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Potassium Intake in Neurological Diseases

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Mini Review

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Abstract

Potassium is a crucial mineral for the muscles as well as the nervous system to function adequately because one of its main objectives is to cooperate with sodium to regulate the amount of fluid inside and outside the cells. As an electrolyte, potassium is known to increase cardiac and muscle activity. Also, the mineral supports the memory function of the brain. In addition, potassium increases memory and learning in healthy individuals. This is one of the main reasons why symptoms reflecting a low level of potassium are synonymous with neurological disease symptoms. In this context, the intake of potassium can be applied as a support for neurological diseases. This mini-review will discuss the potential role of potassium in several neurological diseases.

Keywords: Potassium; Neurological Diseases; Dietary

Introduction

Potassium (K^+) is the crucial cation of intracellular environment. 98% of the total body potassium, which is about 3500-4000 mmol in an adult, is found in the cell. The high concentration of K^+ in the cell is essential for the continuation of normal cell functions such as cell metabolism and growth, cell division, optimal enzyme function, DNA synthesis, volume regulation, and acid-base balance [1]. In particular, skeletal muscles, liver, erythrocytes and bones are rich in potassium. In total, although 2% is in the extracellular area, the stimulant effect on the nerves and muscles makes potassium one of the most vital electrolytes. Potassium is an essential mineral for the muscles as well as the nervous system to function adequately because one of its main objectives is to cooperate with sodium to regulate the amount of fluid inside and outside the cells [2]. The mineral also plays a role in the transmission of electrical impulses and messages from the nerves. This is one of the main reasons why symptoms that reflect a low potassium level are synonymous with neurological disease symptoms.

The resting membrane potential in structures is closely related to the difference in extracellular-intracellular K^+ concentration. In an average diet, 50-150 mmol K^+ is taken daily. Although dietary K^+ intake varies widely from day to day, plasma K^+ levels remain within the narrow limits of 3.5-5.0 mmol/L. This sensitive K^+ balance is achieved by the displacement of potassium between the intracellular and extracellular environments within minutes and by the renal and intestinal excretion (external equilibrium) that occurs within hours-days. The external K^+ balance refers to the balance between the amount of potassium taken and discarded [3]. Degradation of balance may provide a basis for neurological diseases.

Hypopotasemia (hypokalemia) is defined as the plasma K^+ level falling below 3.5 mmol/L (mEq/L). Hypopotasemia may develop as a result of K^+ shifts in the cell, decreased net K^+ intake, or increased net K^+ loss. Hyperpotassemia (hyperkalemia) is the increase of plasma K^+ level above 5.0 mmol / L. It may develop due to positive K^+ balance due to increased uptake or decreased renal excretion or K-shifting out of the cell [4].

Electrolyte abnormalities are common in daily clinical practice and their diagnosis is based on routine laboratory findings. Acute and severe electrolyte imbalances can be manifested by epileptic seizures that can give a single symptom [5]. Unlike other electrolyte abnormalities, hypokalemia or hyperkalemia rarely causes symptoms in the CNS and seizures do not occur. Although adequate K^+ intake in healthy adults is relatively high, for example in patients with a reduced renal function in patients with a tendency to develop 4.7 g per day, especially hyperkalemia, a dietary K^+ restriction of less than 3 grams per day is generally recommended in the treatment of patients with reduced renal function.

Changes in serum levels of extracellular K^+ exert their effects mainly on the function of cardiovascular and neuromuscular systems [6]. Severe K^+ anomaly can therefore lead to fatal arrhythmia or muscle paralysis before CNS symptoms occur. Physicians should therefore be aware of the presence of acute seizures due to electrolyte disturbances and be able to understand the underlying medical conditions that lead to electrolyte imbalance, as this may lead to a way of controlling the disease and initiating a rapid and appropriate treatment.

The likelihood of lifestyle factors such as diet, especially K^+ intake, to change the risk of stroke is suggested by several observational cohort studies, including some new reports. According to the research, K^+ diet is associated with low risk of stroke. High K^+ intake, especially in non-hypertensive women, is associated with fatal risk in all women with the risk of stroke and ischemic stroke. Numerous studies have found that low potassium intake and low serum potassium are associated with increased stroke mortality [7]. Low serum K^+ is associated with a particular risk of stroke in a few diuretic users with atrial fibrillation. Further studies are needed to determine whether changing these factors will prevent paralysis.

Hypertension is the lethal risk factor for stroke. Clinical, experimental and epidemiological evidence suggest that high amounts of K^+ intake are associated with low blood pressure [8]. These findings support the hypothesis that high amounts of potassium from food sources can protect against paralysis-related death.

The effect of high dietary K^+, calcium and magnesium intake on cerebral function is less clear, but iron is also found in Alzheimer's neuropathology. It has been investigated whether this mineral diet intake is associated with a slight risk of cognitive impairment. These findings suggest that dietary mineral intake known to take place in biological processes associated with vascular and Alzheimer's pathology may contribute to disease progression at the beginning of the disease process and may require more attention [9]. In contrast, evidence supports the use of ketogenic dietary treatments for the adult epilepsy, adult malignant glioma and Alzheimer's disease [10]. Since each of these pathophysiological factors may be affected by dietary manipulation, it is reasonable for the diet to change the course and outcome of other neurological diseases that share these and common pathways.

Conclusion

The role of K^+ intake in neurological diseases is remarkable. It is still controversial. Experimental animal models such as epilepsy and multiple sclerosis should be studied before the clinical trials to develop projects that carry out a preliminary study. This can be a key parameter in milder forming the course of the disease.

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