Reducing the Dietary Acid Load: How a More Alkaline Diet Benefits Patients With Chronic Kidney Disease

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It has been proposed that a low-protein diet will slow progression of chronic kidney disease although studies have not always supported this belief. The accepted practice is that 60% to 70% of protein comes from high biological value (HBV) protein, but this limits patient choice and patients struggle to follow the diet. When a diet with only 30% HBV protein was trialed, there was a significant increase in serum bicarbonate, and patients preferred the diet. The dietary advice given in predialysis clinics was changed. HBV protein was restricted to approximately 50% of total protein, bread and cereal foods were allowed freely, and fruits and vegetables (F&V) were encouraged. Patients who followed the diet have seen a slowing of progression and occasionally regression of their renal function. Both observations and scientific literature indicate that this is because of a reduction in the acid content of the diet. When foods are metabolized, most proteins produce acid, and most F&V produce alkali. A typical 21st-century diet produces 50 to 100 mEq H^+ per day which the kidney is challenged to excrete. Acid is excreted with phosphate and is limited to about 45 mEq H^+ per day. With chronic kidney disease, this falls progressively to below 20 mEq H^+ per day. Historically, ammonium excretion was believed to be excretion of acid ($NH_3^+ + H^+ \rightarrow NH_4^+$), but it is now understood to be a by-product in the neutralization of acid by glutamine. The remaining acid is neutralized or stored within the body. Bone and muscle are lost in order to neutralize the acid. Acid also accumulates within cells, and serum bicarbonate falls. The author postulates that reducing the acid load through a low-protein diet with greater use of vegetable proteins and increased F&V intake will slow progression or occasionally improve renal function while maintaining the nutritional status of the individual.

Introduction

LOW-PROTEIN DIETS (LPDS) have been used in the management of chronic kidney disease (CKD) for more than 100 years. Originally these diets were prescribed to reduce uremic symptoms and improve well-being. Since the 1980s, these diets have been prescribed to slow progression of CKD.[1] The composition of these diets has been debated over the years with regard to both the quantity and type of protein consumed[2] and even whether they should be prescribed at all.[3] Since the late 1960s, the most widely used LPD has been the 40 g protein diet or 0.6 g protein per kilogram ideal body weight (IBW). It has been recommended that 60% to 70% of the protein should come from high biological value (HBV) proteins to ensure that the diet provides sufficient essential amino acids to meet protein requirements.[4] Studies using this diet did not show significant slowing of progression[5] and, therefore, many dietitians and medical practitioners may be reluctant to advocate their use.

The recommendation that 60% to 70% of the protein should come from HBV proteins means that most of the protein comes from animal foods and limits the consumption of bread and cereal (low biological value protein or LBV) foods resulting in low energy intake. Special low-protein foods and energy supplements are used to meet the recommended energy intakes (30-35 kcals/kg IBW/ 126-146 kJ/kg IBW). However, patients do not like these special foods, and most patients struggle to follow the diet.[6]

Study and observation of a newly defined LPD with more LBV protein foods has allowed improved dietary flexibility and compliance, but also highlighted the importance of dietary acid load in CKD.[7] This is very significant (Fig. 1). A reduced ability to excrete acid in CKD causes an ongoing accumulation of acid within the body with deleterious effects on muscle mass and bone disease. By reducing the acid load, through a LPD with greater use of LBV proteins and increased fruit and vegetable (F&V) intake, the author postulates that progression to end-stage renal disease can be slowed or improved, while maintaining the nutritional status of the individual. Patients also feel better and have improved appetite.

The approach used for this article has been based on personal observations of several hundred patients following an adapted LPD. These observations were not measured or quantified. They were carried out in a clinical setting

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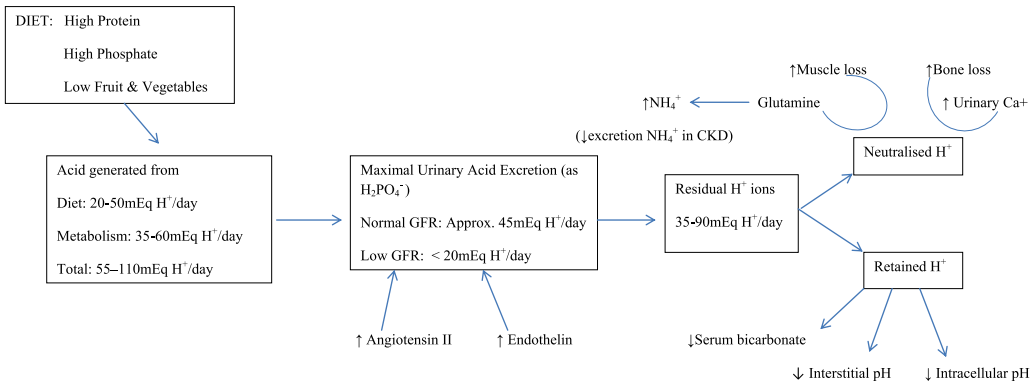

Figure 1. Consequences of a diet with a high dietary acid content in patients with CKD.

with patients who were showing subtle changes in health which were difficult to quantify. However, when these patients are seen over many years and personally known to the dietetic team, a confidence arose that there would be a scientific reason behind these observations. This led to the exploration of the alkaline diet.

A New LPD

A new LPD (0.6 g protein/kg IBW/day) was designed with only 30% protein from HBV proteins, and nitrogen balance studies were used to compare this diet with the conventional LPD (70% HBV) in a group of patients with CKD.[7] On the new LPD, essential amino acid requirements were met, and nitrogen balance was not significantly different although 5 of the 7 patients were in more positive nitrogen balance on the new LPD. Serum bicarbonate increased significantly (25.1-28.3 mmol/L, $P = 0.005$) with the new LPD but all other biochemical and hematological parameters were unchanged. Total energy intake was similar on both diets, but energy obtained from normal foods increased from 1396 kcals/day (5.84 MJ/day) to 1986 kcals/day (8.31 MJ/day; $P = 0.0035$) on the new LPD. In addition, patients preferred the flexibility of the new diet.

Encouraging Observations in the Predialysis Clinic

In 2008, after the positive response from patients to the new LPD, the diet was adapted for routine use in patients attending our predialysis clinics (glomerular filtration rate [GFR] < 20 mL/minute). HBV proteins are restricted to approximately 50% of protein intake (0.6-0.8 g protein/kg IBW), bread and cereal foods are allowed freely for energy, and F&V actively encouraged. It is observed that many patients in the predialysis clinics have low intakes of F&V; however, their consumption is essential to help reduce the acid load. Patients who followed the new diet reported improved appetite and well-being. Subtle improvements with regard to progression of CKD were noticed (personal observation), and more patients had stable (Fig. 2), or occasionally, improved renal function (Fig. 3). The positive effect on patients following the revised LPD has prompted the investigation of acid–base balance in greater detail and the crucial role played by diet.

Background

The acid and alkaline nature of food were recognized more than a century ago. Sherman et al. quantified the potential amounts of acid and alkali by measuring the alkaline ash content of a variety of foods.[8] They suggested that the acid and alkaline elements of the diet should balance each other. Early "nephrologists" (1920s and 1930s) recognized that kidney patients suffered from an excess of acidity. Alkaline diets were used successfully in the treatment of chronic nephritis and hypertension, and patients felt better.[9–11] However, this aspect of diet seems to have disappeared from mainstream medicine and nutrition, although continued to be advocated by the popular press.[12,13] Recently, following extensive studies in animal CKD models,[14–17] Wesson et al. developed the hypothesis that increasing F&V consumption reduces kidney damage and slows progression of CKD. This has been substantiated by them in a number of human studies.[18–20]

Acid and Alkaline Foods

When foods containing protein are metabolized, most proteins release acid (H^+/hydrogen ions) because of the metabolism of amino acids. The amount of acid depends on which amino acids are present: some amino acids are neutral, some acidic, and some alkaline. Lysine, arginine, and histidine are acidic, and when metabolized in the liver generate hydrochloric acid (plus glucose and urea). The amino acids, cysteine and methionine, contain sulfur and are converted to sulfuric acid:

$$\text{Arginine}^+ \rightarrow H^+ + Cl^- + \text{Glucose} + \text{Urea}$$

$$\text{Cysteine} \rightarrow 2H^+ + SO_4^- + \text{Glucose} + \text{Urea}$$

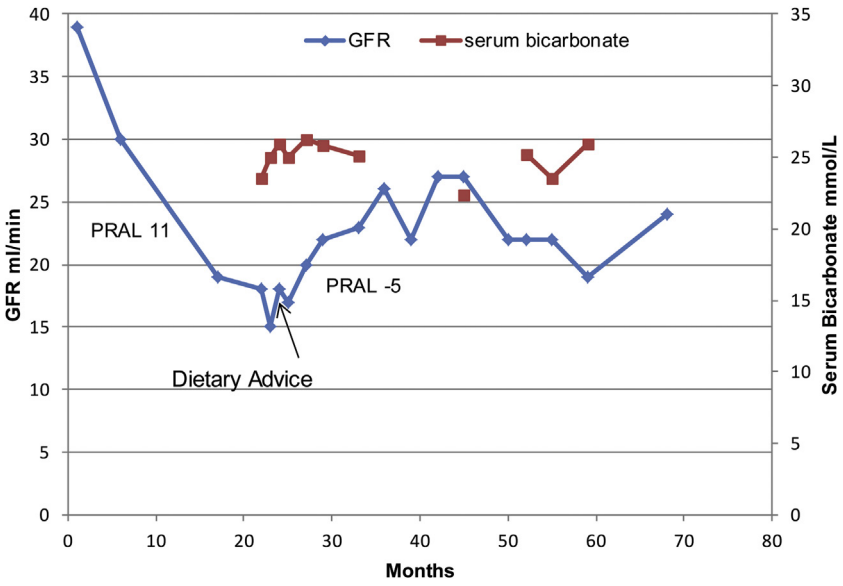

Figure 2. Patient 1 (female, 77 years with hypertension, Type II diabetes, previous stroke and myocardial infarction, osteoarthritis and single functioning kidney) attended predialysis clinic in December 2012 (glomerular filtration rate: 18 mL/minute). Her diet was high in protein (large portions with main and snack meal) with moderate portions of fruits and vegetables. Protein portions were reduced (urea fell from 27 to 19 mmol/L in 2 months) and vegetable portions increased including regular salads. The patient has remained well, although troubled by her arthritis, for almost 4 years (recent GFR 24 mL/minute) and is no longer seen in predialysis clinics.

Most F&V when metabolized produce alkali which neutralizes the acid. F&V contain organic acids such as citric acid and organic salts, for example, potassium citrate. The organic acids, when metabolized, produce equal amounts of hydrogen and base ions, but the organic salts contain base ions but no hydrogen and, therefore, "mop up" hydrogen ions on their metabolism to carbon dioxide and water. This reduces the acid load:

$$\text{Potassium citrate} \rightarrow \text{Citrate}^{3-} + 3\text{K}^{+}$$

$$\text{Citrate}^{3-} + 3\text{H}^{+} + 4.5\text{O}_2 \rightarrow 6\text{CO}_2 + 4\text{H}_2\text{O}$$

Foods which contain phosphate, whether naturally or from food additives, can add acid to the diet. The acidity depends not on the phosphate anion but on the cation to which it is attached and the pH of the food. For example, phosphoric acid (H_3PO_4) in cola drinks is acidic as H^+ is released, whereas the food additive trisodium phosphate (Na_3PO_4) is alkaline and will remove H^+.

$$\text{H}_3\text{PO}_4 \rightarrow \text{H}^{+} + \text{H}_2\text{PO}_4^{1-} \left(\text{excreted as } \text{H}_2\text{PO}_4^{1-} \text{ in the urine}\right)$$

$$\text{Na}_3\text{PO}_4 + 2\text{H}^{+} \rightarrow \text{NaH}_2\text{PO}_4 + 2\text{Na}^{+} \rightarrow \text{H}_2\text{PO}_4^{1-} (\text{in urine})$$

Fats and sugars, unless incompletely metabolized, have only a small effect on acid–base balance.

Net endogenous acid production (NEAP) can be estimated from dietary constituents together with an estimation of organic acids generated from diet and metabolism and excreted in the urine. A number of formulae exist for

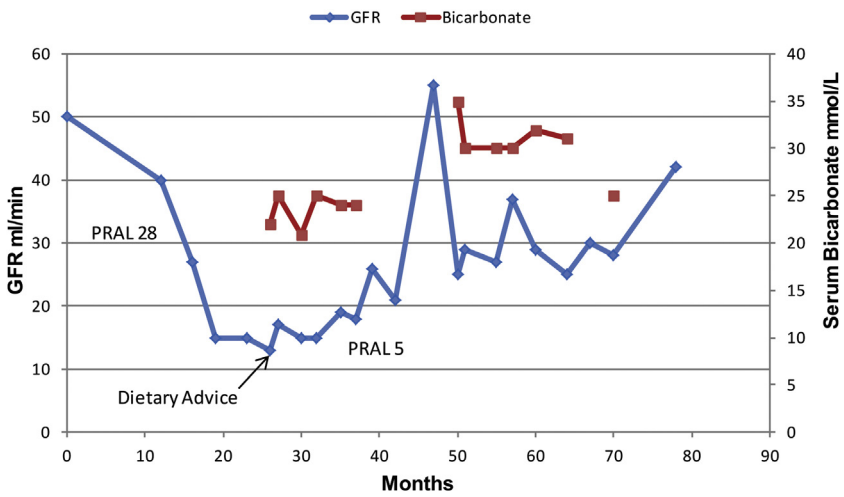

Figure 3. Patient 2 (male, 74 year old, with ischemic/hypertensive nephrosclerosis, severe cardiac dysfunction, and previous myocardial infarction) attended predialysis clinic in June 2009 (glomerular filtration rate [GFR]: 13 mL/minute). His diet was high in protein with some fruit but few vegetables and no potatoes. He was advised on a low-protein diet with added vegetables but no other changes in treatment were made. Protein intake reduced (urea fell from 36 to 14 mmol/L over the first year) and GFR began to improve. Improvements in cardiac function were also noted and he remained relatively well, until dying from a chest infection 4 years 6 months after his first clinic visit (GFR: 42 mL/minute).

Table 1. Estimation of Net Endogenous Acid Production (NEAP)

Acid production can be estimated from diet or measured in the urine: Estimated NEAP = NAE (all mEq/d)

Diet: Several formulae have been proposed to estimate NEAP. Two of these are as follows:

Frassetto[21]: NEAP (mEq/d) = (54.5 × protein [g/d]/potassium [mEq/d]) − 10.2

Remer[22,23]: NEAP (mEq/d) = PRAL + OA_{est}

where PRAL (mEq/day) = 0.49 × protein (g/d) + 0.037 × phosphorus (mg/d) – 0.021 × potassium (mg/d) − 0.026 × magnesium (mg/d) − 0.013 × calcium (mg/d)

$$OA_{est}(mEq/d) = \frac{BSA\ (m^2) \times 41\ (mEq/d/1.73m^2)}{1.73\ (m^2)}$$ or OA_{est} (mEq/d) = BW (kg) × 0.66

Urine: NAE (mEq/d) = NH_4^+ + TA − HCO_3^-

BSA, body surface area; BW, body weight; HCO_3^-, bicarbonate; NAE, net acid excretion; NEAP, net endogenous acid production; NH_4^+, ammonium; OA_{est}, estimate of organic acid production; PRAL, potential renal acid load; TA, titratable acid.

this (Table 1).[21–23] In Remer's formula, the potential renal acid load (PRAL) of foods is estimated and this is a useful tool to compare foods (Table 2). A typical 21st-century diet releases 50 to 100 mEq H^+ per day (PRAL of diet [−20 (vegan) to + 50 (diet rich in animal protein/cereal foods and low F&V) plus 35-60mEq H^+ from endogenous metabolism]).

The Role of the Kidney in Maintaining Acid–Base Balance

The kidney helps maintain acid-base balance by 3 main mechanisms: (1) excretion of acid, (2) neutralization of acid and (3) excretion of anions.

Excretion of Acid

Dietary acid has to be excreted by the kidney. Phosphate is the primary buffer in the urine and accepts H^+ ions ($HPO_4^{2-} + H^+ \rightarrow H_2PO_4^{1-}$). The quantity of phosphate excreted is mainly dependent on dietary phosphate and usually varies between 10 and 45 mmol/day.[24] About 80% of the phosphate filtered at the glomerulus is in the monohydrate form (HPO_4^{2-}) and will remove H^+ ions. The remaining phosphate is already present in the diprotic form ($H_2PO_4^{1-}$) and therefore unable to remove additional hydrogen. When the pH of the urine falls, creatinine, urate, and other anions filtered at the glomerulus act as urinary buffers, removing additional hydrogen ions. Acid excreted with phosphate and other urinary buffers is known as titratable acid (TA).

Neutralization of Acid

Historically, it was believed that ammonia was also a urinary buffer accepting H^+ ($NH_3 + H^+ \rightarrow NH_4^+$) and thus increasing acid excretion. However, it is now recognized that the remaining acid is not excreted but only neutralized within the kidney and ammonium is a by-product of this.[25] Neutralization of acid occurs through the metabolism of glutamine. In the proximal tubule of the kidney, glutamine is taken from the blood stream and broken down to alpha-ketoglutarate (AKG^{2-}) and ammonium. AKG^{2-} consumes 2 H^+ on its metabolism to glucose, thus reducing the acid

Table 2. The Potential Renal Acid Load (PRAL) of Food (mEq/3.5 oz [100 g] and per Average Portion Size)[22]

Food Type	PRAL mEq per 3.5 oz (100 g) Edible Portion	PRAL mEq per Average Portion Size (oz/g of Portion)
Hard cheese, for example, cheddar	20	10 (2 oz/50 g)
Camembert and similar "soft" cheeses	15	7.5 (2 oz/50 g)
Meat–all types	8	8 (3.5 oz/100 g)
Fish–all types	8	8-12 (3.5-5 oz/100-150 g)
Pasta–raw, white	8	6 (2.5 oz/75 g raw weight)
White bread	6	2.5 (1 large slice)
White rice–raw	4.5	3.3 (2.5 oz/75 g raw weight)
Eggs	4	2 (1 egg)
Biscuits	3	1 (3 biscuits)
Peas, beans, lentils	1-3.5	0.8-2.8 (3 oz/80 g portion)
Milk	0.7	1.2 (6 oz/180 mL)
Red wine	−2.4	−4.2 (6 fl oz/175 mL glass)
Vegetables	−2.8	−2.1 (3 oz/80 g portion)
Fruits	−3	−2.4 (3 oz/80 g portion)
Potatoes	−4	−6 (3 egg size)

PRAL (mEq/d) = 0.49 × protein (g/d) + 0.037 × phosphorus (mg/d) − 0.021 × potassium (mg/d) − 0.026 × magnesium (mg/d) − 0.013 × calcium (mg/d).

load and the ammonium is excreted into the lumen of the nephron:

$$\text{Glutamine} \rightarrow \text{Glutamate} \rightarrow AKG^{2-} + 2NH_4^+$$

$$AKG^{2-} + 2H^+ \rightarrow \text{Glucose (or } CO_2 + H_2O)$$

This ammonium is recycled in the loop of Henle via a medullary shunt, a mechanism that allows fine tuning of acid excretion[24] and may allow additional distal [H^+] to be excreted without lowering the pH of the urine.[26] When the diet is very acidic, there are small increases in TA, limited by the amount of phosphate in the diet, but significant increases in ammonium excretion which can increase 10-fold.[27] This gives flexibility to the body allowing it to neutralize large acid loads. However, ammonium excretion takes several days to maximize as glutamine uptake is stimulated in the proximal tubule together with increased production of the enzymes converting glutamine to AKG.[28]

Excretion of Anions

Some organic anions like citrate, oxalate, and urate are excreted in the urine, and this is a loss of alkali to the body as some of these anions, for example, citrate could have been metabolized to alkaline end products. Others, like urate and oxalate, are end products of metabolism. The amount of organic anion excreted is estimated for body surface area and is part of NEAP calculations (Table 2). Organic anion excretion is considered to be fairly constant, although recent studies suggest significant increases of organic anions occur with increased intakes of protein and certain F&V.[29] When the urine is very acidic, some of these anions will be excreted in the urine with H^+ as organic acids and will be measured as part of TA excretion.

The total amount of acid excreted and neutralized by the kidney can be measured directly from 24-hour urine samples and is known as net acid excretion (NAE).

$$\text{NAE(mEq/d)} = NH_4^+ + TA - HCO_3^-$$

When eating an acidic diet, the amount of bicarbonate excreted is negligible to conserve alkali.

How Diet Affects Excretion and Neutralization of Acid in Young Healthy Adults

The composition of the diet directly affects TA and ammonium excretion to maintain acid–base balance. Schuette has studied how changes in protein and phosphate affect urinary acid excretion under metabolic conditions (Fig. 4).[30] A constant diet with 745 g/day of F&V and protein from mixed protein sources were provided. When protein intake was trebled (mainly animal protein), there were small increases in TA excretion but significant increases in ammonium excretion. When phosphate intake was increased using an acidic form of phosphate, arginine phosphate, TA excretion trebled with small increases in ammonium excretion. Increases of similar magnitude were seen when protein and phosphate intakes were increased simultaneously. Other studies have shown that if phosphate intake is increased using a neutral form of phosphate, a source that does not contribute to the acid load, then TA increases significantly but ammonium excretion is halved.[31] If soya protein replaces meat protein, ammonium excretion falls significantly (no data on TA).[32] When large doses of sodium bicarbonate (150 mEq/day) are given, this neutralizes dietary acid resulting in minimal ammonium and TA excretion and increased bicarbonate excretion.[33]

How CKD Affects Excretion and Neutralization of Acid

As GFR falls, the ability of the kidney to excrete and neutralize acid reduces significantly and progressively (Fig. 5).[34] After an acid load (ammonium chloride), TA is about 60% of that found in normal subjects.[35] This is reflected in the modest levels of TA (12-19 mEq/day) reported in patients with CKD stage 3 and 4, on a free diet.[34,36] Further reductions in TA are said to occur in CKD stage 5.

In CKD, ammonium excretion is significantly impaired.[35] Vallet has shown that patients with CKD stage 4 on a free diet had an ammonium excretion of 18.0 mEq/day (see Fig. 5).[34] This is significantly less ammonium than Schuette recorded with healthy adults on either a 50 g protein per day (24 mEq/day) or 150 g protein per day (68 mEq/day) in a diet which contained a significant amount of F&V (9 × 3 oz/80 g portions). Higher ammonium excretion would be needed when F&V intake is lower. When large doses of sodium bicarbonate (120 mEq/day) are given to patients with CKD, both TA and ammonium excretion fall to almost 0, as shown in normal patients.[37]

The reduction in TA and ammonium excretion seen in CKD may be offset, to some extent, by a reduction in organic anion excretion (alkali), as CKD develops but this has not been quantified.[38]

How Excess Dietary Acid Damages the Kidney

Recent studies show that higher NEAP is associated with faster decline in CKD,[39,40] and that when dietary acid is neutralized with alkali or increased consumption of F&V, this reduces markers of kidney injury and progression of disease.[19,41,42] Kidney damage and progression of CKD may be due to increased angiotensin II and endothelin,[14,15,17] both required for the excretion of acid.[43,44] Increased concentrations of renin-angiotensin-

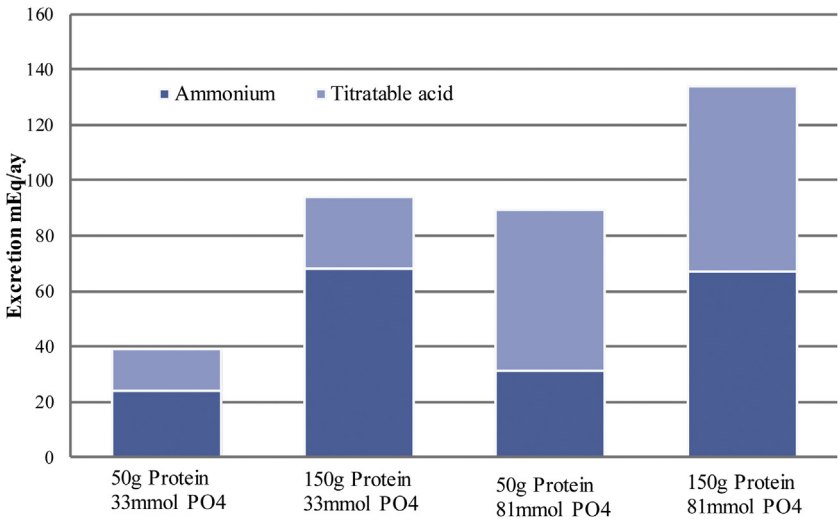

Figure 4. Titratable acid and ammonium excretion in healthy adults on diets of varying protein and phosphorus content. Reprinted with permission from Schuette et al.[30]

aldosterone system are linked to pathology seen with CKD: increased GFR, renal hypertrophy, proteinuria, tubulointerstitial damage, glomerulosclerosis, inflammation (interleukin 6), and reactive oxygen species.[43,45] High cortical ammonia concentrations may also cause tubular toxicity and kidney damage.[46] A more vegetable-based diet also alters the gut flora, and this may result in increased production of short chain fatty acids (alkali)[47] and in the production of less nephrotoxic substances.[48] Slowing of progression may also be through the lowering of blood pressure.[18-20]

Evidence for Accumulation of Acid Within the Body

As CKD progresses, there is a slow but progressive retention of acid unless dietary intake is significantly altered. Goodman found that CKD patients on diets of 0.6 to 1.0 g protein per kilogram retained 19 mEq H^+ per day.[37] In other studies, CKD patients, on an unrestricted diet, retained 12 to 14 mEq H^+ per day.[36,49] Wesson[50] found that acid retention occurs as early as CKD stage 2 in his patients, but these patients were eating very acidic diets (PRAL 62); so this may not be replicated in all patients. Vallet estimates that patients with CKD stages 1 and 2 were able to excrete all the acid in their diets but were retaining acid in CKD stage 4.[34] Patients with normal renal function will retain acid when given a large acid load.[51]

How Does the Body Maintain pH

To maintain health, the pH of the extracellular fluid must be maintained within tight limits (pH: 7.35-7.45). Neutralization, buffering, and storage of acid within the body occur to achieve this.

As serum bicarbonate concentrations fall, muscle protein degrades to neutralize the dietary acid.[52] The rate of catabolism is inversely proportional to the degree of acidosis.[53]

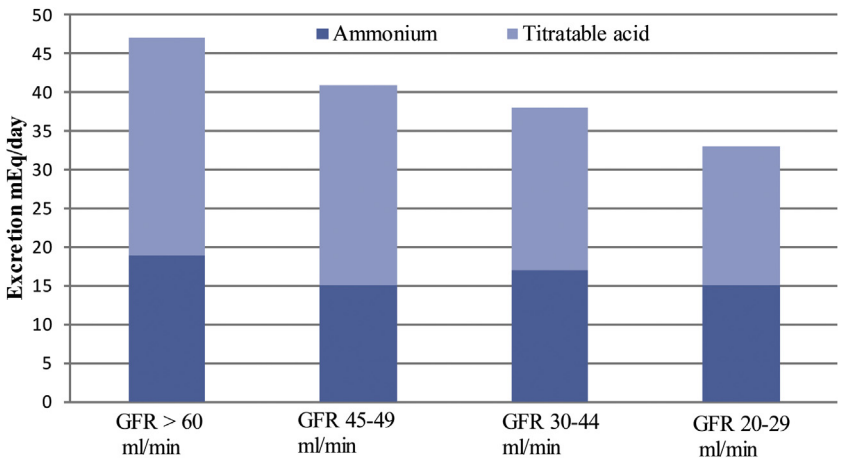

Figure 5. Titratable acid and ammonium excretion in patients with chronic kidney disease on an unrestricted diet, according to measured glomerular filtration rate. Reprinted with permission from Wrong et al.[34]

The amino acids released are used for the hepatic synthesis of glutamine. Muscle wasting is evident in 30% to 50% of patients with CKD and increases as GFR falls.[54]

Acid is also buffered on the reactive surfaces of bone and connective tissues.[33,55] Sodium, potassium, and calcium are released from the bone surface and bone resorption occurs, releasing calcium, bicarbonate, and phosphate in exchange for hydrogen. This contributes to the mineral and bone disorders seen in CKD patients.

Acid is buffered and stored throughout the body. Studies suggest that about 50% of the retained acid is buffered intracellularly with an additional 30% interstitially; but this probably varies with the circumstances.[56] Within blood, acid is buffered with bicarbonate, hemoglobin, plasma proteins, and phosphate. Most patients with GFR <20 mL/minute have low serum bicarbonate (12-20 mmol/L).[57] However, serum bicarbonate often remains stable despite continuous acid loading because of all the additional buffering and storage within the body.

Acid is buffered within cells. The acid diffuses passively across the cell membrane into the intracellular fluid (pH: 6.9-7.2). Low intracellular pH is associated with increased sodium hydrogen exchanger activity (NHE1) and cellular dysfunction.[58] Acid also enters the interstitial space (part of the extracellular space), which has little buffering capacity. In the interstitial space, hormones and neurotransmitters regulate cell function. Low interstitial pH contributes to insulin resistance,[59] a problem evident in CKD patients as they approach dialysis.

Putting it Into Practice

The dietary advice given to patients in predialysis clinic is summarized in Table 3.[60,61] There are additional elements to be taken into account.

Not All F&V are Alkaline

The predominate anions in F&V are citrate and malate, and when metabolized, they release bicarbonate and thus contribute alkali to the body. In general, the amount of potassium present reflects the alkalizing ability of the F&V; thus, potatoes and squash have high alkalizing ability, and apples and pears have less alkalizing ability. However, F&V contain a wide variety of organic anions and not all those present will contribute alkali. For example, oxalate and tartrate anions cannot be metabolized. When these anions are present in large proportions in a food, for example, spinach contains more than 80% oxalate, then the potassium oxalate cannot be metabolized so does not provide any alkali and any oxalic acid present in the food will release H^+ and thus add acid to the body. Some fruits (e.g., cranberries, prunes, plums, and some berries) contain benzoic and quinic acid which are metabolized by gut bacteria to hippuric acid and also increase acid excretion.[62] However, the organic acid content of different F&V is not well documented and varies considerably with the variety, growing and storage conditions, and ripeness of the F&V. The absorption of anions may also be affected by the cooking methods used and other dietary constituents. Until there is a better understanding of this only F&V with high oxalate content has been discouraged.

Salt Increases Acidosis

Patients are advised to follow a "no added salt" diet. Studies have shown that dietary salt independently increases acid load and lowers serum bicarbonate, and this can account for between 50% and 100% of the acid load of the diet.[63] Patients in predialysis clinics have often reduced their intake of "added salt" but may continue to include many processed foods.

Acid Inducing Food and Drink

Carbonated drinks, including fizzy water, contain carbonic acid (pH: 2-5) and those containing phosphoric acid (H_3P0_4), that is, Cola have some of the highest levels of acidity. Patients are advised to stop these and switch to tap water (pH: 7.4). Phosphate-based additives are used widely, and some of these are acidic; for example, calcium acid pyrophosphate ($CaH_2P_2O_7$) used in meat and potato products. Including more fresh and natural foods in the diet should help reduce these.

Consideration of Potassium Intake

Advocating a more alkaline diet to patients with advanced CKD is challenging. The alkaline elements of the diet come with potassium; so the quantity advocated has to be carefully considered for each patient, taking into consideration prescribed medication, serum potassium, current dietary intake, and how the dietary changes advocated will affect total potassium intake. For patients on Angiotensin-converting enzyme inhibitors, Angiotensin receptor blockers or potassium sparing diuretics, often presenting with a raised serum potassium, there is more limited ability to alkalize the diet. Patients are given the usual dietary advice to avoid high-potassium foods and to boil vegetables to reduce potassium content although this also reduces the alkaline elements.[64] However, patients are still encouraged to include the beneficial vegetables and fruit within their dietary restrictions. Hyperkalemia has been seen in a few patients, most often as a result of ongoing reduction in renal function rather than as a direct consequence of the dietary advice.

Potential Benefits of a More Alkaline Diet

The LPD, with increased F&V, lowers NEAP and reduces the amount of acid to be managed by the kidneys. This lowers the stimulation of Angiotensin II, aldosterone, and endothelin, which are required to excrete dietary acid and reduces the accumulation of ammonia within the kidney. These measures reduce kidney damage. Regression of CKD may then be possible, as observed in Patient 2 (Fig. 3). Improvement in renal function in response to diet has been

Table 3. Outline of Dietary Recommendations Given to Patients in Predialysis Clinics to Lower Dietary Acid Load

Food and Drinks	Advice	PRAL Value (Approximate)
Potatoes, sweet potatoes, butternut squash (4 g protein)	Have 1 portion (5-7 oz/150-200 g) a day (unless raised serum potassium)	−6 to −8.0
Green leafy vegetables, for example, kale, broccoli, Brussel sprouts, cabbage. Onions, garlic, celery, zucchini, rutabaga. Salad vegetables, for example, lettuce, cucumber, radish, bell pepper, arugula. Sprouted seeds.	Have 2 to 3 portions (3 oz/80 g) at 2 meals a day (advice individualized according to serum K concentration)	−9 to −13.5
Spinach, sorrel, chard, beetroot	Avoid due to oxalate content	
Peas, sweet corn	Avoid as acidic due to protein content	
Fruit	Have 2 to 4 portions (3 oz/80 g) a day (advice individualized according to serum K concentration)	−5 to −10
Rhubarb	Avoid due to oxalate content	
1/2 pint milk (280 mL)/yogurt daily (9 g protein)	Less acidic than other animal proteins because of citrate in milk and lactate in fermented products.	2.8
2-4 oz (50-100 g) meat or fish (12-24 g protein)	Or equivalent protein portion as egg. Discourage cheese because of high PRAL.	4-8
Lentils, beans, or chick peas. Almonds or hazelnuts	Encouraged to include as alternative to some animal protein meals as lower PRAL (↓ quantities of acidic amino acids) and less damaging.[60,61]	
Bread, breakfast cereals, rice, pasta, biscuits, cakes, pastry (10-20 g protein)	Wholemeal/wholegrain foods are encouraged (lower PRAL) but limited quantities advocated when patient trying to lose weight. All foods acidic but provide essential energy for patients with advanced CKD and are generally low in potassium.	6-15
Fats and oils	Mono and polyunsaturated oils encouraged. Allowed freely	
Sugar and preserves	Allowed freely (unless diabetic or trying to lose weight)	
Fizzy drinks including diet drinks and carbonated water	Avoid. Acidic because of carbonic acid ± phosphoric acid	
Salt	No added salt (as inhibits acid excretion)	
Total PRAL		+5 to −15
Total protein (g)/d	35-57 g	

CKD, chronic kidney disease; PRAL, potential renal acid load.

observed previously and attributed to reduced secretion of Angiotensin II.[65] A more alkaline diet will also reduce the accumulation of acid and reduce the metabolic complications associated with CKD such as muscle wasting, bone disease, and insulin resistance.

Patients feel better. Uremic symptoms are reduced with a conventional LPD[66] but would appear to be further reduced with a more alkaline diet. Patients treated by Lyon in the 1930s felt significantly better on an alkaline diet than an acidic diet, although both diets contained the same amount of protein (1.0 g protein per kilogram).[11]

Conclusion

Studies using conventional LPDs to slow progression of CKD have not been as affective as expected. A revised LPD was trialed with less animal protein and more F&V. This resulted in some patients showing stable renal function, and a few patients have improved renal function. Patients felt better. Further research went into trying to explain this. This took the form of an exploration of acid base. The investigation revealed that in healthy kidneys, there is a limited ability to excrete dietary acid and excess acid is neutralized in the kidneys. But in CKD, both the ability to excrete acid and ammonia, a by-product of the neutralization of acid is significantly impaired. The mechanisms associated with this contribute to the ongoing damage to the kidney. Some acid is retained in the body with deleterious effect. Therefore, the balance between the acid and alkaline foods in our diet is absolutely crucial. This aspect of diet is not part of mainstream dietetic or nutritional teaching. Reducing the acid load of the diet offers an exciting new approach to treating patients with CKD.

Practical Application

An LPD with increased use of vegetable protein and more fruit and vegetables lowers dietary acid intake and reduces the amount of acid to be managed by the kidneys. This reduces kidney damage and may slow progression of CKD. This may also help preserve muscle mass and bone structure. Patients feel better.

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