PRACTICAL ASPECTS

Alkaline Diet and Metabolic Acidosis: Practical Approaches to the Nutritional Management of Chronic Kidney Disease

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The kidneys play an extremely important role in maintaining the body acid–base balance by excreting nonvolatile acids and regenerating and reabsorbing bicarbonate in the kidney tubules. As the individual loses their kidney function, renal excretion of nonvolatile acid produced by metabolism of the diet is impaired, resulting in low-grade metabolic acidosis. With this in mind, it is relevant to better understand the dietary aspects related to the acid–base balance in chronic kidney disease metabolic acidosis and try to provide possible strategies for the nutritional management of these cases. The type of diet can deeply affect the body by providing acid or base precursors. Generally speaking, foods such as meat, eggs, cheese, and grains increase the production of acid in the organism, whereas fruit and vegetables are alkalizing. On the other hand, milk is considered neutral as well as fats and sugars, which have a small effect on acid–base balance. The modern Western-type diet is deficient in fruits and vegetables and contains excessive animal products. Thus metabolic acidosis may be exacerbated by a contemporary Western diet, which delivers a high nonvolatile acid load. The remaining acid is neutralized or stored within the body. Bone and muscle are lost to neutralize the acid and serum bicarbonate falls. Early studies suggest that lowering the dietary acid load with a reduced protein content and vegetable proteins replacements, associated with an increase in fruits and vegetables intake can improve the metabolic parameters of acidosis, preserve bone and muscle, and slow the glomerular filtration rate decline. More studies focusing on the effects of controlled dietary interventions among chronic kidney disease patients are needed to determining the optimal target for nutritional therapy.

Introduction

CHRONIC KIDNEY DISEASE (CKD) is known for being widespread in the Western world. It causes considerable changes in the quality of life and survival of affected individuals while demanding considerable amounts of financial resources for its treatment. Thus, lifestyle adaptations are of utmost importance for the management and prevention of this condition.[1-3]

A common complication of CKD is the metabolic acidosis. The term acidosis refers to a process that causes a low pH in blood and tissues due to a higher concentration of hydrogen ions. The blood gas analysis is used to determine blood pH as well as serum bicarbonate levels (HCO_3), oxygen gases (O_2), and carbon dioxide (CO_2). Under normal conditions, the reference value for the pH is 7.40 ± 0.02, for the partial pressure of carbon dioxide (pCO_2) it is 38 ± 2 mm Hg, and for HCO_3, it is 24 ± 2 mmol/L.[4]

Metabolic acidosis is defined when there is also a decrease in the concentration of HCO_3 below 22 mEq/L in individuals with a reduction in their kidney function.[5] Therefore, when the pH and bicarbonate levels are below those recommended, it is possible to affirm that the metabolic acidosis component is present. If a pCO_2 reduction also appears, it is possible to infer that a respiratory compensation of metabolic disturbances has established in an attempt to balance the concentration of H^+ ions.[6]

The kidneys play an extremely important role in maintaining the acid–base balance in the body by excreting nonvolatile acids and regenerating and reabsorbing bicarbonate in the kidney tubules. As the individual loses their kidney function, reductions in the excretion of these acids and in bicarbonate regeneration, as well as a decrease in the bicarbonate reabsorption by the tubules, will occur.[7]

It is estimated that the metabolic acidosis is present in 2.3% to 13% of individuals in Stage 3 CKD and in 19% to 37% of individuals in Stage 4.[8,9] Low levels of bicarbonate bring several negative consequences to the patients, such as the increased risk for kidney disease progression, higher bone degradation by osteoclast activation, and osteoblast inhibition and an increase in mortality, inflammation, and malnutrition.[10-16]

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Because acidosis is the initial stimulus for protein degradation by the activation of the ubiquitin–proteasome system and the activation of caspase-3, conducting an initial cleavage of muscle proteins, metabolic acidosis presents a direct association with muscle degeneration.[17-19] Ballmer et al.[20] have shown that metabolic acidosis starts to promote negative consequences within just 48 hours causing a negative nitrogen balance and a decreasing albumin synthesis in humans. With this in mind, previous studies have tested the treatments to increase blood pH from acidotic levels to normal levels and they have shown a decrease in protein degradation.[21-23]

Because of the importance of better understanding the nutritional factors related to the development of metabolic acidosis, this article intends to discuss the dietary aspects related to the acid–base balance in metabolic acidosis of CKD. Moreover, it tries to provide possible strategies for the nutritional management in these cases.

Dietary Components and Metabolic Acidosis

In the nutritional treatment of CKD, some variables on diet need an adjusted and individualized control according to the stage of the disease, such as the quantity of energy, protein, sodium, phosphorus, and potassium.[3] Nevertheless, as for the management of the metabolic acidosis in CKD, there are additional elements to be taken into account. According to the literature, the type of diet can deeply affect the body by providing acid precursors, that is, noncarbonic acids (sulfuric acid), or base precursors, alkali salts from organic acids (OAs; citrate and bicarbonate).[24-26]

The first organ responsible for acidosis regulation is the intestine, which adjusts the excretion of the alkali pancreatic juice, depending on the amount of acid-inducing foods ingested. It is believed that the acid-inducing diets affect the kidney by tubular toxicity because of high concentrations of ammonia generated from the metabolism.[26] Therefore, the diet is directly associated with acidemia because of the absorption of acid and alkali derived from food.[27]

One of the nutritional management for patients with CKD in the predialysis stage is the modification of protein sources from 0.6 to 0.8 g of protein per kilogram ideal body weight (IBD).[28,29] However, only the protein reduction may not be effective in the control of acidosis, considering that other foods may also interfere in this metabolic disturbance.

In light of these observations, Remer and Manz[24] developed a model known as Potential Renal Acid Load (PRAL). To our knowledge, this method evaluates the net acid load produced by diets from nutrient intake data. For clinical applicability, the professional can calculate the nutrients in records, recalls, or food frequency questionnaires using nutrition software. Thus, it is possible to estimate the total acidity of the diets.[27]

Potential Renal Acid Load

This method has already been tested in controlled experiments and has been shown a good correlation between the diet and the acid–base balance.[30] This model takes into account the intake of protein, phosphorus, potassium, magnesium, and calcium, and it is based on the average intestinal absorption rate of the respective nutrients and in the urinary excretion of OAs, translated by the following formula:

$$\begin{aligned}\text{PRAL (mEq/d)} = {} & 0.49 \times \text{protein (g/d)} \\ & + 0.037 \times \text{phosphorus (mg/d)} \\ & - 0.021 \times \text{potassium (mg/d)} \\ & - 0.026 \times \text{magnesium (mg/d)} \\ & - 0.013 \times \text{calcium (mg/d)}\end{aligned}$$

When the value of PRAL for a type of food is inferior to 0, it is considered that this food increases the alkalinity of the body fluids and, when superior to 0, the food increases the production of acids in the body.[24] Generally speaking, foods such as meat, eggs, cheese, and grains increase the production of acid in the body, whereas fruits and vegetables (F&V) are alkalizing foods. On the other hand, milk is considered neutral as well as fats and sugars, which have a small effect on acid–base balance.[24,31,32]

These different effects on the acid–base balance occur because the metabolism of foods that contain protein metabolizes the amino acids and, therefore, releases hydrogen ions causing the changes in the blood pH. The quantity of released acid will depend on which amino acids are present because some are classified as neutral, others as acidic, and some as alkaline. Lysine, arginine, and histidine are considered acidic and, when metabolized, they generate hydrochloric acid, just as cysteine and methionine, which contain sulfur and are converted into sulfuric acid.[32]

Metabolism of potassium salts found in F&V, including citrate and malate, leads to the consumption of hydrogen ions and, consequently, to an alkalizing effect.[33] Considering this, the quantity of potassium, in general, reflects the alkalizing capacity of the food; therefore, potatoes and pumpkins have a higher alkalizing ability than apples and pears. However, some F&V contain oxalate in their composition, an OA that form the potassium oxalate, which cannot be metabolized, not providing any alkali or hydrogen ion to the body. Thus, the consumption of F&V with high oxalate levels is discouraged.[32]

It is important to point out that the quality of protein also needs to be evaluated to quantify the acidity of the diet. Animal proteins have an elevated amount of phosphorus and contribute to acidify body fluids. Except for milk which has the quantity of phosphorus compensated by the quantity of calcium. On the other hand, vegetable proteins have phosphorus in the form of phytate, which is less