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Review Article

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Electrocardiogram Morphology Changes in Patients with Electrolytes Imbalances

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ABSTRACT

Electrolyte imbalances are a common occurrence in patients with various medical conditions, and their impact on cardiovascular health cannot be underestimated. The electrocardiogram (ECG) is a valuable tool for monitoring cardiac function, and changes in its morphology can provide important insights into electrolyte abnormalities. Electrolyte abnormalities can be life-threatening, and their management depends on the speed of onset of the electrolyte's derangement and the patient's existing comorbidities. Electrolyte imbalances can affect the heart's electrical activity and can be seen on an ECG. The specific ECG changes will depend on the specific electrolyte imbalance. This article explores the relationship between electrolyte imbalances and morphological alterations in the ECG. By understanding these changes, healthcare professionals can improve the diagnosis, treatment, and management of patients with electrolyte disturbances, ultimately enhancing patient outcomes and reducing the risk of cardiac events.

Background

One of the ways to assess the effects of electrolyte imbalances on the heart is through electrocardiogram (ECG) morphology changes. An ECG records the heart's electrical activity and can detect abnormalities in its rhythm and conduction system. Electrolyte imbalances can cause distinct changes in the ECG, which can serve as important diagnostic clues. A common electrolyte abnormality that affects the ECG is hyperkalemia, which refers to elevated levels of potassium in the blood. In patients with hyperkalemia, the ECG typically shows tall, peaked T-waves, prolonged PR intervals, and widened QRS complexes. These abnormalities reflect a delay in ventricular repolarization and can lead to potentially life-threatening arrhythmias, such as ventricular fibrillation [1].

On the other hand, hypokalemia, a potassium deficiency, can also cause ECG abnormalities. In patients with hypokalemia, the ECG often reveals flattened T-waves, prominent U-waves, and a prolonged QT interval. The prolonged QT interval increases the risk of developing a type of arrhythmia known as torsades de pointes, which can lead to fainting or sudden cardiac arrest. Another electrolyte imbalance that affects the ECG is hypercalcemia, which is an excess of calcium in the blood. Hypercalcemia can cause a shortening of the QT interval on the ECG, which predisposes individuals to a condition called ventricular tachycardia. Ventricular tachycardia is a rapid heart rhythm originating from the ventricles, and if left untreated, it can deteriorate into ventricular fibrillation, potentially resulting in cardiac arrest. Conversely, hypocalcemia, a calcium deficiency, can also have ECG effects. The ECG may show a prolonged QT interval in patients with hypocalcemia, similar to the ECG changes seen in hypokalemia. The prolonged QT interval increases the risk of developing arrhythmias such as torsades de pointes [2].

Introduction

Electrolyte abnormalities can be life-threatening, and their management depends on the speed of onset of the electrolyte derangement and the patient's existing comorbidities. Electrolytes are important in many of the reactions in the human body, such as muscle contraction and conduction of action potentials; thus, their derangement can cause widespread and not always obvious clinical signs and symptoms [3]. Electrolyte imbalance is usually diagnosed in the lab, but clinical signs (such as absent reflexes or peripheral oedema) and other investigations (such as tall T waves on the ECG) may point to an underlying defect that needs to be investigated and ruled out or treated [4].

The electrocardiogram is an extremely sensitive method of detecting certain types of electrolyte imbalance. The form of the normal electrocardiogram depends on the cells' normal ionic constitution and, in particular, the extracellular fluid that bathes the cardiac cells [5]. Any signs of alteration in or the ratio between the electrolyte content of this fluid may directly or indirectly result in significant electrocardiographic changes. The electrolytes which may produce the most profound effects of the electrocardiogram when they deviate from their normal levels are calcium and potassium [6]. The important electrolytes pertaining to the electrocardiogram are potassium, calcium, and the relative concentration of these substances among themselves. Acidosis

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has an effect as well. Many electrolytes, such as sodium and magnesium, known to alter the ECG in experimental animals, do not produce changes in man because they do not reach low concentration levels [7].

One specific area of interest in electrocardiogram morphology changes is its association with electrolyte imbalances. Electrolytes are electrically charged particles that play a crucial role in maintaining the proper functioning of cells, organs, and bodily systems. Imbalances in electrolyte levels can profoundly affect the heart's electrical activity, leading to significant changes in the ECG waveform. One of the most commonly observed ECG morphology changes in patients with electrolyte imbalances is the presence of prolonged QT interval. The QT interval represents the time from the beginning of the QRS complex to the end of the T wave and reflects the duration of ventricular depolarization and repolarization. Electrolyte imbalances, such as low levels of potassium (hypokalemia) or high levels of calcium (hypercalcemia), can lead to a lengthening of the QT interval, increasing the risk of life-threatening arrhythmias, such as Torsades de Pointes [8].

Another important ECG morphology change associated with electrolyte imbalances is the appearance of U waves. U waves are small, positive deflections that follow the T wave and are usually not visible under normal conditions. However, the U wave becomes more pronounced and visible in patients with electrolyte imbalances, particularly hypokalemia. The exact mechanism behind U wave formation is not fully understood, but it is believed to be related to abnormal repolarization of the ventricles [9]. In addition to QT interval prolongation and U wave appearance, electrolyte imbalances can also cause alterations in the ST segment and T wave morphology. For example, hyperkalemia, characterized by high levels of potassium, can lead to ST-segment elevation, peaked T waves, and even the development of ventricular fibrillation. On the other hand, hypocalcemia, or low calcium levels, may result in ST segment depression and flattened T waves.

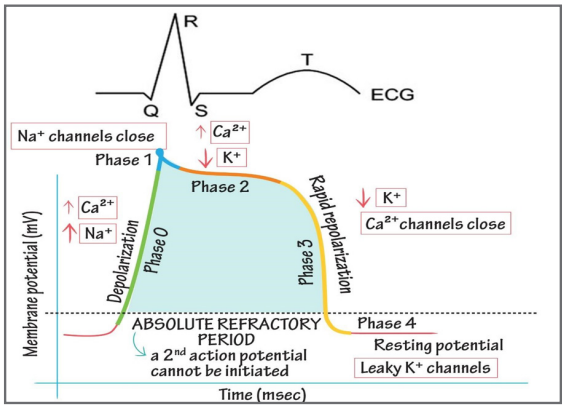
ECG Changes due to Electrolyte Imbalance (Electrolyte Disorder)

The normal cardiac action potential may be altered by electrolyte imbalance due to intra and extracellular electrolyte concentration changes. Because myocyte depolarization and repolarization depend on intra and extracellular shifts in ion gradients, abnormal serum electrolyte levels can profoundly affect cardiac conduction and the electrocardiogram EKG [10]. Changes in extracellular potassium, calcium, and magnesium levels can change myocyte membrane potential gradients and alter the cardiac action potential. These changes can result in incidental findings on the 12-lead EKG or precipitate potentially life-threatening dysrhythmias [11]. The EKG may be used to estimate electrolyte imbalances' severity and assess the risk of severe arrhythmias. This is possible because a correlation exists between the severity of the electrolyte imbalance and the changes visible to the EKG.

Electrophysiology of the Heart

Depolarization: The electrical charge of a cell is altered by a shift of electrolytes on either side of the cell membrane. This change stimulates muscle fibres to contract. Repolarization: Chemical pumps re-establish an internal negative charge as the cells return to their resting state [12].

The normal state of cardiac cell membrane polarization depends upon maintaining a normal ionic balance across the membranes, with K+ being the most important. Because changes in intracellular K+ concentration are proportionately much smaller than changes in extracellular K+ concentration, it follows that the absolute level of extracellular K+ concentration is the single most important factor affecting the cell membranes [13].



Cardiac muscle cell action potential

- **Phase 0: rapid depolarization phase**
  - Sodium (Na+) rapidly into the cell.
  - Calcium (Ca++) slowly into the cell.
- **Phase 1: early Repolarization phase**, Sodium (Na+) channels closed.
- **Phase 2: plateau phase**
  - Potassium (K+) rapidly out of the cell.
  - Calcium (Ca++) slowly into the cell.
- **Phase 3: Rapid Repolarization phase, Calcium (Ca++) channels closed.**
  - Potassium and sodium ion positions are reversed.
  - Potassium (K+) rapidly out of the cell.
- **Phase 4: resting potential phase**, the cell membrane is impermeable to Na+, and K+ moves out of the cell [14].

Specific Electrolyte Disorders

Sodium (Na+): Increased (hypernatraemia) and decreased (hyponatremia) sodium levels do not have any effect on the ECG, nor cardiac rhythm or impulse conduction (Non-significant changes on the ECG) [15].

Electrocardiogram Morphology Changes Associated with Hypercalcemia

Calcium (Ca++): Changes in extracellular calcium concentration profoundly affect the duration of the plateau (phase 2) of the action potential. The plateau duration increases at low extracellular calcium concentrations and shortens at high calcium concentrations [16].

Hypercalcemia, or high calcium level: is a common metabolic emergency that occurs when serum calcium is elevated above the normal range [13].

- Normal serum corrected calcium = 2.1 - 2.6 mmol/L
- Mild hypercalcaemia = 2.7 - 2.9 mmol/L
- Moderate hypercalcaemia = 3.0 - 3.4 mmol/L
- Severe hypercalcaemia = greater than 3.4 mmol/L

Etiology: Primary hyperparathyroidism and malignancies cause 90% of all cases of hypercalcaemia. Less common causes are Prolong immobilization, sarcoidosis, thyrotoxicosis, familial hypocalciuric hypercalcaemia, Addison's disease, renal failure, tamoxifen, lithium, thiazide diuretics, D vitamin, and excessive intake of calcium supplements [3].

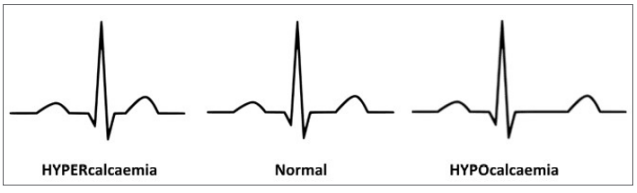
ECG Changes due to Hypercalcemia

A shortened QT interval is one of the most common ECG morphology changes seen in patients with hypercalcemia. The QT interval represents the time the heart repolarizes or resets itself after each contraction. Higher calcium levels can disrupt hypercalcemia's normal repolarisation process, leading to a shortened QT interval on the ECG. This change is significant because a shortened QT interval can increase the risk of developing life-threatening arrhythmias, such as ventricular tachycardia.

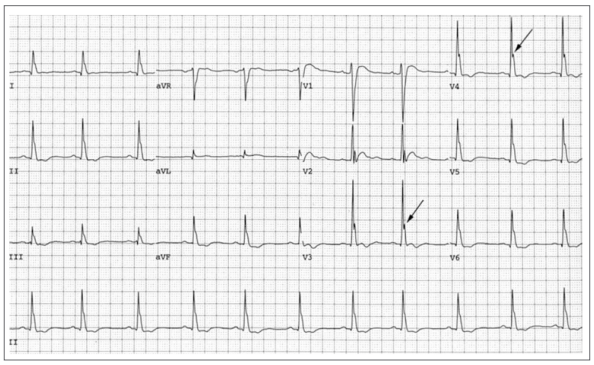
Another ECG morphology change seen in hypercalcemia is an increase in the amplitude of the T wave. The T wave represents the repolarization of the ventricles of the heart. In hypercalcemia, high calcium levels can affect the movement of potassium ions, which are crucial for the repolarization process. This disruption can lead to an exaggerated T wave on the ECG, indicating abnormal ventricular repolarization. Additionally, hypercalcemia can also cause a U wave to appear on the ECG. The U wave is a small deflection seen after the T wave, representing the repolarization of the papillary muscles in the heart. In hypercalcemia, the increase in calcium levels can disrupt the normal repolarization process of these muscles, leading to the appearance of a U wave on the ECG. This change is significant because an increased U wave can also increase the risk of developing arrhythmias [17].

It is important to recognize and understand these ECG morphology changes in patients with hypercalcemia because they can help in the early identification and management of this electrolyte imbalance. By identifying these specific ECG changes, healthcare providers can initiate appropriate treatment strategies to correct the hypercalcemia and prevent potential cardiac complications. Regular monitoring of the ECG in patients with hypercalcemia is essential to assess the effectiveness of treatment and ensure the restoration of normal cardiac function [18].

The main change is the shortening in the Q-T interval on the ECG. The T wave duration is unaffected, but the ST segment duration is shortened. Patients with hypercalcemia are more sensitive to digitalis and may present with various arrhythmias [19]. In severe hypercalcemia, Osborn waves (J waves) may be seen. Ventricular irritability and VF arrest have been reported in severe hypercalcemia.



ECG Example



Osborn Waves in Severe Hypercalcemia

The interval between the onset of QRS and the onset of the T wave (Q-oT) became shorter at higher calcium levels, although the T wave was pro- longed, and the QT interval became more normal (Surawicz and Knilans, 2008). Hypercalcemia usually does not alter the P and T waves' morphology, but a slight, statistically significant increase in T wave duration was reported.

Electrocardiogram Morphology Changes Associated with Hypocalcemia

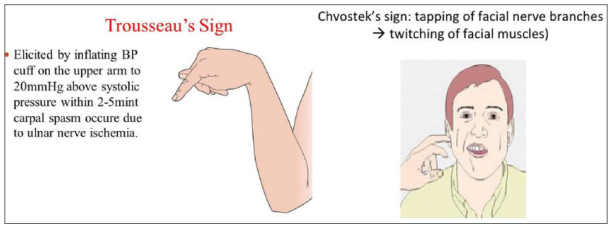
Hypocalcemia is a condition characterized by low levels of calcium in the blood. Calcium plays a crucial role in various physiological processes, including proper cardiac function. When calcium levels are decreased, it can lead to changes in the electrical conduction system of the heart, reflected in ECG findings. One of the most notable ECG morphology changes associated with hypocalcemia is the prolongation of the QT interval. The QT interval represents the time taken for both ventricular depolarization and repolarization. The prolonged QT interval in hypocalcemia is primarily due to delayed ventricular repolarization. This delay increases the risk of developing life-threatening arrhythmias, such as torsades de pointes, a polymorphic ventricular tachycardia.

Another ECG morphology change related to hypocalcemia is the presence of a shortened ST segment. The ST segment reflects the period between ventricular depolarization and repolarization. In hypocalcemia, the decreased calcium levels result in early repolarization of the ventricles, leading to the shortened ST segment. This change can potentially be misinterpreted as myocardial ischemia or other cardiac conditions. Furthermore, hypocalcemia can cause the appearance of U waves on the ECG. U waves are small, positive deflections that follow the T wave. They represent the final phase of ventricular repolarization. In hypocalcemia, the decreased calcium levels disrupt the normal balance between potassium and calcium currents during repolarization, leading to the appearance of U waves. However, it is important to note that this change is not specific to hypocalcemia and can also occur in other electrolyte imbalances or cardiac conditions.

Hypocalcemia or low calcium level: occurs when calcium levels fall below the normal range [3].

- Normal serum corrected calcium = 2.2 - 2.6 mmol/L.
- Mild-moderate hypocalcaemia = 1.9 - 2.2 mmol/L.
- Severe hypocalcaemia = < 1.9 mmol/L.

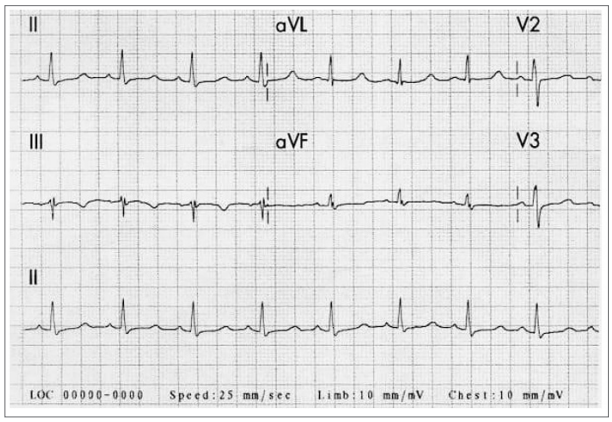
Etiology: Acute pancreatitis, pancreas surgery, alkalosis (hyperventilation), rhabdomyolysis, septicemia (sepsis), osteolytic cancer metastases, abnormal calcium absorption (gastrointestinal) and resorption (from primary urine), renal failure, small bowel syndrome, parathyroid gland surgery, use of bisphosphonates, excess calcitonin, use of phenytoin, use of phosphate substitution [14].



ECG Changes due to Hypocalcaemia

The main ECG change is **prolongation of the Q-T interval**. There is no unchanged in T wave duration, but the ST segment is prolonged.

- Hypocalcaemia causes QTc prolongation primarily by prolonging the ST segment.
- Dysrhythmias are uncommon, although atrial fibrillation has been reported.
- Torsades de pointes may occur but is much less common than with hypokalemia or hypomagnesaemia [20].



Hypocalcaemia Causing a Long QTc (510ms)

The duration of the ST segment is inversely related to the plasma calcium concentration. Usually, lengthening of the ST segment, Q-aT, and the QT interval are the only ECG abnormalities; but the QTc interval seldom exceeds 140 percent of normal [16].

Electrocardiogram Morphology Changes Associated with Hyperkalemia

One of the most noticeable ECG changes associated with hyperkalemia is the presence of peaked T-waves. Under normal circumstances, the T-wave represents the repolarization of the ventricles. However, in hyperkalemia, the high potassium levels disrupt the normal potassium-sodium balance across the myocardial cell membrane, increasing the action potential duration. This prolonged repolarization manifests as tall, narrow, and peaked T-waves on the ECG. The rarer the condition, the more pronounced the T-wave changes will likely be [21].

Another significant ECG morphology change observed in hyperkalemia is the widening of the QRS complex. The QRS complex reflects the depolarization of the ventricles, and in hyperkalemia, the increased intracellular potassium levels impair the conduction system, leading to delayed or slowed depolarization. This results in a prolonged QRS duration, exceeding the normal 80-120 milliseconds range. The widened QRS complex can indicate ventricular conduction disturbances, such as bundle branch blocks, which are commonly associated with hyperkalemia [22].

Furthermore, hyperkalemia can also lead to the disappearance of P-waves on the ECG. The P-wave signifies the depolarization of the atria, but potassium-induced conduction abnormalities can disrupt the normal atrioventricular (AV) conduction, causing atrial depolarization to be masked within the QRS complex. Consequently, the P-wave may become so closely fused with the QRS complex that it is no longer identifiable, resulting in a "pseudo-normalization" of the P-wave. It is worth noting that the ECG morphology changes associated with hyperkalemia can be potentially life-threatening. In severe cases, excessively high potassium levels can cause complete heart block or ventricular fibrillation. Therefore, prompt recognition of these changes is crucial for timely intervention, as hyperkalemia should be treated aggressively to restore a normal potassium level and prevent cardiac arrhythmias [23].

Potassium (K+): Potassium plays a key role in both depolarization and repolarization, which is why potassium imbalance may cause dramatic ECG changes. These are of utmost clinical significance. There is a rather strong correlation between plasma potassium level, ECG changes, and the risk of arrhythmia. Therefore, the ECG may be used to estimate the severity of hyperkalemia [3].

Hyperkalaemia: is a common cause of electrolyte-induced cardiac conduction disturbance. A well-defined series of changes at the cellular level leads to characteristic evolutionary changes in the surface electrocardiogram. Initial high T waves and shortened intervals give way to prolongation of conduction and lethal dysrhythmias as the serum potassium level rises.

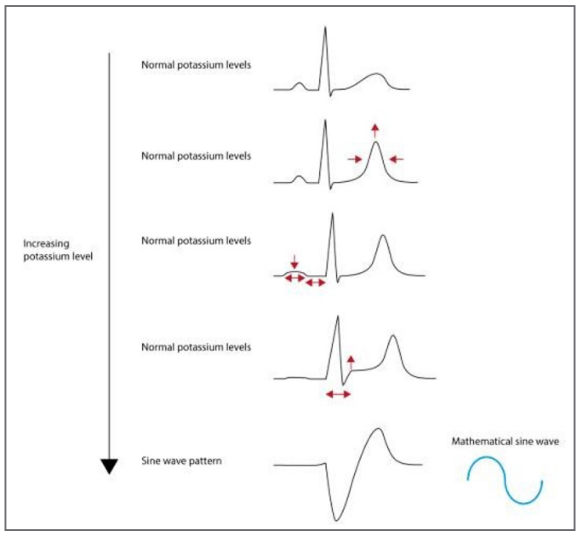
- Hyperkalaemia is defined as a potassium level > 5.5 mEq/L
- Moderate hyperkalaemia is a serum potassium > 6.0 mEq/L
- Severe hyperkalaemia is serum potassium > 7.0 mE/L

Etiology: Severe hyperkalemia is usually the result of several interacting factors, such as renal failure, insufficient corticosteroid substitution, acidosis, hemolysis and massive muscle damage. Potassium substitution may be the etiology. Potassium-sparing diuretics, ACE inhibitors and angiotensin receptor blockers (ARBs) may also cause hyperkalaemia. Insulin deficiency, Addison's disease and digoxin intoxication may also cause hyperkalemia [24].

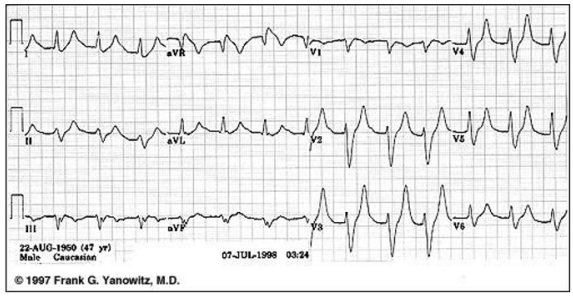
The typical progressive changes of hyperkalaemia are as follows:

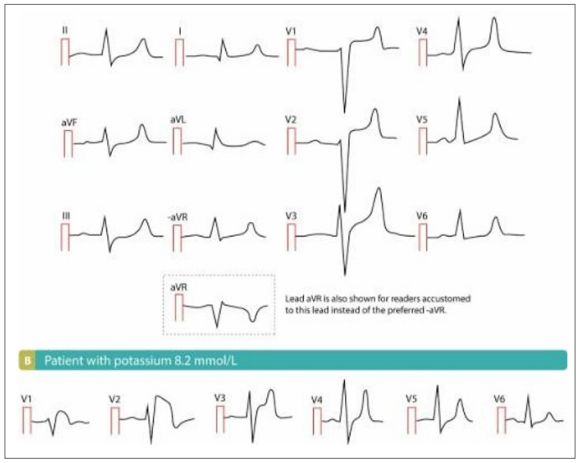
1. The appearance of tall, pointed, narrow T waves.
2. Decreased P wave amplitude decreased R wave height, widening of QRS complexes, ST segment changes (elevation/depression), Hemi block (esp. left anterior) and 1st-degree heart block [25].
3. Advanced intraventricular block (very wide QRS with RBBB, LBBB, and bi- or tri-fascicular blocks) and ventricular ectopic.

4. Absent P waves, very broad, bizarre QRS complexes, AV block, VT, VF or ventricular asystole [26].



Serum potassium level of > 9.0 mEq/L causes cardiac arrest due to: Asystole Ventricular fibrillation PEA with bizarre, wide complex rhythm ECG changes seen in hyperkalaemia.





Electrocardiogram Morphology Changes Associated with Hypokalemia

Hypokalemia refers to a condition where there is an abnormally low concentration of potassium ions in the bloodstream. Potassium plays a crucial role in various physiological processes, including maintaining the normal electrical functioning of the heart. Electrocardiogram (ECG) is a commonly used diagnostic tool to assess the electrical activity and rhythm of the heart. In patients with hypokalemia, there are distinct ECG morphology changes that can provide valuable insights into the impact of this electrolyte imbalance on cardiac function [27].

One of the most notable ECG morphology changes seen in hypokalemia is the appearance of flattened T waves. T waves represent the repolarization of the ventricles, and in a normal ECG, they should be rounded and upright. However, in hypokalemia, potassium deficiency can disrupt the normal repolarization process, leading to the flattening or inversion of T waves. This alteration in T wave morphology can be observed in multiple leads of the ECG, indicating a generalized effect on ventricular repolarization [28].

Another characteristic ECG morphology change associated with hypokalemia is the development of U waves. U waves are small deflections that follow the T waves, representing delayed repolarization of the Purkinje fibers and papillary muscles. These U waves are typically not seen in a healthy ECG, but in hypokalemia, they can become prominent and taller. The appearance of U waves in ECG can be attributed to the prolonged ventricular repolarization caused by low potassium levels. In severe cases of hypokalemia, a potentially life-threatening ECG morphology change known as Torsade de Pointes can occur. Torsade de Pointes is characterized by a twisting pattern of the QRS complex around the baseline, resembling a ribbon. If not promptly treated, this arrhythmia can lead to ventricular fibrillation and sudden cardiac death. The development of Torsade de Pointes in hypokalemia is believed to be due to the prolongation of the QT interval, which measures ventricular depolarization and repolarization. Hypokalemia can prolong the QT interval, increasing the risk of arrhythmias, including Torsade de Pointes [1].

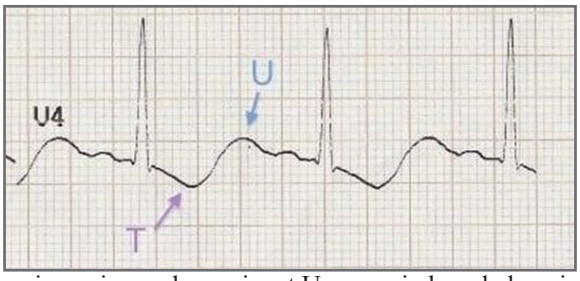
Hypokalaemia: the serum potassium level drops below 3.5 mEq/L. In moderate [3].

- Hypokalaemia is defined as a potassium level < 3.5 mmol/L
- Moderate hypokalaemia is a serum level of < 3.0 mmol/L
- Severe hypokalaemia is defined as a level < 2.5 mmol/L

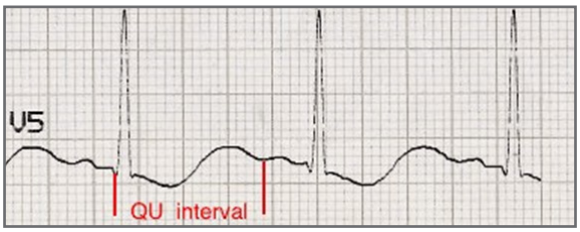
Etiology: Diarrhea, excess vomiting, alcoholism, malnutrition, acute medical illness, primary or secondary aldosteronism, excess intake of licorice, glucose infusion, diuretics, adrenergic agonists, corticosteroids, insulin [29].

ECG Changes in Decreasing Order of Frequency are: ECG Changes when K+ < 2.7 mmol/l

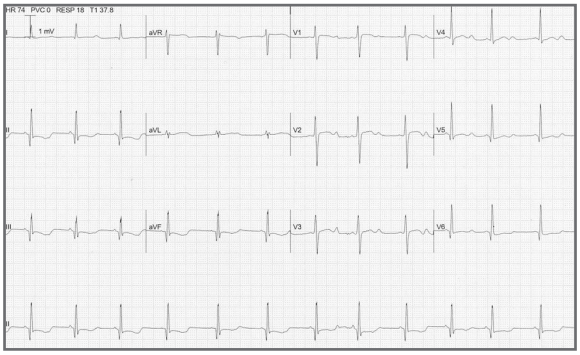
- Prolongation of the QRS duration increased P wave amplitude and duration.
- Prominent U waves (best seen in the precordial leads)
- Apparent long QT interval due to a fusion of the T and U waves (= long QU interval)
- ST segment depression decreased T wave amplitude [16]. With worsening hypokalaemia
- Frequent supraventricular, ventricular ectopic and heart blocks.
- Supraventricular tachyarrhythmia: AF, atrial flutter, atrial tachycardia
- Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes [24].



T wave inversion and prominent U waves in hypokalaemia



Long QU interval in hypokalaemia


ST depression, T wave inversion, Prominent U waves and Long QU interval.

Hypokalaemia may cause acquired long QT syndrome (LQTS) and predisposes to torsade de Pointes (polymorphic ventricular tachycardia). Hypokalaemia may also cause monomorphic ventricular tachycardia. Hypokalaemia potentiates the proarrhythmic effects of digoxin [30].

Magnesium: After potassium, magnesium is the most abundant cation (positively charged ion) in intracellular fluid and performs many important bodily functions. For example, it promotes enzyme reactions within the cell during carbohydrate metabolism [3].

- The normal **range** for **blood magnesium** level is 1.7 to 2.2 **mg/dL**

Hypermagnesaemia is rare, but severe hypermagnesaemia may cause atrioventricular and intraventricular conduction disturbances, which may culminate in third-degree (Complete) AV block or asystole.

Hypomagnaesemia may potentiate the pro-arrhythmic effect of digoxin. Hypomagnasemia may also predispose to supraventricular and ventricular tachyarrhythmias [31].

- **Hypomagnesemia:** is defined as a serum magnesium level of less than 1.7 mg/dL [3].

In hypomagnesaemia, there is flattening of the T waves, ST segment depression, prominent U waves and, occasionally, a prolonged P-R interval occurs. In hypermagnesaemia, there may be a prolonged P-R interval and widened QRS complexes [25].

- ECG changes of hypomagnesaemia resemble that of hypokalaemia.
- ECG changes of hypermagnesaemia resemble that of hyperkalaemia.
- Hypokalaemia, hypomagnesaemia and hypercalcaemia aggravate digitalis toxicity [16].

Implications for Clinical Practice

One of the most significant implications is the importance of considering electrolyte imbalances as a potential cause of ECG abnormalities. Clinicians should be aware that even mild fluctuations in electrolyte levels can lead to ECG changes that mimic other cardiac conditions. As a result, these imbalances should not be overlooked or dismissed as minor issues but rather should be thoroughly evaluated and appropriately addressed.

Furthermore, these findings highlight the need to regularly monitor electrolyte levels in patients with known cardiac conditions or those at risk for electrolyte imbalances. Ensuring optimal electrolyte balance is essential for proper heart functioning, and maintaining these levels within a narrow range can help prevent cardiac arrhythmias and other adverse cardiac events. Regular monitoring, therefore, is crucial for timely intervention and management of electrolyte imbalances.

Another implication is the importance of healthcare providers being well-versed in the interpretation of ECGs and able to recognize the subtle changes associated with electrolyte imbalances. Knowledge of the typical ECG patterns associated with specific electrolyte abnormalities can aid in early identification, prompt treatment, and, ultimately, better patient outcomes. Additionally, emphasizes the need for continued education and training for healthcare professionals in this area, as the accurate interpretation of ECGs is a skill that requires ongoing practice and updating [32].

Conclusion

Electrolyte imbalances can lead to significant changes in electrocardiogram morphology, potentially detrimental to cardiac function. These changes can serve as important clinical indicators, highlighting the need for careful monitoring and prompt intervention to restore electrolyte balance. Additionally, understanding the specific electrocardiogram patterns associated with different electrolyte imbalances can aid in identifying and managing these conditions, ultimately improving patient outcomes. Further research is needed to fully elucidate the underlying mechanisms and develop targeted therapeutic interventions to prevent and manage electrolyte imbalances to mitigate their impact on cardiac health.

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