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ELECTROLYTE'S IMBALANCE AND ATRIAL FIBRILLATION

Emergency Medicine

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ABSTRACT

The contribution of the perpetuation of atrial fibrillation is caused by electrical remodeling in which calcium, sodium and potassium channels could refer to changes in the ion channel protein expression, development of fibrosis, gene transcription and ion channel redistribution. Calcium and magnesium could influence the risk of atrial fibrillation which is the leading cause of cardiac death, heart failure and ischemic stroke. The elevated serum concentration of calcium had a higher range of in-patient's mortality, increased total cost of hospitalization and increased length of hospital stay as compared to those without hypocalcaemia in atrial fibrillation patients. Moreover, chloride channels could affect homeostasis, atrial myocardial metabolism which may participate in the development of atrial fibrillation. Up to a 50% risk of incidence of AF are higher in which left ventricular hypertrophy, sudden cardiovascular death and overall mortality relate to a low serum magnesium level. Additionally, magnesium prevents the occurrence of AF after cardiac surgery, whereas greater levels of serum phosphorus in the large population-based study and the related calcium–phosphorus products were linked with a greater incidence of AF. Numerous clinical studies had shown the high preoperative risk of AF that is linked with lower serum potassium levels. The conventional risk factor of increased risk of new onset of AF events could independently link with high dietary sodium intake which enhances the fibrosis and inflammation in the atrium but the mechanism remains unknown. Many drugs were used to maintain the electrolyte imbalance in AF patients.

KEYWORDS

CALCIUM

Small conduction calcium-activated potassium channels are propagated of triggered impulses from the pulmonary veins to the atria that are caused by calcium. The expression of these channels increased due to the rapid stimulation. In the pulmonary veins, an action potential is shortened. Within the atria, heart failure has resulted because of the structural changes and remodeling of calcium cycling which support persistent atrial fibrillation. Hypocalcaemia is linked with left ventricular hypertrophy, shortened QT interval, hypertension, vascular calcification and arrhythmias. On the other way, hypocalcaemia is related to prolonged QT intervals, heart failure and life-threatening cardiac arrhythmias. Both decreased and increased serum concentrations of calcium are linked with increased mortality.

Deo M et al.'s study had reported in cardiac cells; intracellular calcium dynamics have been recognized as an important contributor in life-threatening ventricular arrhythmia including ventricular fibrillation as well as ventricular tachycardia and increasing the prevalent atrial arrhythmias such as flutter and atrial fibrillation.

Chloride

After sodium, the most abundant electrolytes in serum is the chlorine which has a key role in the regulation of body fluids, acid-base status, electrolyte balance, and the preservation of electrical neutrality.

Moreover, the function of chlorine channels involves cell volume regulation, regulation of excitability, ionic homeostasis, transepithelial transport, etc., in the plasma membrane. Duan et al. reported that the chloride channel was related to various cardiovascular diseases including hypertension, ischemic, myocardial hypertrophy and heart failure.

The evidence of the significance of chloride intracellular channel 4 (CLIC4) was involved in cellular differentiation, endothelial tubulogenesis, apoptosis and inflammation. CLIC4 expresses in cardiomyocytes, lung alveolar septae and vascular endothelial cells. By modulating mitochondrial function, chloride channels play an important role in cardio protection from ischemic-reperfusion injury and cardiac function. Additionally, Jiang et al.'s study demonstrated the up regulated CLIC1, 4, 5 is differentially expressed in patients with atrial fibrillation. The authors indicate in results that chloride channels could affect homeostasis, atrial myocardial metabolism and also participate in the development of atrial fibrillation.

Magnesium

The fourth most important and abundant cation is the magnesium in the human body as well as intracellular tissues and also the second most prevalent cation. Many physiologic roles of magnesium involve in protein transport, enzyme activity and become an essential part of all adenosine triphosphate-utilization systems. It is related to cardiovascular disorders; for example, reducing dietary intake of magnesium has been related to a higher risk of AF, hypertension, ischemic heart disease, heart failure-related hospitalization and new-onset heart failure. On the different organ systems, magnesium has various significant pharmacological as well as physiological effects that involve the mechanism of action such as membrane stabilization, calcium antagonism and regulation of energy transfer.

Moreover, up to a 50% risk of incidence of AF are higher, in which left ventricular hypertrophy, sudden cardiovascular death and overall mortality relate to a low serum magnesium level. Intravenous magnesium directly affects myocardial potassium channels, prolongs the PR interval, has voltage-dependent and indirect effects on sodium and calcium channels and elevated the refractory period of ante grade atrioventricular node conduction.

Phosphorus

Phosphorus is an essential mineral that is naturally present in many foods and available as a dietary supplement. Phosphorus is a component of bones, teeth, DNA, and RNA. It is important for many biologic functions, such as energy exchange, cellular signal transduction as well as mineral metabolism and also is an independent predictor of atrial fibrillation.

Potassium

Potassium (K) is an important macro mineral nutrient and principal cation in intracellular fluid, which regulates the osmotic pressure; muscle contraction participates in acid–base balance, cell membrane function and more in human. A high dietary intake of potassium has a protective role against the kidneys, cardiovascular system and bones diseases.

The risk of cardiovascular disease has increased especially cardiac arrest and ventricular arrhythmias that had shown an association with serum potassium (< 3.5 mmol/l), especially in hypokalemia. Various studies had reported the relationship between the risk of AF and serum potassium. Moreover, numerous clinical studies had shown that the high preoperative risk of AF was linked with lower serum potassium levels.

Sodium

Sodium allows to build up an electrostatic charge on cell membranes as well as transmission of nerve impulse when the charge is allowed to dissipate by a moving wave of voltage change in the organism. It is also classified as a dietary inorganic micro mineral for animals. Moreover, Frisoli et al.'s study had stated the independent relation of blood pressure and high salt intake that could increase the risk of heart failure, stroke, protein uric renal disease and left ventricular hypertrophy (LVH)

The conventional risk factor of increased risk of new onset of atrial fibrillation events is independently linked with high dietary sodium

intake. They had reported the first study about the relationship between the cumulative incidence of AF events and dietary sodium intake. It is also possible that AF is connected with sodium intake which enhances fibrosis and inflammation in the atrium but the mechanism remains unknown. Similarly, Cavusoglu et al.'s study reported that hyponatremia was independently related to the occurrence of atrial fibrillation.

The occurrence of AF had increased the hypokalemia and hyponatremia. Pulmonary veins and sinoatrial nodes play a critical role in the pathophysiology of AF. So, low sodium, as well as low potassium, was differentially modulated pulmonary veins and sinoatrial node electrical properties. Low sodium and low potassium-induced slowing of sinoatrial node beating rate and genesis of pulmonary veins burst firing which could contribute to the higher occurrence of AF during hyponatremia or hypokalemia. In addition, Takase et al.'s study also highlights the association of salt intake with the presence and development of AF in the general population, including other factors rather than salt intake had a much more prominent impact on the progress of AF. Further, the authors had been suggested the complementary role of salt intake for the prediction of atrial fibrillation.

There are certain clinical entities, including chronic heart failure, pneumonia, coronary artery bypass surgery, chronic kidney disease and hypomagnesaemia, all of which could result in hyponatremia which also predispose the patient to the process of atrial fibrillation. There was a strong negative correlation between serum sodium level and heart rate. The main cause of hyponatremia was the reason for the high ventricular rate in patients with lower concentrations of sodium that blocks the inhibitor function of the AV node on accessory pathways.

CONCLUSION

This review concludes that electrolytes imbalance plays a significant role in the pathogenesis of atrial fibrillation. To manage the electrolytes imbalance in AF subjects, numerous drugs were used. Future studies need to find the exact mechanism of these electrolytes in AF. More research studies are required to find the diet management of these electrolytes in AF subjects.

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