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An Unusual Presentation of Hypokalemic Ecg Mimicking as Ischemia

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ABSTRACT *Hypokalemia is common problem encountered in day to day practice. Symptoms seldom occurs unless the plasma K+ concentration is <3mmol/l.. Fatigue, myalgias and muscle weakness of lower extremities are common complaints. More severe hypokalemia leads to progressive weakness,hypoventilation due to respiratory muscle involvement and eventually ending in complete paralysis. We report a case of mild hypokalemia with ECG changes mimicking ischemia i.e., T-wave inversions from V1 to V6 without U waves and ST segment depression which got normalised after correcting hypokalemia.*

Introduction:
Despite common occurrence of hypokalemia with ECG changes like ST depression, U-waves, prolonged QT/QU interval, prolonged PR interval and T wave flattening/inversion, we present a case of global T-wave inversions in chest leads without U waves mimicking ischemia.

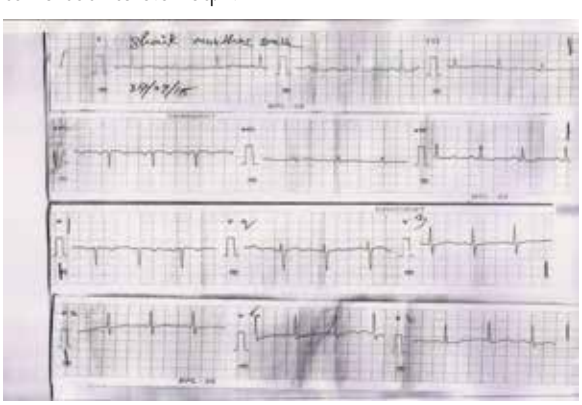
Case report:
48 yrs female brought to casuality at 2pm with complaints of weakness of both lower limbs, giddiness since morning. H/O present illness started as fever since 10 days, high grade, intermittent not associated with chills or rigors and had been diagnosed as enteric fever with widal positive and is on treatment. Now for the past 2 days patient had non bilious vomitings,5-6 episodes per day not subsiding on regular medication. Patient is known hypertensive since 3yrs and is regularly on Amlodipine 5mg. No other relevant history present.

Physical examination revealed power of both lower limbs to be 3/5, deep tendon reflexes: Right knee jerk – 0 and Left knee jerk +1,absent ankle jerks both sides. Power of upper limbs and upper DTR were normal.

Laboratory investigations showed: RBS was 122mg/dl , Serum sodium-134meq/L, Serum potassium-2.7 meq/L and Serum chloride was 94 meq/L.RFT'S and LFT'S were within normal limits. THYROID profile was normal, LIPID profile within normal range.

ECG: showed HR-75/min, normal PR interval, normal QRS complex voltage and interval, without ST segment depression, without prominent U waves and with pan T wave inversions in V1 to V6.ECHO showed mild concentric LVH other than that echo is normal without any regional wall motion abnormality. Next day 24hrs urine potassium showed 25.7mmol/l(with 25-125 normal reference interval), USG abdomen and CHEST XRAY PA VIEW showing normal findings. Diagnosed as hypokalemia with vomiting as etiological factor

Patient was treated with oral potassium chloride syrup and ringer lactate not going for i.v. potassium chloride drip and with increased coconut water intake and bananas. On 3rd day ECG changes normalized and serum potassium levels came back to 5.5meq/L.



Discussion:
Hypokalemia is one of the most commonly encountered fluid and electrolyte abnormalities in clinical medicine. It can be an asymptomatic finding identified only on routine electrolyte screening, or it can be associated with symptoms ranging from mild weakness to sudden .Patients receiving diuretics are at the highest risk, with as many as 50% developing serum potassium levels of less than 3.5 mEq/liter (1).ECG changes in hypokalemia are due to delayed ventricular repolarization .The earliest ECG change is decrease in the T-wave amplitude. As potassium levels decline further, ST segment depression and T-wave inversions are seen, while the PR interval can be prolonged along with an increase in amplitude of P wave. The U wave is described as a positive deflection after the T wave, often best seen in mid- precordial leads (V2 and V3). When the U wave exceeds the T-wave amplitude, the serum potassium level is < 3 mEq/L. In severe hypokalemia (Serum K+ <2.5) T- and U-wave fusion occurs with giant U waves

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masking the smaller preceding T waves becomes apparent on the ECG. With severe hypokalemia (Serum K+ < 2.5) Frequent supraventricular and ventricular ectopics occurs. Supraventricular tachyarrhythmias: AF, atrial flutter, atrial tachycardia. Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes.

Here ECG shows global T-wave inversions in all chest leads without any ST segment depressions,without change in the amplitude of P wave and without any prominent U waves and without supraventricular or ventricular ectopics.

The symptoms of hypokalemia are nonspecific and predominantly are related to muscular or cardiac function. Weakness and fatigue are the most common complaints. The muscular weakness that occurs with hypokalemia can manifest in different ways (eg, dyspnea, constipation or abdominal distention, exercise intolerance). Rarely, muscle weakness progresses to frank paralysis. With severe hypokalemia or total body potassium deficits, muscle cramps and pain can occur with rhabdomyolysis.

Occasionally, a patient may complain of worsening diabetes control or polyuria due to a recent onset of hyperglycemia or nephrogenic diabetes insipidus. Patients also may complain of palpitations. Psychological symptoms may include psychosis, delirium, hallucinations, and depression. Patients are often asymptomatic, particularly with mild hypokalemia. Symptoms that are present are often from the underlying cause of the hypokalemia rather than the hypokalemia itself.

There are so many causes for hypokalemia which were broadly divided into two main groups: renal K+ losses and gastrointestinal losses, others are through redistribution into cells and decreased dietary intake. Redistribution into cells- alkalosis, insulin excess(2), catecholamine , Beta2-agonists ,dopamine, dobutamine, theophylline (2,3) ,hypokalemic periodic paralysis.

Gastrointestinal losses (urine K<20-30mmol/day)-with alkalosis: vomiting, nasogastric aspiration, with acidosis: diarrhea, laxative abuse, villous adenoma of rectum, bowel obstruction/fistula, uterosigmoidostomy. Renal losses(urine K>20-30 mmol/day)-with hypertension: primaryhyperaldosteronism- Conns syndrome, secondary aldosteronism -renal ischemia, other forms of mineralocorticoid receptor activation- Cushings syndrome/ectopic ACTH , corticosteroid therapy, liquorice/ carbenoxolone ,apparent mineralocorticoid excess ,Liddle syndrome

With normal blood pressure: with alkalosis-diuretic therapy (loop and thiazide), Barters syndrome (4) and Gitelmans syndrome(5), with acidosis-renal tubular acidosis type 1&2,carbonic anhydrase inhibitor therapy. With variable pH-post-obstructive diuresis , recovery after acute tubular necrosis, Magnesium depletion.

Conclusion:
Eventhough T wave changes like flattening /inversions are common in hypokalemia pan T wave inversions in all chest leads from V1 to V6 mimicking ischemia without ST segment depression and U waves is unusual finding and hypokalemia should be considered in such conditions.

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