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Perioperative Potassium Disorders

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Abstract

Because of the high incidence and the association with serious unexpected adverse events during and after surgery, perioperative K^+ disorder has aroused widespread concern, especially in patients with cardiac and renal diseases. Potassium assessment and management need for improved perioperative standards and greater reliability in the approach to surgical patients. It is necessary to evaluate the severity of K^+ disorder and whether there is a life-threatening emergency according to the patient's history, symptoms, sign and laboratory tests. When $K^+ < 2.5$ mmol/L or $K^+ > 6.5$ mmol/L, serious cardiac damage seems inevitable and an immediate intervention is suggested.

Electrolyte abnormalities are common in perioperative settings, among which potassium disturbance has attracted wide attention because of its association with serious adverse events, such as life-threatening arrhythmia, cardiac arrest and respiratory muscle paralysis. Abnormalities in K^+ uptake, excretion, distribution and interaction between K^+ and other ions can affect the perioperative K^+ homeostasis.

Approximately 98% of the body potassium is found in the intracellular fluid and 2% in the extracellular fluid [1]. Under normal conditions, the intracellular potassium concentration is 140-150 mmol/L, and the extracellular (including plasma and interstitial fluid) potassium ion concentration is 3.5-5.0 mmol/L. The intracellular potassium concentration is 30 times or more than the extracellular concentration. The gradient of potassium between the inside and outside of the cell is mainly maintained by Na^+-K^+-ATPase, and its concentration difference can maintain the generation of resting potential and the generation and conduction of electrical excitation.

Due to multiple factors such as preoperative gastrointestinal preparation, fasting, the effects of comorbidities and medications, perioperative blood K^+ concentration fluctuates and the incidence of acute K^+ disorder increases perioperatively [2]. Hypokalemia (defined as a serum or plasma $K^+ < 3.5$ mmol/L) is more likely to occur than hyperkalemia (defined as a serum or plasma $K^+ > 5.5$ mmol/L). It is reported that the incidence of hypokalemia in hospitalized patients is about 21.0%, and in emergency patients it is as high as 49.9%, and that patients undergoing gastrointestinal surgery have a higher risk of hypokalemia, up to 70.37%, even if they are admitted to the hospital with normal routine tests [3,4]. Because of the high incidence and the association with serious unexpected adverse events during and after surgery, potassium management have aroused widespread concern.

Hyperkalemia can be diagnosed when the blood potassium is more than 5.0 mmol/L, and attention should be paid to distinguish it from pseudohyperkalemia [5]. It is necessary to evaluate the severity of hyperkalemia and whether there is a life-threatening emergency according to the patient's history, symptoms, sign and laboratory tests. It is suggested that acute hyperkalemia be divided into mild (5.0 mmol/L ~ 5.9 mmol/L), moderate (6.0 mmol/L ~ 6.4 mmol/L) or severe (≥ 6.5 mmol/L) according to serum potassium ion concentration and ECG changes such as peaked T wave, flattened P wave, prolonged PR interval, widened QRS and even ventricular fibrillation [1,6].

Hypokalemia can be classified as mild (blood potassium 3.0-3.4 mmol/L), moderate (blood potassium 2.5-2.9 mmol/L), or severe (blood potassium < 2.5 mmol/L), and the more severe the degree, the more likely it is to be symptomatic. Potassium disturbance is closely related to adverse cardiovascular events. The effect of hypokalemia on the heart is mainly changes in electrocardiogram (ECG), arrhythmia and impaired myocardial function caused by changes in physiological properties of cardiomyocytes and conduction bundles. In patients with poor basal cardiac function, severe hypokalemia can induce or aggravate cardiac insufficiency. Among them, arrhythmia is the most serious complication of hypokalemia. Potassium between 3.0 and 3.5 mmol/L cause ECG changes (flattening or inversion of T waves). In the range of 2.5-3.0 mmol/L, hypokalemia can cause significant prolongation of the Q-T interval, reduced U- and P-wave amplitudes, flattening of the T-wave, ST-segment depression (0.5 mm), atrioventricular block (prolonged PR-interval), and premature ventricular contractions. $K^+ < 2.5$ mmol/L is associated with atrial fibrillation

and multifocal atrial tachycardia, premature atrial and ventricular contraction, bradycardia, ventricular fibrillation, syncope, sudden cardiac death, and heart failure. Besides, moderate and severe hypokalemia can also cause neuromuscular symptoms, such as muscle weakness and fatigue.

There is still controversy about the impact of potassium on the ability to perform surgery due to uncertainty in clinician acceptance of potassium risk values. Considering that potassium disturbance can increase the risk of intraoperative arrhythmias, potassium levels are often a concern of anesthesiologists and a consideration for whether to cancel or postpone scheduled surgery. This paper points out that there is a lack of consensus on the impact of hyperkalemia on schedule surgery, which is due to the differences in doctors' perception and acceptance of hyperkalemia risks [7]. However, patients with a K^+ value ≥ 6.0 mmol/L within 24 hours of a scheduled surgery were 2.40 times more likely to have a cancelled surgery compared to patients with a K^+ between 5.1-5.9 mmol/L, which is in line with the clinical situation. As for hypokalemia, it is not necessary to postpone the surgery when 2.8 mmol/L ≤ K^+value ≤ 3.5 mmol/L, but reasonable supplement should be given during the operation to maintain potassium homeostasis. If K^+ is lower than 2.8 mmol/L, the decision to operate should be made on the basis of the patient's status, and potassium supplementation should be considered before surgery in patients with arrhythmias.

Since the causes of potassium disturbance are multifactorial, the managements are to deal with the root cause as well as to correct the risk factors. The treatment depends on the severity, symptoms and ECG abnormalities. For patients with hyperkalemia with or without ECG changes, immediate use of intravenous calcium is the first-line treatment, which can antagonize the effect of potassium on myocardial action potential and stabilize cell membrane electricity. There are also some managements to promote the intracellular transfer of potassium, including insulin-dextrose mixture, beta-2 agonists; carbonate (if combined with acidosis) and diuretics (if without oliguric stage of renal failure or severe hypovolemia). In mild hypokalemia, oral potassium supplementation (10-20 mmol each time) is the first choice. If patients cannot tolerate it and have gastrointestinal reaction, intravenous potassium supplementation should be considered when necessary. In moderate hypokalemia, consider intravenous potassium supplementation. In severe hypokalemia, intravenous potassium supplementation is preferred. Generally, 10-15 ml of 10% potassium chloride injection is added to 500 ml of 0.9% saline for continuous intravenous infusion, and it should be noted that the concentration of potassium supplementation should not exceed 40 mmol/L and the rate should not exceed 20 mmol/L. At the same time, continuous electrocardiogram and blood potassium monitoring should be performed and adjusted accordingly.

Except ketamine, most anesthetic drugs have certain degrees of inhibitory effects on sympathetic nerve and cardiac function, so the tolerance of myocardial cells to K^+ disorders in patients during operation decreases. When K^+ < 2.5 mmol/L or K^+ > 6.5 mmol/L, serious cardiac damage seems inevitable and an immediate intervention is suggested. It showed that serum potassium might predict short-term outcomes after surgery and preoperative hypokalemia was an independent risk factor for overall complications [8].

ERAS procedures were particularly associated with a lower supplementation of potassium and a higher incidence of hypokalemia in patients after surgery. As a result, hypokalemia might delay the postoperative recovery of gastrointestinal motility, and animal experiments have further demonstrated that hypokalemia impairs intestinal permeability and leads to bacterial translocation, both of which are detrimental to the postoperative recovery of gastrointestinal function [4,9]. Oral or intravenous K^+ supplementation could be an adopted ERAS program for the patients with high risk factors of K^+ disorders undergoing surgery (Table 1).

Overall, clinicians should pay attention to potassium disorders in the perioperative period, especially in

Table 1: Clinical manifestations and managements of hyperkalemia and hypokalemia.

	Hyperkalemia	**Hypokalemia**
Clinical manifestation		
Gastrointestinal tract	Nausea, vomiting, diarrhea	Ileus, constipation
Neuro-muscular	Weakness, ascending paralysis, respratory failure	Weakness, fatigue, tendon reflexes paralysis, respiratory failure
Heart	Cardiac arrhythmia	Cardiac arrest, pulseless electric activity, asystole
ECG	Peaked T wave, flattened P wave, prolonged PR interval, widened QRS, VF	U Waves, ST-T changes, cardiac arrhythmia
Managements	Calcium therapy; Insulin-dextrose; beta-2 agonists; carbonate if combined with acidosis; loop or thiazide diuretics; dialysis	Oral or intravenous potassium supplement

patients with cardiac and renal diseases. Potassium assessment and management need for improved perioperative standards and greater reliability in the approach to surgical patients.

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