Review Article

An overview on diagnostic and management approach of plasma potassium imbalances in emergency setting

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Abstract

Introduction: One of the most important investigations of the emergency room is the routine electrolytes test, which, in many cases, will provide some overall picture of the patient's status and symptoms. Electrolyte imbalance may be fatal, therefore, early detection and treatment are crucial. These ion imbalances are prevalent with nearly a fourth of hospitalized patients. Potassium imbalance is one of the most important issues, due to its complications and many causes that can be behind it such as: vomiting and drugs. Clinicians should familiarize themselves with such disorders, in order to prevent complications, and provide the best outcome possible in such cases. **Objectives:** In this article, we aimed to review and discuss the pathophysiology of potassium imbalance, and its clinical features and management in an emergency department setting. **Methods:** PubMed database was used for articles selection, papers on were obtained and reviewed. **Conclusion:** In most patients, presenting with hypo or hyperkalemia, the cause can be apparent from history. The role of an ER physician goes deeper with the usage of laboratory tests as a confirmation, along with close cardiovascular monitoring and treating other imbalances. The clinician shall never be afraid of being more aggressive in treating potassium imbalances, especially when severe, symptomatic, refractory, and/or cardiac complications were noted. This can include IV administration of potassium (if the patient is hypokalemic), and 24 hours ECG monitoring. However, some patients may still need hospitalization and a further multidisciplinary approach may be needed.

Keywords: Potassium imbalances, pathophysiology, clinical features, diagnosis, and management

Introduction

Fluids and electrolytes are essential elements to homeostasis; they have an important role especially at the cellular level (e.g. cell signaling, tissue perfusion, and acid-base balance). [1] Fluids and electrolytes' disturbances are common problems in the emergency room (ER), and in severely ill patients. [2] Potassium, one of the integral electrolytes, has imbalances in (21%) of hospitalized patients and (2–3%) of outpatients.[3]

Physicians are required to be skillful when taking history and examining patients as electrolyte imbalance symptoms are often generalized and non-specific; subsequently, ordering the relevant diagnostic tools should be prioritized before applying therapeutic measures. Disturbances within many bodily systems are linked to electrolyte imbalances, most notably: gastrointestinal, renal, and endocrine systems.[4, 5] In this paper, the pathophysiology, clinical features, and emergency management of potassium imbalance is discussed.

Methodology:

Using Pubmed, Google Scholar, and Microsoft Academic, the following keywords and terms were used: electrolytes, hypokalemia, hyperkalemia, and potassium. Mesh terms used were: ((“electrolyte imbalance"[Mesh]) AND (“Pathophysiology"[Mesh] OR "Management"[Mesh] OR "Emergency")) other terms were also used including: ((“hypokalemia "[Mesh]) AND (“Pathophysiology"[Mesh] OR "Management"[Mesh] OR "Emergency")). In regards to

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the inclusion criteria, the papers were selected based on the inclusion of one of the following topics; hypokalemia, hyperkalemia, diagnosis, clinical features, and management. Exclusion criteria were all other papers that had none of the topics as their primary endpoints.

Discussion

Potassium is an abundant intracellular fluid (ICF) cation. Of the 3,500 mEq that constitutes the total pool of the body, about (98%) is confined to the intracellular fluid (ICF), at concentrations of (140–150mEq/l), and only (2%) is located within the extracellular fluid (ECF). [6] A special portal impeded within the cell membrane called sodium-potassium adenosine triphosphatase pumps is considered as the regulatory gate between the ECF and ICF concentration of potassium. [7] Since the ICF content of potassium is higher than ECF, the change of the total potassium content will influence a fluctuation of the ECF concentration rather than the ICF. [6] Moreover, the potassium gradient results in the process of resting membrane potential, influencing the cardiac and neuromuscular cells excitability and signaling. [6, 8]

The importance of this cation comes in the integration of normal cellular function, especially of neurons and myocytes; it has an essential role in the action potential; therefore, the tight regulation of its concentration is necessary to keep the norm.[6] Therefore, the regulation of potassium concentration includes hormones such as: catecholamines and insulin that manipulate the ICF. [9, 10] However, the ECF is managed solely by the renal system, hence, (90%) of potassium is excreted in the urine. Sweat loss and stool formation contribute to the remaining (10%).[6, 8]

Pathophysiology of Hypokalemia

Hypokalemia occurs when the serum potassium concentration drops below 3.5mEq/l (normal range: 3.5-5.5mEq/l). The causes can be divided into four major groups: firstly, dilution hypokalemia and decreased intake; secondly, transcellular misdistribution; thirdly, loss of potassium by way of the gastrointestinal tract; and finally, loss of potassium through urine. [6, 11] Furthermore, a low potassium level causes disturbances in electrical conduction at the cellular membrane, which consequently impairs the neuromuscular function. [12] This leads to muscular weakness, and cardiac conduction-abnormalities—due to hyperpolarization at the neuromuscular junction level. [13, 14]

Pathophysiology of Hyperkalemia

Hyperkalemia is the condition in which the potassium value exceeds 5.5 mEq/L. This disease is life-threatening, especially at 7.0mEq/L levels and more, at such concentrations cardiotoxic effect ensues. [4] Incrementing potassium in the serum could be due to elevated potassium intake, decreased renal potassium excretion, or a shift of potassium from the intracellular to the extracellular space. [4, 6]

Additionally, one of the most common drugs that can cause hyperkalemia are angiotensin-converting enzyme inhibitors, trimethoprim-sulfa, and potassium-sparing diuretic agents can adversely cause hyperkalemia. Moreover, in metabolic acidosis, there is a tendency for potassium to transfer from the intracellular space into the extracellular space, in exchange with hydrogen ions. [4, 15] The excretion of potassium ions by the renal tubular cells is decreased due to the increased hydrogen ion content. (Hyperkalemia often occurs in the setting of renal failure. [4, 16]

Clinical Features of Hypokalemia

Usually, the signs and symptoms start appearing when the levels are near/at (2.5 mEq/L). the signs accompany severe hypokalemia are: paralysis, muscle cramps, lethargy and confusion, paresthesia, respiratory muscle impairments, inability to concentrate urine, anorexia, vomiting, and decreased bowel mobility. [12, 13] Due to the tendency of HCO_3 reabsorption promotion at the renal proximal tubule, this can sustain metabolic acidosis. In turn, alkalosis drives potassium intracellularly, thus, more worsening of the potassium imbalance. [5, 17]

The ECG findings usually appear at the potassium levels of (2.5 mEq/L) and below, these findings may include: depressed ST segment, flattening or T wave inversion, presence of elevated U wave, high P wave amplitude; prolongation of PR interval; and prolonged QRS interval. Cardiac arrhythmias that may appear as a consequence include sinus bradycardia, primary heart block, paroxysmal atrial tachycardia, and atrioventricular dissociation. Hypokalaemia does not commonly lead to life-threatening arrhythmias.[14, 18]

Clinical Features of Hyperkalemia

The cardiac and neurological symptoms of a patient with hyperkalemia are due to impaired neuromuscular transmission. Symptoms may include muscular weakness or paralysis, general fatigue, paresthesia, and palpitations [4] The ECG changes include peaked T-waves, a decrease in, or absence of, P waves, a prolonged PR interval, bundle branch blocks, and a sequential progression in the widening of the QRS complex to resemble a sine wave, ventricular fibrillation and finally a-systole; These ECG abnormalities can ensue when the serum potassium concentration exceeds (7.5 mEq/L). [14] These abnormal cardiac excitations caused by hyperkalemia are more detrimental to the patient than are those that accompany hypokalemia. [16, 19]

Management of Hypokalemia

Severe hypokalemia necessitates an aggressive intravenous substitution and continuous cardiopulmonary monitoring. [4] Potassium phosphate or preferably potassium chloride are recommended as potassium salts; potassium bicarbonate is recommended only in the specific setting of metabolic acidosis. [19] An important note is that the rate is more critical than the total amount administered. [20] The outlines of emergency treatment include cardiovascular observation and

close laboratory monitoring. Initiate potassium chloride (KCl) infusion, the adjusted dose must not exceed 20 mmol/L/h, as there is potential rebound hyperkalemia at higher dosages. The infusion rate must be adapted to the severity of the clinical disorder but is generally a (10 mmol/L) KCl IV over 5–10 min in life-threatening hypokalemia. Any deficits in magnesium should be sufficiently repleted. Thereafter, an underlying cause should be identified and managed appropriately as to prevent any future disturbances. [5]

Management of Hyperkalemia

Immediate treatment is required especially in the presence of any ECG abnormality due to the potential fatal arrhythmias. Thus, the determination of life-threatening levels of potassium is essential. [21] It is recommended that any patient presenting with ECG abnormalities correlated with hyperkalemia, or a serum level of 6mml/L and more should have immediate management (Table 1). [4, 22] When the patient has markedly persistent hyperkalemia, hemodialysis management is preferred. [23]

Table 1. Emergency treatment in hyperkalemia—hyperkalemia and ECG abnormalities, acute hyperkalemia .6.0 mmol/l

Continuous ECG monitoring
10–20 ml of 10% calcium gluconate intravenously over 2–5 min with abnormal ECG
Effect within 1–3 min, lasting for 30–60 min
Can be repeated if there is no effect within 5–10 min
Use extreme caution in patients taking digitalis.
10 U of regular insulin with 50 ml of 50% dextrose IV
No dextrose necessary in hyperglycemic patients
Effect onset within 15–20 min and peak within 30–60 min, lasting for 4–6h
Expected reduction in the concentration of plasma potassium: 0.5–1.5mmol/l
Careful monitoring of glucose.
20–60 ml of 8.4% $NaHCO_3$ intravenously—not proven unanimously
Haemodialysis
Ethacrynic acid 50–100 mg IV or Furosemide 40–80 mg IV
Sodium polystyrene sulphonate
Oral dose: 20 g with 100 ml of 20% sorbitol every 4–6 hours
Effect after 4–6h
Enema: 50g with 50ml of 70% sorbitol plus 100–150 ml water, retained for at least 30–60 min, better 2h
Intestinal perforation and necrosis described in the post-operative patient; laxatives
Other than sorbitol may be preferable.
Coexisting electrolyte disturbances have to be corrected

Conclusion

Delay in recognition and fastidious management of electrolyte disturbances can be of disastrous consequences, in particular of cardiac and neurological injury. Many researches have been done regarding potassium disturbance, to establish a clear etiological and pathological basis and thus, establishing a better approach for a better outcome. Despite there are no specific symptoms, quick identification is still of medical importance to prevent further deterioration. The treatment depends on the potassium amount in the serum. In hypokalemia, establishing whether it is caused by a cellular shift, or by an ionic deficit is essential. Potassium phosphate or potassium chloride, are recommended as potassium salts; the adjusted dose must be at 20 mmol/L/h. Management of hyperkalemia should have priorities for transient cardiac membrane stabilization and intracellular shift of potassium until the kidney can provide adequate elimination. A dose of 10–20ml of 10% calcium gluconate, followed by 10units of regular insulin with 50ml 50% dextrose can be sufficient in most cases in controlling the elevated serum potassium. Familiarizing oneself with clinical features and applying the correct therapeutic plans are key to managing electrolyte disturbances.

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