Nutrition in patients with acute renal failure

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Introduction

Acute renal failure (ARF) is a common disease affecting approximately 5% of all hospitalized patients and 10–30% of the patients in intensive care units. The mortality rate is still very high (above 50%), despite advances in the management of critically ill patients and in the renal replacement therapies. Pre-existing or hospital-acquired malnutrition is an important factor contributing to high mortality seen in patients with ARF [1–4]. Nutritional support has, therefore, been accepted as an important part of the management of ARF.

During the course of ARF, multiple metabolic changes occur. ARF affects both fluid, electrolyte, acid-base balance and the metabolism of proteins, amino acids, carbohydrates, lipids and energy. The metabolic alterations in ARF patients are determined not only by acute loss of renal function, but also by the type and intensity of renal replacement therapy. Furthermore, the underlying disease plays an important role in the development of metabolic changes [2,4,5].

Protein and amino acid metabolism

The hallmark of metabolic changes in ARF is protein catabolism and negative nitrogen balance. The causes of hypercatabolism are complex and manifold. The major stimulus of protein catabolism in ARF is insulin resistance. The maximal insulin-stimulated protein synthesis is decreased and protein degradation is increased. As a result, excessive release of amino acids from muscle occurs, and gluconeogenesis and ureagenesis are increased due to hepatic uptake of these amino acids from the circulation. On the other hand, synthesis of some amino acids in the kidney is impaired, while catabolism of peptide hormones is retarded because of renal failure. Acidosis and release of proteases from activated leukocytes can stimulate protein breakdown. Moreover, inflammatory mediators such as tumor necrosis factor-α (TNF-α) and interleukins as well as renal replacement therapy by itself can mediate hypercatabolism. Additionally, inadequate nutrition contributes to the loss of lean body mass in ARF [2,4–7].

Carbohydrate metabolism

ARF is frequently associated with hyperglycaemia mainly because of insulin resistance. Despite the clearance of insulin is decreased and plasma insulin concentration is elevated, maximal insulin-stimulated glucose uptake by skeletal muscle is decreased by 50%. Hepatic gluconeogenesis, mainly from conversion of amino acids released during protein catabolism occurring in ARF, is increased and is insensitive to the negative feedback that is normally activated by glucose loading. The synthesis of glycogen within the muscles is also deficient [2,4,5].

Lipid metabolism

In patients with ARF, triglyceride content of plasma lipoproteins is elevated. Total cholesterol and in particular high-density lipoprotein cholesterol levels are decreased whereas low-density lipoprotein cholesterol and very-low-density lipoprotein cholesterol are increased. The main cause of these abnormalities is the impairment of lipolysis [4,5].

Energy metabolism

Energy expenditure remains unchanged and near-normal in uncomplicated ARF such as monofactorial ARF. In contrast, oxygen consumption and resting energy expenditure increases by 30% and even more when sepsis or systemic inflammatory response syndrome is associated with ARF [2].
Metabolic effects of renal replacement therapies

Renal replacement therapies, especially extracorporeal therapies (haemodialysis and continuous renal replacement therapies (CRRT)) have significant metabolic and nutritional consequences. Protein catabolism is increased via substrate losses, activation of protein breakdown from release of leukocyte-derived proteases, and release of cytokines such as TNF-α and interleukins stimulated by blood–membrane interaction during dialysis. Membranes used in haemofiltration are more porous and small proteins are also filtered. Moreover, many water soluble substances such as vitamins and carnitine are lost during extracorporeal therapies. Peritoneal dialysis as a renal replacement therapy especially results in protein and amino acids losses [2,4–6].

Nutrient requirements in patients with ARF

If there is no associated hypercatabolic state, the intake of protein or amino acid should be 1.0 g/kg body weight/day. Hypercatabolic patients with ARF should receive 1.2 g, maximally 1.5 g protein/amino acid/kg body weight/day. These amounts include amino acid/protein losses by intermittent haemodialysis, any form of CRRT or by peritoneal dialysis [5,7]. Carbohydrates are considered as the main source of energy in patients with ARF. Glucose, not exceeding 5 g/kg body weight/day is acceptable. Recommended prescription for fat is approximately 0.5–1.0 g/kg body weight/day. Energy supply should comply with a mixture of glucose and lipids in the ratio of 60:40 or 50:50 and, energy intake should not exceed 35 kcal/kg body weight/day. Supplementation of water soluble vitamins are also recommended, especially in patients requiring any form of dialysis [2,4,5].

Route of administration

Enteral nutrition should be the primary type of nutritional support for patients with ARF, because even small amounts of nutrients can help to support intestinal functions. Unfortunately, it is impossible in many patients to meet all the requirements by the enteral route alone and parenteral nutrition (supplementary and/or temporarily) may become indispensable [5].

Conclusion

Nutritional support is of vital importance to maintain lean body mass, stimulate immune and repair functions, and decrease the mortality rate in the management of the patients with ARF. So, this support should thus be established as early as possible during the course of ARF, although some gastrointestinal side effects of enteral nutrition and some technical, metabolic, and infectious complications of parenteral nutrition can be observed.

References