKI could be diagnosed and staged [4••]. The proposed staging system retains the emphasis on acute alterations in serum creatinine and/or urine output.

According to the Acute Kidney Injury Network [4••], diagnostic criteria for AKI include an abrupt (within 48 hours) reduction in kidney function, currently defined as an absolute increase in serum creatinine ≥ 0.3 mg/dL (≥ 26.4 μmol/L), a percentage increase in serum creatinine ≥ 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria < 0.5 mL/kg per hour for > 6 hours). Three stages for AKI were implemented based on the modified RIFLE criteria (Table 1) [4••].

Nutrition Assessment in AKI

Malnutrition is an independent risk factor for hospital mortality and is common in patients with AKI. Anorexia, impaired protein metabolism and transport, oxidative stress, metabolic acidosis, nutrient losses through the hemodialyzer, and patient comorbidities are contributing factors to malnutrition [5]. Additionally, hypermetabolism
A creatinine clearance of more than 50 mL/min/1.73 m², the method for measuring a patient's nitrogen balance requires degradation. For an accurate determination, the standard and protein catabolic rate (PCR; measurement of net protein (UNA; measurement of the net rate of protein catabolism), intake minus nitrogen output), urea nitrogen appearance (UUN, measurement of net protein degradation). For an accurate determination, the standard method for measuring a patient's nitrogen balance requires a creatinine clearance of more than 50 mL/min/1.73 m², which makes utilizing the nitrogen balance in patients with AKI difficult. In patients with AKI, ascertaining the UNA is less laborious and more accurate. Table 2 describes UNA and PCR calculation [8,9].

**Table 1. Staging system for acute kidney injury**

<table>
<thead>
<tr>
<th>AKI stage</th>
<th>RIFLE classification</th>
<th>Serum creatinine</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>R</td>
<td>Increase in serum creatinine ≥ 0.3 mg/dL or increase to ≥ 150%–200% (1.5- to 2-fold) from baseline</td>
<td>&lt; 0.5 mL/kg per hour for &gt; 6 h</td>
</tr>
<tr>
<td>2</td>
<td>I</td>
<td>Increase in serum creatinine to &gt; 200%–300% (&gt; 2- to 3-fold) from baseline</td>
<td>&lt; 0.5 mL/kg per hour for &gt; 12 h</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>Increase in serum creatinine to &gt; 300% (&gt; 3-fold) from baseline or serum creatinine ≥ 4.0 mg/dL with an acute increase of at least 0.5 mg/dL</td>
<td>&lt; 0.3 mL/kg per hour for 24 h or anuria for 12 h</td>
</tr>
</tbody>
</table>

AKI—acute kidney injury; RIFLE—risk, injury, failure, loss, end-stage renal disease.
(Data from Mehta et al. [4••].)

**Table 2. Urea nitrogen appearance and protein catabolic rate calculations**

| UNA, g/d = UUN + [(BUN2 - BUN1) × 0.6 × BW1] + [(BW2 - BW1) × BUN2] |

Where: Net protein breakdown = UNA × 6.25; UUN = urinary urea nitrogen, g/d; BUN1 = initial collection of blood urea nitrogen, predialysis, g/L; BUN2 = final collection of blood urea nitrogen, predialysis, g/L; BW1 = postdialysis weight, kg; BW2 = predialysis weight, kg

PCR, g/d = UNA × 6.25

*Protein intake fluctuations and varying catabolic presentations may result in the calculation of erroneous results.
BUN—blood urea nitrogen; BW—body weight; PCR—protein catabolic rate; UNA—urea nitrogen appearance; UUN—urinary urea nitrogen.

and hypercatabolism are common because AKI is a complication resulting from surgery, trauma, thermal injury, multiorgan failure (MOF), and sepsis. Proper nutrition aimed at minimizing these effects is imperative [5,6].

Nutritional evaluation of a patient with AKI includes physical assessment, anthropometric measurements, and laboratory values. However, because of the disease process, the patient’s resulting edema may alter anthropometric measurements, and fluid retention and declining renal function may potentially result in erroneous visceral protein markers (e.g., albumin, prealbumin). Body weight before the onset of illness usually is a better representation of the patient’s “true” weight than their actual body weight. Weight gains of 0.5 to 1 kg/d usually represent fluid retention and must not be used when determining caloric/protein nutritional goals and modifications [7].

The futility of albumin and prealbumin as markers to identify malnutrition and nutrition response in AKI has led investigators to identify an alternative marker. Insulin growth factor has been investigated and successfully used to assess a renal patient’s response to nutritional support as well as a specific marker for malnutrition in patients undergoing hemodialysis. However, because information is limited, further research is necessary before insulin growth factor can be recommended for routine use [7].

Also used to assess protein breakdown in the AKI patient are nitrogen balance (measurement of nitrogen intake minus nitrogen output), urea nitrogen appearance (UNA; measurement of the net rate of protein catabolism), and protein catabolic rate (PCR; measurement of net protein degradation). For an accurate determination, the standard method for measuring a patient’s nitrogen balance requires a creatinine clearance of more than 50 mL/min/1.73 m², which makes utilizing the nitrogen balance in patients with AKI stage 1 AKI is associated with pre- or postrenal injury. Identification of fluid status and protein delivery is necessary. Inadequate hydration may be corrected by fluid replacement and/or appropriate modification of diuretic therapy. Azotemia resulting from overzealous protein delivery may be remedied by decreasing the provision of protein. Prerenal injury resulting from excess volume may require sodium and fluid restriction while monitoring diuretic response. Obstruction resulting in postrenal injury requires correction of the obstruction.

Nutritional energy and protein requirements in stage 1 AKI are based on the patient’s underlying disease state or complication. Stage 1 AKI has a limited effect on energy expenditure. Likewise, the provision of electrolytes, vitamins, minerals, and trace elements is based on the patient’s presentation and accompanying complications.

**Nutrition Support Therapy**

**AKI stage 1**

Stage 1 AKI is associated with pre- or postrenal injury. Identification of fluid status and protein delivery is necessary. Inadequate hydration may be corrected by fluid replacement and/or appropriate modification of diuretic therapy. Azotemia resulting from overzealous protein delivery may be remedied by decreasing the provision of protein. Prerenal injury resulting from excess volume may require sodium and fluid restriction while monitoring diuretic response. Obstruction resulting in postrenal injury requires correction of the obstruction.

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**AKI stage 2**

In stage 2 AKI, the focus is on avoiding exacerbation of the kidney injury and, usually, on correcting a state of either volume depletion or excess. The need for nutrition support therapy varies in stage 2 AKI and, when necessary, can have a therapeutic and/or supportive role. Significant blood loss, diuretic abuse, prolonged vomiting, diarrhea, lack of access to adequate hydration, and abdominal vascular surgery can lead to renal hypoperfusion, requiring aggressive resuscitation. Abdominal