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HYPOKALEMIA

ABSTRACT

A low serum potassium concentration is perhaps the most common electrolyte abnormality encountered in clinical practice. Of the total body potassium content, 90% is sequestrated within cells. This compartmentalization depends on active transport through the cell membrane by a sodium-potassium pump. Normal serum potassium levels are considered to lie roughly between 3,6 and 5,0mmol/L. Hypokalemia may be diagnosed when the serum potassium level is less than 3,6mmol/L. Potential causes include inadequate dietary potassium intake, as well as depletion induced by abnormal losses. Hypokalemia may occur rarely becauses of an abrupt shift of potassium from the extracelular compartement into cells. Sometimes hypokalemia may be familial. Genetic hypokalemia is linked to disorders of mineralocorticoid hormone synthesis or action (glucocorticoid-remediable hyperaldosteronism, congenital adrenal hyperplasia, apparent excess of mineralocorticoids), to renal tubular disorders (Liddle's syndrome, Bartter's and Gitelmann's syndrome, tubular acidosis) or to disorders of cellular transfer of potassium (hypokalemic periodic paralysis). In other case drugs prescribed by physicians are the most common cause of hypokalemia.

Patients with hypokalemia often have no symptoms. In severe hypokalemia generalized weaknes, lassitude and constipation are common. In patients with myocardial ischemia, heart failure, left ventricular hypertrophy middle-to-moderate hypokalemia increases the likelihood of cardiac arrhythmias. Hypokalemia increase systolic and diastolic blood pressure when sodium intake is not restricted.

Potassium replacement is the cornerstone of therapy. A dosage of 20mmol/24h of potassium in oral form is generally sufficient for the prevention of hypokalemia and 40 to 100mmol/24h is sufficient for its treatment.

Key words: hypokalemia