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Electrolyte Abnormalities on ECG

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Abnormalities in the levels of certain serum electrolytes in the body can cause characteristic changes or an ECG. The myocytes in the heart depend on shifting gradients of these ions for depolarization and repolarization, which in turn leads to contraction and relaxation of the myocardium. Changes in extracellular calcium and potassium levels affect these gradients.[1] Not all patients with abnormalities in their serum electrolyte levels exhibit these changes.

Hypocalcemia is associated with a prolonged QT interval on ECG. Causes of hypocalcemia include primary or secondary hypoparathyroidism, vitamin D deficiency, and chronic kidney disease (CKD). Prolongation of the QT interval may trigger arrhythmias such as *Torsades de pointes*. It is important to note that the QT interval should be corrected for heart rate and is known as the QT_c (**Figure 1**). Although no specific symptoms are associated with QT prolongation, hypocalcemia can cause muscle tetany and hyperactive tendon reflexes. Hypocalcemia is treated with replacement of the calcium.

Hypercalcemia, on the other hand, causes a shortening of the QT interval. Unlike hypocalcemia, exces calcium is not associated with arrhythmias and is generally well tolerated by the heart. Causes of hypercalcemia include malignancy, CKD, and adrenal insufficiency. Treatment may include administration of bisphosphonate medications or glucocorticoids.

Hypokalemia is associated with a series of characteristic ECG changes involving the T wave. The earliest change causes a flattening of the T wave. As potassium levels continue to decrease, the ST segment starts to depress and can invert the T wave. Lastly, with severely decreased potassium levels, a characteristic U wave may appear. The U wave is a wide, positive wave that occurs after the T wave and before the next P wave (**Figure 2**). The levels at which these changes can occur vary from patient t patient, but one or all of these changes occur in up to 80% of patients.[2] Muscle weakness may occur

with very low potassium levels, especially if the change occurs rapidly.[3] Some common causes of hypokalemia are diuretic medications, vomiting, and diarrhea. Hypokalemia is treated with potassium replacement.

Hyperkalemia is also associated with characteristic ECG changes. The earliest and most common change that occurs is a peaking of the T waves, during which they become tall and appear tentlike. This change may be confused with the changes that occur in myocardial ischemia, which we will discuss in next month's installment of this department. During hyperkalemia, the P waves may also flatten and become absent. Lastly, with more severe elevations (usually >7.0 mg/dL), the QRS widens and fuses with the T wave, creating a characteristic rhythm known as the sine wave pattern (**Figure 3**). Untreated hyperkalemia may lead to AV block, asystole, and ventricular fibrillation.[4] In addition to the ECG changes, patients may experience muscle weakness or paralysis that can mimic Guillain-Barre syndrome.[5] Causes of hyperkalemia include medications such as ACE inhibitors and potassium sparing diuretics, renal failure, and hypoaldosteronism.

ECG CHALLENGE

Below is an ECG from a 51-year-old male with end-stage renal disease who presented to the emergency department after missing his dialysis twice in 1 week (**Figure 4**). He reports generalized fatigue.

Using the stepwise approach to analyze the ECG, we observe the following:

1. Is the ECG **regular**? No. The QRS complexes march out irregularly, and the P waves are flat or absent. You can see the absent P waves best towards the middle of the rhythm strip, on the bottom of the ECG.

2. What is the **rate**? Find a QRS complex on or near a dark line. A) Counting the large boxes, we see that there are between 2 1/2 and 4 large boxes before the next QRS complex because the rhythm is irregular. This method is unreliable here. B) There are about 9 QRS complexes in 6 seconds (30 large boxes), which estimates the rate at 9 x 10 = 90 bpm. In an irregular rhythm like this one, this method works best. C) As stated above, this method does not work well for this irregular rhythm.

3. There is **not a P wave** for every QRS, as they are mostly absent.

4. The **PR interval** cannot be determined here.

5. The **QRS complex** is about four to five small boxes, which is 0.16-0.20 seconds. This is a widened QRS complex, which could suggest bundle branch block, non-specific intraventricular conduction block, or an electrolyte abnormality such as hyperkalemia.

6. There is **no ST segment** elevation here. This may be difficult to see; however if you look at the J point (the point at which the ST segment leaves the QRS complex), it is not elevated. The T waves are markedly peaked, which is consistent with hyperkalemia or early changes of myocardial ischemia.

7. There are **no U waves**.

This patient exhibited ECG changes of a wide complex irregular rhythm with markedly peaked T waves. The patient did have additional laboratory studies performed, most importantly a basic metaboli panel, which showed a potassium level of 6.9 mg/dL. He also had negative cardiac enzymes. The patient was treated with kayexalate to help excrete the potassium through the GI tract as well as insulin plus glucose to drive potassium back into the cells. Following these therapies, the patient's ECG returned to a normal sinus rhythm.

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