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Association of Cardiovascular Complications in Potassium Disorders with Cardiovascular Disease, Diabetes Mellitus and Chronic Kidney Disease

Siddhartha Sawadekar, Someshwar Moholkar
Department of Clinical Pharmacy Practices, R. C. Patel Institute of Pharmaceutical Education and Research, Shirpur, Dist. Dhule, Maharashtra, India

ABSTRACT

Potassium disorders are common in patients with cardiovascular disease, chronic kidney disease, and diabetes mellitus. Hypokalemia and hyperkalemia are important electrolyte abnormalities that have generally been defined based on serum potassium concentrations develop with impaired renal function, and individuals with diabetes mellitus and chronic kidney disease are at greatest risk, as both may contribute to the development of the serious cardiovascular disease. Potassium concentrations have been associated with a higher risk of atrial fibrillation, ventricular fibrillation, hypertension and risk for arrhythmia. Hypokalemia is defined based on serum potassium concentrations <3.5 mEq/L is a risk factor of atrial fibrillation, while ventricular fibrillation, and sudden cardiac death. Hyperkalemia is defined based on serum potassium concentrations >6.0 mEq/L is the show effect of bradycardia, asystole. Potassium is important for the primary ion mediating cardiac repolarization in the hypokalemic condition they produce the effects of myocardial refractory periods and increases the risk for arrhythmia condition. And hyperkalemia causes slowed conduction and sometimes block conduction effect on the progressive result in asystole. The kidney maintaining the body potassium. In the case of chronic kidney disease, the capacity to maintain potassium level in the body is facing the alterations and the result is a problematic condition for transcellular electrolyte shifts. This condition increases the risk for hyperkalemia or hypokalemia and associated problems. Potassium abnormalities are one of the important reasons for electrolyte imbalances seen in DKA and risk factors for fatal arrhythmia. This condition increases the risk for hyperkalemia or hypokalemia and associated problems. We are studying the cardiovascular risk association between a serum potassium level and cardiovascular disease, chronic kidney disease, diabetes mellitus conditions.

Keywords : Cardiovascular Complications, Potassium Disorders, Cardiovascular Disease, Diabetes Mellitus, Chronic Kidney Disease

I. INTRODUCTION

Potassium is the most common intracellular cation and plays a major role in nerve and muscle cell function. Potassium disorders or dyskalemia conditions are relatively common in clinical practice. Long-term regulation of potassium homeostasis is mainly managed by renal excretion of potassium due to changes in dietary intake [1,2].In these disorders, conditions such as chronic kidney disease and diabetes mellitus may interfere with the capacity of the kidneys to balance potassium intake with potassium excretion and lead to dyskalemia. Hypokalemia is usually caused by hypertension. Undiscovered hypokalemia is the major cause of iatrogenic morbidity and mortality in cardiac patients

at associated risk of arrhythmia [3]. Potassium concentrations < 3.5 mEq / L have already been associated with greater risk of atrial fibrillation, whereas Q-T prolongation, the hazard of torsade des pointes, ventricular fibrillation and sudden cardiac death can happen with potassium values < 3.0 mEq / L [4,5,8]. While hypokalemia has usually been characterized based on serum potassium concentrations < 3.5 mEq / L, evidence indicates that slight decreases in serum potassium (3.5–<4.0 mEq / L) may be correlated with excess mortality in heart failure and/or chronic kidney disease patients [5,6,9]. Hyperkalemia usually creates with damaged renal function, and individuals with Hearth failure or DM and chronic kidney disease are more at risk. [7] Potassium levels > 6.0 mEq / L have been related with peaked T waves, large QRS complexes, bradycardia, asystole, and sudden death.[5] Serum potassium was analyzed and the distribution of serum potassium and prevalence of cardiovascular disease all-cause risk in the study with cardiovascular disease, chronic kidney disease, diabetes mellitus, and all 3 conditions.

II. The Role of Potassium In The Heart Function

Rhythmic contractions of the heart are regulated by periodic changes in the membrane potential of cardiac myocytes, called action potentials. The cardiac action potential consists of five phases Phase 0, which only lasts a few milliseconds, is a phase of rapid depolarization. Short and small repolarization (phase 1) is followed by a long plateau at a depolarized level (phase 2). The repolarization of the potential of the plateau is called phase 3. The final phase 4 continues with the next rapid depolarization. The action potential is the outcome of a coordinated action of inward (depolarizing) and outward (hyperpolarizing) ionic currents. The outward component is transported by K+ ions by potassium-permeable transmembrane proteins, the potassium channels. Regulation of the essential mechanical properties of the myocardium, such as rate of contraction, the interval between excitation of atria and ventricles, which defines the gap between contractions of these chambers and force of contraction by K+ ions, by working on three different tissues. In the sinoatrial node (SA node), potassium current regulates the rhythm of the heart related to its role in diastolic depolarization, a phase between two action potentials.[2,10,11,17]

In the atrioventricular node (AV node), potassium permeability determines the time necessary for depolarization to reach the threshold needed to induce an action potential that is then conducted to the ventricle. In ventricular myocardium, contractility depending on the length of the action potential which is also regulated by potassium channels. Calcium-permeable channels are opened during the plateau phase of the action potential.Ca2 + ions moving through such channels into the cytosol specifically activate the contractile cardiomyocyte system, enter the sarcoplasmic reticulum through its calcium pump (refilling effect) or trigger the release of additional Ca2 + ions from this intracellular Ca2 + store.[12]

Therefore, in general, any activity that contributes to a reduction in the plateau phase as a result of increased K+ permeability of sarcolemma produces a decrease in contractility. Potassium currents also monitor the level of the action potential plateau. A positive shift of the plateau voltage up to a potential of at least +20 m V due to a reduction of potassium conductivity enhances the contraction force. This is partially due to an increased inflow of calcium into L-type calcium channels. Another component that determines the Ca2 + balance is the sodium-calcium exchanger, which replaces one Ca2 + ion for three Na+ ions across the sarcolemma. As movement is electrogenic, depolarization stimulates the influx of Ca2 + ions or affects the influx of Ca2 + ions and thus

improves the net inward transmission of Ca2 + ions.[13,14,15,16]

III. Disorders of Potassium Homeostasis Hypokalemia

Hypokalemia is categorized as a plasma concentration of K+ below 3.5 mEq / L. Hypokalemia characteristics impact on muscle resting membrane potential (MPR) In muscles, low plasma K+ leads to hyperpolarized myocyte, which tends to be more highly resistant to inhibition. It induces fatigue myalgia and weakening, particularly noticeable in the broad proximal skeletal muscles of the hip and thigh. [12,18] Increasing hypokalemia induces respiratory muscle weakness and, finally, systemic muscle dysfunction, like paralytic illus. In cardiac myocytes, potassium ion activity is directly related to plasma K+ concentration.[19] Hypokalemia, therefore, decreases K+ conduct and causes a continuous repolarization phase of the potential for cardiac action. Hypokalemia results from abnormal losses, transcellular shifts or inadequate intake. [22-22] Abnormal losses are most common after the kidney can effectively reduce potassium excretion in response to decreased intake, inadequate intake is usually the causative agent of hypokalemia but often contributes to hypokalemia in patients. [21]

Hyperkalemia

Hyperkalemia is defined as a plasma K+ concentration greater than 5 mEq/L. [18,23] as for hypokalemia, the manifestations of hyperkalemia also relate to neuromuscular and acid-base effects. Increased extracellular fluid K+ concentration forces K+ into the cells through the always-open leaky potassium channels, leading to a slight depolarization. A constant state of depolarization effects excitability and, once again, the effect is fatigue and weakness. If severe, respiratory muscle weakness can be a life-threatening complication. In cardiac myocytes, hyperkalemia causes increased K+ conductance. [13,18] Since the K+ current is responsible for repolarization, the first manifestation of hyperkalemia is rapid repolarization, reflected on the electrocardiogram as a sharp, peaked T wave. Increased K+ conductance also makes the resting membrane potential more negative, to a level at which Na+ channels start getting inactivated.[24] Hyperkalemia can also induce an atrioventricular nodal block reflected as a prolonged PR interval and a ventricular rhythm with wide QRS complexes.[24,25] P wave amplitude decreases, and may not be detectable, due to an 'electrical paralysis' of the atria.[26] With increasing severity of hyperkalemia, the widened QRS tends to merge with the peaked T wave, producing a characteristic sine wave pattern.[27] Eventually, the cardiac arrhythmia deteriorates into ventricular fibrillation or asystole, leading to death. Hyperkalemia is caused by excess potassium intake, impaired potassium excretion, or transcellular shifts.[28] The etiology of hyperkalemia is often multifactorial, with impaired renal function, medication use, and hyperglycemia as the most common contributors.[15,29]

IV. Dyskalemia Related To Cardiovascular Complications

This membrane potential is guarded by a complex and fine regulation of extracellular potassium concentration that is important to cardiac and skeletal muscle function. Acute and chronic hypokalemia and hyperkalemia may induce differences in the membrane potential of skeletal myocytes and cardiac myocytes, which may lead to muscular paralysis and fatal arrhythmia, respectively. Sudden cardiac death may be induced by dyskalemia and is a leading cause of death in people with hypertension, diabetes, cardiovascular disease, and chronic kidney disease. Dyskalemias are very prominent and closely associated with both DM, chronic kidney disease and cardiovascular disease. Both conditions raise the risk of hyperkalemia, mainly due to reduced renal

potassium excretion. Electrophysiological Effects of Potassium Because K+ serves as the primary ion mediating cardiac repolarization, the hypokalemic state is highly arrhythmogenic. Hypokalemic states produce complex effects on myocardial refractory periods and the potential for triggered arrhythmia. In contrast, hyperkalemia causes slowed conduction and conduction block which, if sufficiently progressive, can result in asystole. Hyperkalemia may also attenuate the effects of antiarrhythmic agents and repolarizing K+ currents.[7,19,31,32] Electrophysiological Effects of Potassium Since K+ is the main ion mediating cardiac repolarization, the hypokalemic state is strongly arrhythmogenic. Hypokalemic states generate complex effects on refractory myocardial periods and the potential for caused arrhythmia. Hyperkalemia induces risen K+ conductance in cardiac myocytes. [33] Since K+ current is responsible for repolarization, the first symptom of hyperkalemia is rapid repolarization. Improved K+ conductance also makes the RMP more negative to the level at which the Na+ channels start to be inactivated. [35] Hyperkalemia can also induce an atrioventricular nodal block. [20,21] With the rising frequency of hyperkalemia, the development of a characteristic of cardiac arrhythmia improves into ventricular fibrillation or asystole, leading to death.[31,34,36] In cardiac myocytes, potassium ion conductivity is directly correlated to plasma concentration K+.[31]Hypokalemia reduces K+ conductance and leads to a prolonged repolarization phase of the cardiac action potential.[12]Hypokalemic states produce complex effects on myocardial refractory Periods and the possibilities for arrhythmia. Hyperkalemia on the other side causes low conduction and conduction blocks which, if sufficiently progressive, can lead to asystole. Hyperkalemia may also attenuate the effects of antiarrhythmic agents and K+ repolarizing currents. [12,37] The National Council Potassium in Clinical Practice indicates that patients with congestive heart failure, cardiac arrhythmia or hypertension should maintain serum K+ levels of ≥4.0 mmol / L. [30]

Dyskalemia Related Cardiovascular Complications In Kidney Disease

The kidney plays an important role in the control of overall potassium reserves in the body.[15] In progressive chronic kidney disease, the body's ability to manage normokalemia in the face of changes in potassium intake and transcellular electrolyte shifts becomes progressively damaged. Chronic kidney disease estimates the possibility of hyperkalemia and related mortality. Hyperkalemia may lead to cardiovascular risk in chronic kidney disease in effect by direct impacts on cardiac conduction. Raises in extracellular potassium reduce the cardiomyocyte resting potential of the membrane, reducing the rate of rising of phase 0 of the cardiac action potential slowing of impulse conduction, excessive depolarization of the membrane, and reducing of repolarization time. [38,42,43] Hyperkalemia progression produces the commonly associated evolution of the surface electrocardiogram through peaked T waves, PR prolongation, QRS enlargement, sinoatrial arrest, sine wave appearance, and potentially, ventricular fibrillation and asystole. Bradyarrhythmias and heart blocks may also occur. Cardiovascular mortality.[6,39] Renal factors affecting K+ control include urinary flow rate, extracellular fluid volume diuretic intake, Na+ consumption, acid-base balance, mineralocorticoid toxicity, renal tubular disorder, and Mg++ depletion. Renal tubular acidosis is a typical presentation of hyperchloremic metabolic acidosis with a normal anion gap triggered by renal tubular dysfunction. Is triggered by abnormal urinary acidification owing to defective acid secretion in the collection ducts. There is also a related K+ conservation deficiency. [40,41]

Dyskalemia Related Cardiovascular Complications In Diabetic

Diabetic patients also develop electrolyte disorders. These are the results of insulin deficiency, hyperglycemia. Hyperglycemia sets the internal environment for osmotic diuresis while at the same time causing dilution in electrolyte concentrations. [44,46,59,60,61] The osmotic impact of glucose results in a reduction in circulating blood volume and a change in fluid from intracellular spaces that induces cell dehydration. These disorders are particularly common in decompensated diabetes, especially in diabetic ketoacidosis or non-ketotic hyperglycemic disorders. Hyperosmolary Syndrome. Diabetes mellitus is linked to hypo and hyperkalemia as well as hypo and hyperkalcemia, reflecting the coexistence of hyperglycemia-related mechanisms, which tend to change serum potassium and calcium in opposite directions. In diabetes mellitus, an increased incidence of electrolyte abnormalities due to various pathophysiological factors such as nutritional status, gastrointestinal absorption capacity, co-existing acid-based abnormalities, pharmacological agents, mainly renal disease alone or in combination, play a key role.[46] The causes of hypokalemia in diabetes include: (1) potassium K+ redistribution from the extracellular to the intracellular fluid compartment (2) gastrointestinal K+ loss due to malabsorption syndromes (diabetes-induced motility disorders, bacterial overgrowth, chronic diarrheal disorders) ;(3)K+ renal loss. Hypomagnesemia can induce hypokalemia, likely because a low intracellular concentration of Mg2 + stimulates the outer renal medullar K+ channel to further secrete it.[60] Hypokalemia is associated with impaired insulin secretion and decreased peripheral glucose utilization resulting in carbohydrate intolerance and hyperglycemia.[48,60] Exogenous insulin may induce slight hypokalemia because it promotes the entry of K+ into the skeletal muscles and hepatic cells by rising the activity of the Na+K+ ATP pump .[49] Increased epinephrine secretion due to insulin-induced hypoglycemia may also play a contributing role. [50] The majority of patients with diabetic ketoacidosis (DKA) are markedly K+ depleted. The average K+ deficit is 3-5 mEq/kg, but it can exceed 10 mEq/kg in some cases.[51,52] Several factors contribute to DKA-related potassium depletion, including vomiting, increased renal loss due to osmotic diuresis and ketoacide anion excretion, and cell loss of K+ due to glycogenolysis and proteolysis.[51,53] Hyperglycemia increases serum osmolality resulting in the movement of water out of cells. The loss of intracellular water leads to an increased intracellular K+ concentration, favoring a gradient for K+ to move out of the cells. Simultaneously, the friction forces between solvent (water) and solute can result in K+ being carried along with water through the water pores in the cell membrane.[46] Insulin treatment lowers K+ concentration driving K+ into cells. In diabetic subjects with hypertension, myocardial infarction/ischemia, or heart failure as chronic conditions, the risk of hypokalemia-related complications is particularly higher. Also, as diabetic patients are sometimes diuretic patients, diuretic-associated hypokalemia should be taken into account in this setting.[48] Hyperkalemia may be caused by an increase in plasma tonicity occurring from the redistribution of potassium from intracellular space to extracellular space.[53,63] In patients with type 2 diabetes, insulin-mediated glucose uptake is impaired but the cellular uptake of potassium remains regular, a condition consistent with the difference of intracellular pathways following the activation of the insulin receptor.[54] Potassium efflux from the cell is caused by intracellular dehydration due to the osmotic transcellular movement of water. This movement creates a favorable gradient for potassium efflux.[55] Reduced glomerular K+ filtration that interferes with the excretion of K+ is associated with hyperkalemia. The most important trigger factor for persistent hyperkalemia in diabetes is decreased

tubular K+ secretion due to hyporeninemic hypoaldosteronism syndrome. [47] Diabetic patients with medications proven to conflict with K+ homeostasis are at increased risk of hyperkalemia. [56,57] In such cases, close monitoring of K+ is fully justified [58].

IV. DISCUSSION

The function of potassium in regulating the capacity of the resting cell membrane, there are major risks of serious cardiac disease It is also important to note that there is a correlation between serum potassium defects and chronic kidney disease and DM. Conditions that have increased the risk. Hypokalemia and hyperkalemia may cause a change in the membrane potential of cardiovascular myocytes, resulting in fatal arrhythmia, and rapid cardiovascular death may occur in people with hypertension, diabetes, cardiovascular problem, and chronic kidney disease. Because physicians may be aware of the sensitive nature of this relationship with membrane potential, it is protected by the complex and fine regulation of extracellular potassium concentrations that are critical to cardiac and skeletal muscle function. The connection between intracellular and extracellular potassium plays a very important role in determining the electrophysiological properties of cardiac tissue. Potassium is the regulation of coronary function and the usual potassium level is the protection of potentially significant cardiovascular risk in the patient. Since potassium is essential for primary ion mediating cardiac repolarization Hypokalemia and hyperkalemia may cause a change in the potential of the membranes of cardiovascular myocytes, resulting in fatal arrhythmias, and sudden cardiovascular failure may occur in individuals with hypertension, diabetes, cardiovascular failure, and chronic kidney disease. Kidney disease observes the possibility of hyperkalemia and is related to the cardiovascular structure. Hyperkalemia or hypokalemia may add to the cardiovascular risk of chronic kidney disease to a limited extent through direct effects on the conduction of the heart. Diabetic nephropathy is one of the parts of diabetes mellitus, which ultimately leads to a renal problem which is the reason for electrolyte abnormality in diabetic patients. Electrolyte disorder is common in patients with diabetes, which may be the result of a change in electrolyte distribution, and is identified with hyperglycemia-instigated osmotic fluid movements or with complete body deficiency due to osmotic diuresis, the risk of death gradually increased with dyskalemia and was differentially higher in those with Heart Failure, chronic kidney disease or diabetes mellitus.

V. CONCLUSION

Dyskalemias are closely associated with cardiovascular disease, including those with chronic kidney disease and diabetes mellitus. Such disorders increase the probability of hyperkalemia, mainly due to reduced renal excretion of potassium. Risk factors for hyperkalemia involve kidney disease, heart disease, diabetes mellitus, potassium-sparing diuretics, angiotensin-converting enzyme inhibitors, b-blockers, angiotensin receptor blockers, angiotensin receptor blocker neprilysin combinations age high dietary potassium intake muscle injury and acidosis. Even if it is well recognized that hyperkalemia is important for people with chronic kidney disease and heart disease, who are mostly treated with drugs that block the renin-angiotensin-aldosterone system and therefore impair the ability of the kidneys to excrete potassium, it is less well recognized that hypokalemia is also related with an increased risk of death among these populations. They also found a U-shaped relationship between serum potassium concentration and all-cause mortality, But the frequency of the correlation of hypokalemia and mortality was stronger than that of hyperkalemia and mortality. Hypokalemia has been

reported to be associated with increased risk of death and hyperkalemia with increased risk of cardiovascular events among those enrolled in the Antihypertensive and Lipid-Lowering Treatment Study a strong association between the hyperkalemic event (inpatient or outpatient), hyperkalemia. These studies show that low-and high-potassium levels in the diet and blood are associated with an increased risk of death. Although these findings do not provide a causal link between dyskalemia and mortality, they recommend careful consideration and surveillance of serum potassium, especially in high-risk populations.

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