Perspective
Volume 11:12, 2021

Journal of Blood & Lymph

ISSN: 2165-7831 Open Access

A Brief Note on Nutritional Management in Kidney Disease

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About the Study

Plasma potassium is filtered freely at the glomerulus before being reabsorbed in the proximal nephron and loop of Henle in large amounts (90 percent to 95 percent). Potassium is primarily excreted through distal tubular secretion. Potassium secretion in the renal tubules is an energy-intensive process. Renal dysfunction disrupts tubular function, resulting in potassium retention in the kidneys. This is in contrast to renal sodium management, where kidney injury usually results in renal sodium waste due to poor sodium reabsorption. Potassium can be reabsorbed or excreted in the distal collecting duct. The principal influence on renal potassium management is aldosterone. Aldosterone increases potassium excretion *via* increasing the number of sodium and potassium channels and Na-K-ATPase pumps. Tubular injury that blunts the aldo sterone response,i.e. renal tubular acidosis type IV, on the other hand will decrease renal potassium excretion.

Medications that limit the renin-angiotensin-aldosterone system's production or function, such as angiotensin converting enzyme inhibitors, angiotensin receptor blockers, or beta blockers, reduce renal potassium excretion through their influence on aldosterone and can cause hyperkalemia. In patients with CKD, ACEI and ARBs can be dangerous. Spironolactone and eplerenone produce hyperkalemia by blocking aldosterone's activity at its receptor in a competitive manner. Aldosterone secretion can be inhibited by chronic heparin medication. NSAIDs can potentially cause hyperkalemia by reducing kaliuresis, as a result of reduced glomerular filtration.

Renal potassium excretion is connected to salt absorption in the distal nephron. As a result, boosting sodium supply to the distal tubule increases urine potassium excretion. Hypokalemia is commonly caused by circumstances such as a high-salt diet, thiazide or loop diuretic medication, Bartter's syndrome, and other acute or chronic salt-wasting renal disorders. Hyperkalemia is caused due to low-salt diet or diseases that reduce sodium distal transport, such as congestive heart failure. Positively charged ions, such as trimethoprim and amiloride, produce hyperkalemia by obstructing potassium excretion in the distal nephron by blocking luminal sodium channels.

As GFR decreases, renal potassium excretion is maintained by increasing potassium fractional excretion. Hyperkalemia is uncommon at GFRs above 15 mL/min (serum creatinine 3.0 mg/dL), unless aldosterone secretion or function is compromised. Extrarenal potassium processing, particularly increased gastrointestinal excretion becomes more significant in dissipating an acute potassium load when GFR falls below 15 mL/min.

Many factors, in addition to nutrition, play a role in evaluating an increased serum potassium level in a patient with renal impairment. Fasting state, insulin level, acid-base status, -blocker therapy, serum PTH level, and heparin therapy can all impact plasma potassium levels, as previously stated. Because transfused blood can contain between 5 and 50 mEq/L of plasma potassium, depending on the storage duration of the blood, it can be an essential supply of potassium.

If the venipuncture technique is incorrect or if abnormally increased quantities of erythrocytes, platelets, or leukocytes are present, pseudo hyperkalemia might occur. When blood is allowed to clot before serum is obtained, these cells release intracellular potassium. These causes of pseudo hyperkalemia can be avoided by collecting blood in heparin to get plasma and collecting blood using a syringe rather than a vacuum tube.

How to cite this article: Romanoff, Mary ."A Brief Note on Nutritional Management in Kidney Disease ." *J Blood Lymph* 11 (2022) : 274.

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Received: December 21, 2021; **Accepted**: January 04, 2022; **Published**: January 11, 2022