CASE REPORT

ECG Manifestations of Multiple Electrolyte Imbalance: Peaked T Wave to P Wave (“Tee-Pee Sign”)

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The surface electrocardiogram (ECG) is a useful instrument in the detection of metabolic disturbances. The accurate characterization of these disturbances, however, may be considerably more difficult when more than one metabolic abnormality is present in the same individual. While “classic” ECG presentations of common electrolyte disturbances are well described, multiple electrolyte disturbances occurring simultaneously may generate ECG abnormalities that are not as readily recognizable. We report a case of hyperkalemia, with concurrent hypocalcemia and hypomagnesemia resulting in (1) peaking of the T wave, (2) a prominent U wave, and (3) prolongation of the descending limb of the T wave such that it overlapped with the next P wave. In this particular ECG from a patient with combined electrolyte imbalance, we have dubbed the unusual appearance of the segment between the peak of the T wave to the next P wave as the “tee-pee” sign.

Ann Noninvasive Electrocardiol 2009;14(2):211–214

multiple electrolyte imbalance; “tee-pee” sign

The cardiac action potential is generated by movement of ions across the cardiac cell membrane. Alterations in the normal levels of these ions may lead to altered electrical activity. These changes may manifest as abnormalities in the surface electrocardiogram (ECG) and therefore serve as a useful instrument in the detection of metabolic disturbances. Some characteristic patterns, such as the peaked T waves seen with hyperkalemia, for example, are virtually diagnostic.¹ The severity of electrolyte imbalances can also, on some occasions, be estimated on the basis of the ECG changes. However, in the presence of *multiple* electrolyte disturbances, the ECG signal may be significantly distorted, making both the diagnosis of the metabolic disorder(s) and the estimation of severity of the metabolic disturbance(s) more difficult.

CASE REPORT

A 62-year-old man with a history of chronic lymphocytic leukemia (CLL) presented to the Emergency Department complaining of a 4-day history of vomiting and abdominal pain. He had recently been treated with prednisone for warm autoimmune hemolytic anemia. On examination his blood pressure was 94/62 mmHg, his heart rate was 111 beats/min, and his respiratory rate was 22/min. His temperature was 37.9°C and he required a FIO₂ of 60% to maintain a SaO₂ greater than 92%. His abdomen was tender, distended, and the skin was mottled. Computed tomography of his abdomen revealed Richter's transformation of his CLL. He was admitted to the intensive care unit for management of sepsis and initiation of chemotherapy.

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Figure 1. Twelve-lead ECG showing peaked T waves most prominent in the precordial leads. The QT interval is difficult to discern because of prominent U waves.

A 12-lead ECG showed normal sinus rhythm at 80 beats/min (Fig. 1). There were peaked T waves in the precordial leads. The appearance of the T waves was unusual with a prolongation of the descending limb of the T wave, merging into the next P wave. Part of this prolongation was due to a prominent U wave.

Serum electrolytes confirmed the diagnosis of hyperkalemia with a potassium level of 6.7 mmol/L (normal range 3.5–5.2 mmol/L). Concurrently, the patient was found to have severe hypocalcemia with a calcium level of 1.46 mmol/L (normal range 2.15–2.65 mmol/L). Also noted was a critically low magnesium level of 0.35 mmol/L (normal range 0.8–1.0 mmol/L).

The patient was treated with fluid resuscitation, intravenous regular insulin, kayexalate per rectum, calcium gluconate intravenously, and magnesium sulfate, intravenously. Following correction of his electrolytes, the T waves normalized in less than 12 hours.

DISCUSSION

Peaked T Waves

The classic descriptions of hyperkalemia and hypocalcemia are listed in Table 1. Tall, narrow,

and peaked T waves are the earliest ECG sign of hyperkalemia. These changes are often seen when the serum potassium exceeds 5.5 mEq/L.^{1,2} The corrected QT interval is either normal or shortened. In the case presented here, the serum potassium was found to be significantly elevated and serves to explain the peaked T waves that normalized upon correction of the hyperkalemia. However, we note that the QT initially appeared prolonged and difficult to quantify because of a prominent U wave—findings distinctly unlike those typical of hyperkalemia. To determine the QT interval in this case, we used the slope intercept technique to identify the end of the T wave as the intercept of an isoelectric level and a line tangential to the point of maximum T-wave slope.^{3,4} When measured to the point at which this tangent crossed the isoelectric line, the QT interval was found to be within normal limits (Fig. 2).

U Wave

Following the initial steeply sloped portion of the T wave, a U wave is apparent (Fig. 2, arrow). In chronic magnesium deficiency, the ECG often resembles that of hypokalemia, showing ST depression, prolongation of the QRS duration, and prominent U waves.^{5,6} Hypocalcemia and hyperkalemia

Table 1. Most Frequent Single Ion Disturbances and ECG Manifestations

Single Ion Disturbances	Expected ECG Abnormality
Mild hyperkalemia (5.5–6.5 mmol/L)	Tall “peaked” T waves with narrow QT ¹
Moderate hyperkalemia (6.5–8.0 mmol/L)	Peaked T waves, prolonged PR interval, decreased amplitude of P waves, widening of QRS complex ¹
Severe hyperkalemia (> 8.0 mmol/L)	Absence of P waves, intraventricular blocks, widening of QRS complex, eventual “sine-wave” pattern, VF, asystole ¹
Hypocalcemia	Prolongation of QTc by lengthening of ST segment without change in T-wave duration ^{3,5}
Hypomagnesemia	Effects not clearly known Chronically: ST segment depression, flattening of T wave, prolongation of PR interval and QRS, prominent U waves ^{3,5,6}

ECG = electrocardiogram; VF = ventricular fibrillation.

are not associated with either prolongation of the T-wave duration nor with the presence of U waves. Thus, the U wave in this example is more compatible with the documented concomitant hypomagnesemia (0.35 mmol/L) (Table 2).

The “Tee-Pee Sign”

The ECG presented in this case has an additional interesting feature in the precordial leads occurring after the U wave. Following the U wave, we would

Figure 2. Lead V₄ amplified. Note the peaked T wave with normal duration and the prolongation of the segment from the peak of the T wave to the P wave of the next complex (includes the U wave). The tangent drawn along the slope of the initial portion of the T wave descending limb shows that the QT could be normal if the U wave is excluded. In the second beat, a depiction of a real tee-pee was superimposed on the T wave to show the similarities with the unusual morphology of the complex described above.

Table 2. Multiple Ion Disturbances and ECG Manifestations

Multi Ion Disturbances	ECG Abnormality
Hypocalcemia and hyperkalemia	Peaked T wave, prolonged ST segment.
Hypocalcemia, hyperkalemia, and hypomagnesemia	Peaked T wave, U wave, prolongation of the segment between the peak of the T wave and the next P wave ("tee-pee" sign).

ECG = electrocardiogram.

have expected the ECG signal to return to the iso-electric line. Instead, the descending limb of the T wave appears prolonged, and continues to merge into the subsequent P wave. We have proposed that this unusual "tenting" of the descending limb of the T wave be termed the "tee-pee sign" as its outline is reminiscent of the conical dwellings (teepees or tipis) made of animal skins or birch bark and constructed by Native North Americans of the Great Plains (Fig. 2). The term also refers to the unusual relationship between the T and P waves. This appearance has not been described previously in association with hyperkalemia or hypomagnesemia. Nor can it be attributed to the concomitant hypocalcemia. Usually, hypocalcemia manifests as prolongation of the QTc interval on the ECG.² However, the prolongation of the QT interval is in fact due to an increase in the duration of the ST segment, and not due to prolongation of the T-wave duration.^{3,5} Surawicz et al.⁵ have described that the QTc interval in hypocalcemia seldom exceeds 140% of normal; otherwise, an additional electrolyte abnormality is probably present and if a QT interval is

prolonged, it may in fact be a QU interval. Thus, the Tee-pee sign must be a manifestation of multiple electrolyte imbalances rather than a product of a single disturbance.

CONCLUSION

This case illustrates an interesting example of how multiple electrolyte abnormalities may modify various components of the ECG. The result in this case was an interesting combination of effects including peaked T waves, prominent U waves, and an unusual prolongation of the segment following the U wave ("tee-pee" sign). This appearance was attributed to a significant electrolyte disturbance consisting of concurrent hyperkalemia, hypocalcemia, and hypomagnesemia. Early recognition of this sign may contribute to rapid correction of electrolyte imbalance, preventing potential serious complications.

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