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Review Article

Common electrocardiogram manifestations in electrolyte imbalance

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ABSTRACT

Abnormalities in serum electrolyte levels and impaired electrolyte homeostasis may aberrate cardiac conduction by altering cardiac ion current kinetics. Oftentimes, the bedside electrocardiogram (ECG) can render expeditious insight and prompt emergency therapy while laboratory investigations of fluid, acid-base and electrolyte imbalances are underway for establishing a definitive diagnosis. The most frequent electrolyte aberrations affecting the ECG include disturbances of potassium, calcium, and magnesium concentrations. Electrolyte dyshomeostasis impacts the depolarization and repolarization phases of action potential in cardiomyocytes by modifying potentials across their cell membranes. Further, individual electrolyte disturbances often have wide-ranging consequences due to their effect on one another. Serum potassium concentration changes can cause considerable repercussions on myocyte conduction and potentially resulting in ECG changes. The ECG changes in hypokalemia mimic those of myocardial ischemia. With severe hypokalemia, the telltale ECG change is the development of U waves. In hyperkalemia, the primary ECG manifestation is T wave tenting, that is seen as a symmetrical narrowing or peaking but with oftentimes a wide deflection and high amplitude. With very severe hyperkalemia, a slurring “sine-wave” appearance may be observed on the ECG due to significant prolongation and widening of QRS complex and subsequent fusion with the T wave. Hyper-and hypo-calcemia mainly change the action potential span (phase 2), which causes either shrinking (in hypercalcemia) or extension (in hypocalcemia) of the QT interval. The effect on the QT interval is mainly the result of an alteration of the ST-segment time span. Both situations can impact T wave structure. No classic ECG presentation is observed in hypo-or hyper-states of magnesium. Bedside electrocardiography serves as an important aid in forming diagnoses and managing patients presenting with electrolytic dyshomeostasis.

Keywords: ECG, Electrolyte abnormality, Dysrhythmias

INTRODUCTION

The cardiac action potential is generated through voltage-gated ion channels mediating flow of ion currents via specific channels embedded in the cell membrane. Abnormalities in serum electrolyte levels and impaired electrolyte homeostasis may aberrate cardiac conduction by altering cardiac ion current kinetics. These disturbances may be associated with clinically unimportant alterations in the surface ECG or lead to lethal dysrhythmias. Oftentimes, the bedside ECG can render expeditious insight and prompt emergency therapy while laboratory investigations of fluid, acid-base and electrolyte imbalances are underway for establishing a definitive diagnosis. The clinical disorders that cause hypo- and hyper-electrolytemia are related to the most frequent and clinically significant abnormalities of cardiac rhythm linked with electrolyte imbalance.[1] Although reviewed individually in the discussion, it is essential to consider the presence of dynamic physiologic interactions among electrolytes, and that individual electrolyte disturbances may have wide-ranging consequences due to their effect on one another.

LITERATURE SEARCH

A literature search for was carried out the national library of medicine database and Google Scholar search engines to identify publications addressing common ECG changes found in electrolyte imbalance conditions. Only articles in the English language were included. A limit to "full text" availability was put in the search query. Potentially relevant scientific articles were identified. The reference lists of identified publications were also scanned. Further, grey literature on the childhood chronic illnesses was also identified using Google search engine.

DISCUSSION

The most frequent electrolyte aberrations affecting the ECG include disturbances of potassium, calcium, and magnesium concentrations. Electrolyte dyshomeostasis impacts the depolarization and repolarization phases of action potential in cardiomyocytes by modifying potentials across their cell membranes. As mentioned earlier, though the electrolytes are reviewed individually below, combination of disorders is possible clinically due to the dynamic physiologic interrelationship between these cations.

Potassium

Potassium plays a significant role in maintaining the potential across cell membrane, and in depolarization and repolarization of the myocytes. Serum potassium concentration changes can cause considerable repercussions on myocyte conduction and potentially resulting in ECG changes. Although sometimes ECG changes do not accompany serum potassium disturbances, electrocardiography is beneficial for screening and gauging the scale of serum potassium abnormality and the urgency of medical care.[2,3]

Hypokalemia

Hypokalemia is a consequence of renal and gastrointestinal losses, extra–intracellular shift, or insufficient potassium consumption.[4] A diagnosis of hypokalemia is common due to the predominance of patients on medications. Other conditions that precipitate hypokalemia are diarrhea and vomiting. Additionally, it may co-occur with metabolic disturbances like hypomagnesemia. As serum potassium concentration falls, the transmembrane potassium gradient is reduced. The impact on the cell membrane is a rise in the resting membrane potential and prolonging of the action potential, specifically phase 3 cardiac repolarization and refractory periods.[5]

The primary ECG change related with hypokalemia is a lowering of the T wave amplitude. As potassium concentrations decrease even more, depression of the ST segment and actual T wave inversions may be observed. Prolongation of the PR interval can occur in addition to a rise in P wave amplitude. With severe hypokalemia, the telltale ECG change is the development of U waves. The U wave is a positive deflection succeeding T wave that is most commonly observed in the mid-precordial leads, such as V2 and V3. These alterations have been noted in over 80% cases with potassium levels below 2.7 mEq/L.[6] With severe hypokalemia, prominent U waves can often slur the smaller prior T waves or after P waves.[7] The ECG changes in hypokalemia mimic those of myocardial ischemia. Additionally, it may be challenging to distinguish a U wave from a notched T wave that is found in hyperkalemic patients. In such cases, the notched T wave possesses a narrow base with widening of the QRS interval in some cases.

Due to the larger duration of action potential and refractory period, hypokalemic cases are more prone to developing certain dysrhythmias.[5] The extended QT/QTU interval precisely, can result in Torsades de pointes or ventricular tachycardia. A pseudo-prolonged QT interval may occur that, in truth, is a QU interval with a missing T wave. It has also been noted that hypokalemic cases are more predisposed to ventricular fibrillation.[8] Therapeutic intervention targets supplementation of potassium via parenteral and oral routes, and discerning of cause of hypokalemia.

Hyperkalemia

According to several studies, hyperkalemia correlates well with ECG manifestations. ECG irregularities have been observed in patients with serum potassium elevations as small as 5.5 mEq/L.[2] Moreover, there is a foreseeable ECG progression with further elevation of serum potassium levels. With the elevated extracellular potassium levels, transmembrane permeability is

heightened, resulting in an ingress of potassium into the myocytes. The transmembrane potential gradient undergoes as shift, with a lowering of the resting potential and the speed of phase 0 of the action potential. The influx of potassium results in the shrinking of the action potential and causes a delay in myocytic conduction.[2] As a result, these changes lead to a slowing of conduction. A number of ECG manifestation in hyperkalemic cases stem from these conduction aberrations. The first ECG manifestation is T wave tenting, that is seen as a symmetrical narrowing or peaking but with oftentimes a wide deflection and high amplitude.[9] Further, pseudo-normalization (upright flip) of inverted T waves related to left ventricular hypertrophy can occur with hyperkalemia.[10] An accelerated terminal phase of repolarization is responsible for these T waves manifestations and are mainly observed in the precordial leads. This phenomenon is often noted when potassium concentrations rise above 5.5 mEq/L.

Suppression of cardiac conduction follows further elevation of serum potassium. Cellular action potential reduces as a result of sodium channel inactivation propagated by a fall in atrial and ventricular transmembrane potential. Because of earlier sensitization of atrial tissue, flattened P wave and prolonged PR interval are observed prior to QRS interval extension. These changes usually take place when potassium concentrations rise beyond 6.5 mEq/L.[5] Further increase in serum potassium concentration causes a progressive diminishing of P wave, as the atrial myocytes become more sensitive with rising potassium levels.

Sinoatrial and atrioventricular conduction gets suppressed as the serum concentrations elevate beyond twice their normal concentration. These consequently lead to sinoatrial and atrioventricular blocks, accompanied often by escape beats. Other blocks such as intraventricular conduction delay, bundle branch block and fascicular blocks are also possible. It has been reported that bypass tracts show higher sensitivity to conduction delays due to potassium rise, that can cause the ECG to normalize and lose the delta wave in case of Wolff-Parkinson-White syndrome.

With very severe hyperkalemia, a slurring "sine-wave" appearance may be observed on the ECG due to significant prolongation and widening of QRS complex and subsequent fusion with the T wave. This observation is a pre-terminal event unless prompt therapy is begun. The life-threatening event is either asystole due to total ventricular conduction block, or ventricular fibrillation.[5]

Even though the abovementioned ECG development explains the hallmark features of hyperkalemia, these manifestations are not necessarily seen every time. Metabolic disturbances like alkalosis, hypernatremia, and hypercalcemia can counter the transmembrane impacts of hyperkalemia and dampen the ECG manifestations related to high potassium concentrations.[11]

Therapeutic intervention for fatal hyperkalemia target blocking the consequences on myocyte transmembrane potential and cardiac conduction and reducing extracellular potassium. Treatment response is immediate and noticeable on ECG. Calcium is effective in checking the actions of extracellular potassium rise on cardiomyocytes within minutes of delivery by reestablishing a more suitable electrical potential across the cell membrane. Calcium is generally given as calcium chloride or gluconate, although calcium chloride provides a greater quantity of calcium per unit volume. It must be noted though that calcium may promote toxic dysrhythmias like asystole in case of digitalis toxicity. Sodium bicarbonate, magnesium, beta 2 adrenergic agonists, and a preparation of glucose and insulin, all effect intracellular transport of potassium thereby reducing the extracellular potassium concentration. At the end, surplus body potassium may be eliminated using sodium polystyrene or dialysis.

Calcium

Extracellular calcium ion levels are strictly controlled via an intricate homeostatic mechanism involving the parathyroid hormone and regulated by effector cells present in kidney, bone and intestine. The potential between intracellular and extracellular calcium is 1000- to 10,000-times, achieving swift transmembrane shifts via "gated" channels. Calcium is essential for a multitude of modulatory processes, skeletal muscle contraction, regulation of enzymatic reactions, and is a major player in myocyte electrical activity and myocardial contraction.[12] Hyper-and hypo-calcemia mainly change the action potential span (phase 2), which causes either shrinking (in hypercalcemia) or extension (in hypocalcemia) of the QT interval. The effect on the QT interval is mainly the result of an alteration of the ST-segment time span. Both situations can impact T wave structure.

Hypocalcemia

Hypocalcemia is usually observed with functional deficiency of parathyroid hormone, either as a total absence (primary type), succeeding parathyroidectomy or in relation to a pseudo-deficiency. Other etiologies involve vitamin D deficiency, congenital disorders related to calcium regulation, chronic kidney failure, acute pancreatitis, and sepsis. Hypocalcemia is generally seen in cases of terminal illness, with incidence reports exceeding 50% cases.[13] Further, in some cases, hypocalcemia is linked with hypomagnesemia. Neuromuscular irritability is the key characteristic of hypercalcemia, with carpal-pedal spasm being the main physical observation that may proceed to frank tetany, laryngospasm, or tonic-clonic seizure manifestations.

The principal ECG change of hypocalcemia is the elongation of the QTc interval. Hypo-calcemic states extend phase 2 of action potential with the effect regulated via the rate of change of serum calcium levels

and activity of the myocytic calcium channels. Elongation of the QTc interval is related with early after-repolarizations and precipitated dysrhythmias. Torsades de pointes may result from hypocalcemia although it is less prevalent than with hypokalemia and hypomagnesemia.

While ECG conduction aberrations occur frequently, extreme hypocalcemia-induced dysrhythmias like heart block and ventricular dysrhythmias are less common.[13] The onset of dysrhythmias is often linked with other comorbidities like structural cardiopathies, ischemia or in relation to drug therapy like for digitalis and catecholamines. Grave symptoms and fatal dysrhythmias necessitate prompt therapeutic interventions in the form of parenteral calcium salts. Additionally, related electrolyte disturbances involving hypomagnesemia, phosphate disturbances and acidemia might need correction. Vitamin D and oral calcium supplements can be given in chronic cases.

Hypercalcemia

Hypercalcemia is the hallmark feature of hyperparathyroidism. It is usually chronic, mild with low morbidity. Extreme hypercalcemia with the serum concentrations more than 14 mg/dL can be caused in these cases if dehydration arises due to loss via gastrointestinal tract, diuretics administration, consumption of large quantities of calcium salts. Hypercalcemia is primarily seen in cases of metastatic non-parathyroid cancers. An advanced rated of bone resorption markedly raises the amount of filtered calcium and deranges sodium reuptake from kidneys, resulting in a cascade of volume decline and exacerbated hypercalcemia. Symptomatology of hypercalcemia is comparatively indistinct, involving fatigue, dullness, diminished motor strength, anorexia, nausea, constipation and abdominal discomfort.

The ECG changes in hypercalcemia are the opposite of hypocalcemia with classical presentation of irregular shrinkage of QTc interval. Medically important rhythm aberrations related to hypercalcemia are infrequent as the rise in extracellular calcium is commonly not related to precipitated dysrhythmias. Disturbances involving cardiac conduction are possible, with the most prevalent type being bradydysrhythmias.[14]

Treatment for hypercalcemia is usually initiated based on the clinical presentation rather than the serum concentrations, though empiric treatment is often initiated at concentrations of 14mg/dL in asymptomatic cases as well. In case of hypoalbuminemia, assessed serum calcium concentration may obscure high increases in free ionized extracellular calcium. The cornerstone of therapy is intravenous volume repletion and bisphosphonates that suppress osteoclastic bone resorption. Loop diuretics encourage calciuresis but are not recommended in case of hypovolemia. Extreme cases may need dialysis.

Magnesium

Magnesium, a predominantly intracellular cation, contributes to numerous enzymatic reactions and is important in hormonal modulation. It is essential for the maintenance of cellular ionic balance with the regulation of sodium, potassium, and calcium. Magnesium therapy was traditionally indicated in cases of pregnancy related eclampsia. Current indications of magnesium include acute wide-complex tachydysrhythmias. It has also shown therapeutic potential in cases of asthma, myocardial infarction, and acute cerebral ischemia.[15]

Hypomagnesemia

The most frequently observed magnesium abnormality is hypomagnesemia which results due to decreased dietary consumption, higher losses, or change in intracellular–extracellular distribution. In contrast to other electrolyte imbalances, hypomagnesemia does not have a hallmark presentation, and measured serum concentrations do not show accurate correlation with clinical signs and symptoms. A wide range of physiological adversities are precipitated by hypomagnesemia such as central nervous system consequences like seizures, altered mental status, cardiovascular sequelae such as dysrhythmias and vasospasms, endocrine problems like hypokalemia and hypocalcemia, and muscular issues like bronchospasm and muscle weakness.

Hypermagnesemia

Hypermagnesemia is generally diagnosed in cases of renal failure involving excessive ingestion of magnesium salts by patients. Intravenous dosing errors are often implicated in symptomatic iatrogenic cases. Extreme symptoms comprise central nervous system depression, areflexia, respiratory failure, and in rare circumstances, cardiac arrest.

No classic ECG presentation is observed in hypo-or hyper-states of magnesium. Hypomagnesemia is implicated in the indirect potentiation as well as direct onset of supraventricular and ventricular dysrhythmias. Magnesium is beneficial in the treatment of atrial tachydysrhythmias. However, rhythm conversion and serum magnesium levels do not show correlation. Magnesium sulfate is administered as a first-line drug in the management of torsades de pointe.[16] Lastly and briefly, sodium imbalances show no classic ECG manifestations on their own but in cases of intraventricular conduction disturbances primarily stemming from hyperkalemia, hypernatremia shrinks, and hyponatremia elongates the QRS interval.[17]

CONCLUSION

Although most of the ECG irregularities observed in patients in the emergency department or intensive care are a result of cardiac diseases primarily, ECG changes

do not always indicate a cardiac disease. Cardiac action potential and cardiomyocytic depolarization and repolarization are impacted by intracellular and extracellular concentrations and distributions of cations such as potassium, calcium, and magnesium. Alterations in extracellular concentrations of these electrolytes can have an enormous influence on cardiac conduction. ECG manifestations related to these imbalances can aid in their diagnosis and management.

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