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Can Hyperkalemia Masquerade Acute Myocardial Infarction - The Pseudoinfarction Pattern

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Abstract: ***Hyperkalemia which is defined as serum potassium level more than 5.5 meq/L is a dangerous life threatening metabolic abnormality which needs an aggressive treatment to reduce the mortality to certain extent. Herewith we discuss an interesting case of a male in his 50s who presented to the ER with acute flaccid paralysis and ECG showing a pseudoinfarction pattern.***

Keywords: Hyperkalemia, sine wave, ventricular fibrillation

1. Introduction

Hyperkalemia is caused because of several pathophysiological mechanisms. It includes increased intake, excessive endogenous potassium load, redistribution of potassium from intracellular to extracellular fluid, decreased excretion by the kidneys. The hospital mortality is high in patients with hyperkalemia as the frequency of life threatening arrhythmias is very high among hyperkalemic patients. The prevalence of hyperkalemia is also increasing due to the increased use of ACE inhibitors, potassium sparing diuretics and beta blockers. ECG is one of the most important tools to detect hyperkalemia. The changes is ECG pattern also helps to know the level of hyperkalemia and to asses the severity. However Hyperkalemia presenting with ST elevation (pseudoinfarction pattern) must be differentiated from acute myocardial infarction.

2. Case Report

A 50 year old male who is a farmer by occupation, non smoker, non alcoholic with no known comorbidities presented to the ER with complains of weakness of all 4 limbs for 4 hours from the time of admission. The weakness initially started in the lower limbs then progressed to the upper limb. There was no history suggestive of cranial nerve disturbances. There was no history of any sensory disturbances, preceding headache, loss of consciousness, chest pain, breathlessness or palpitation. No fever, vomiting or diarrhea. No past history of any significant drug intake, cervical cord injury or bowel bladder disturbances. On examination his vitals were stable. He was normotensive and there was no resting tachycardia with an oxygen saturation of 98% in room air. The patient was concious, oriented, afebrile. Clinical examination of cardiovascular and respiratory system showed no abnormality. Abdomen was soft. Central nervous system examination revealed no abnormality in speech, memory, language or attention span. His tone was decreased in all 4 limbs. Power was found to be 3/5 in both upper limbs and 1/5 in both lower limbs. His deep tendon reflexes were normal. His bilateral Plantar reflexes were mute. All other superficial reflexes were normal. Bilateral pupils were normal and equally reacting and No nystagmus. His cranial nerve examination showed no cranial nerve dysfunction. Sensory examination showed no abnormality to pain, touch, pressure, vibration and position sense. Cerebellum function tests were not performed as the patient was not able to stand. There was no spinal tenderness.

So a provisional diagnosis of acute flaccid paralysis was made with differential diagnosis of Hypokalemic/ Hyperkalemic paralysis, Acute demyelinating polyneuropathy, heavy metal poisoning, Myasthenia gravis and Botulism.

His blood investigation showed normal Complete blood count, renal function test, liver function test and Thyroid function test. HisSr. potassiumwas 9.6meq/L. Sr. magnesium - 2.4meq/L, Sr. calcium - 11.1meq/L, Sr. phosphate - 3.0meq/L

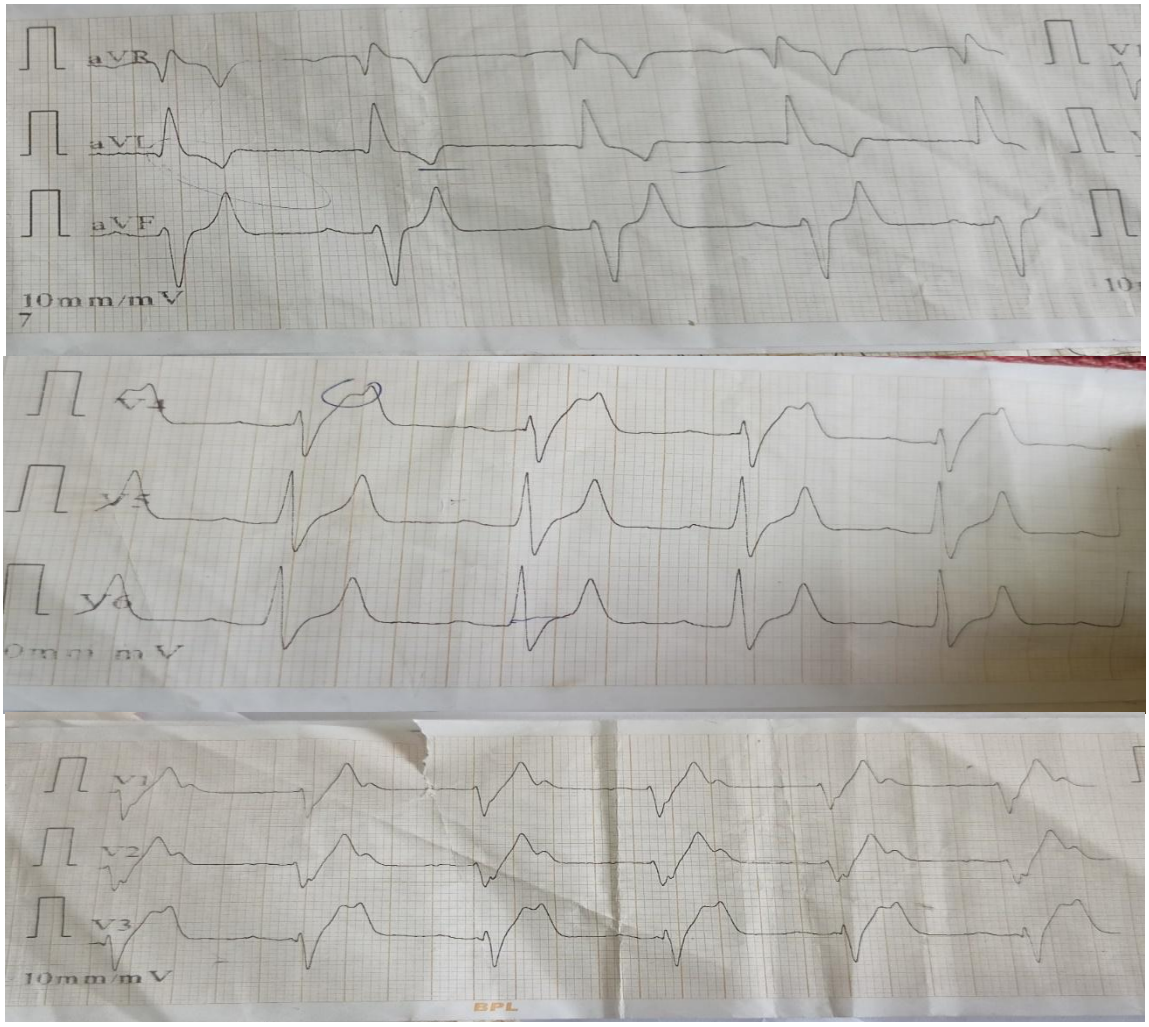

Figure 1: ECG at the time of admission

His ECG at the time of presentation showed HR - 68/min, flattening of P waves, widening of QRS complex and ST segment elevation in V1 - V4 leads. The initial ECG was suggestive of hyperkalemia.

Because of doubtful ST elevation in ECG the cardiac enzymes were taken and it showed CKMB was 23 U/L, and cardiac troponin was negative. Bed side ECHO revealed no regional wall motion abnormality. His serum cortisol level was also within normal limits, ABG showed pH - 7.3, HCO3 - 14.1; PCO2 - 27.4 (metabolic acidosis), urine potassium was 22.4meq/L. (15 - 25meq/L).

He was immediately treated with 3 doses of calcium gluconate (10ml of 10% calcium gluconate ivover 3 mins) under continuous cardiac monitoring. His ECG showed no changes. He was also started on repeated cycles of insulin dextrose buffer (10 unit of human actrapid in 100 ml of 25%dexrose). Simultaneously he was also started on salbutamol nebulization. After 2 hours of initial resuscitation his serum potassium level of 7.2meq/L.

After 4 hours of resuscitation, the patient complained of chest pain and his ECG showed HR - 70/min, Tall T waves in V1 - V4, ST elevation in V1 - V6 and reciprocal ST depression in aVL.

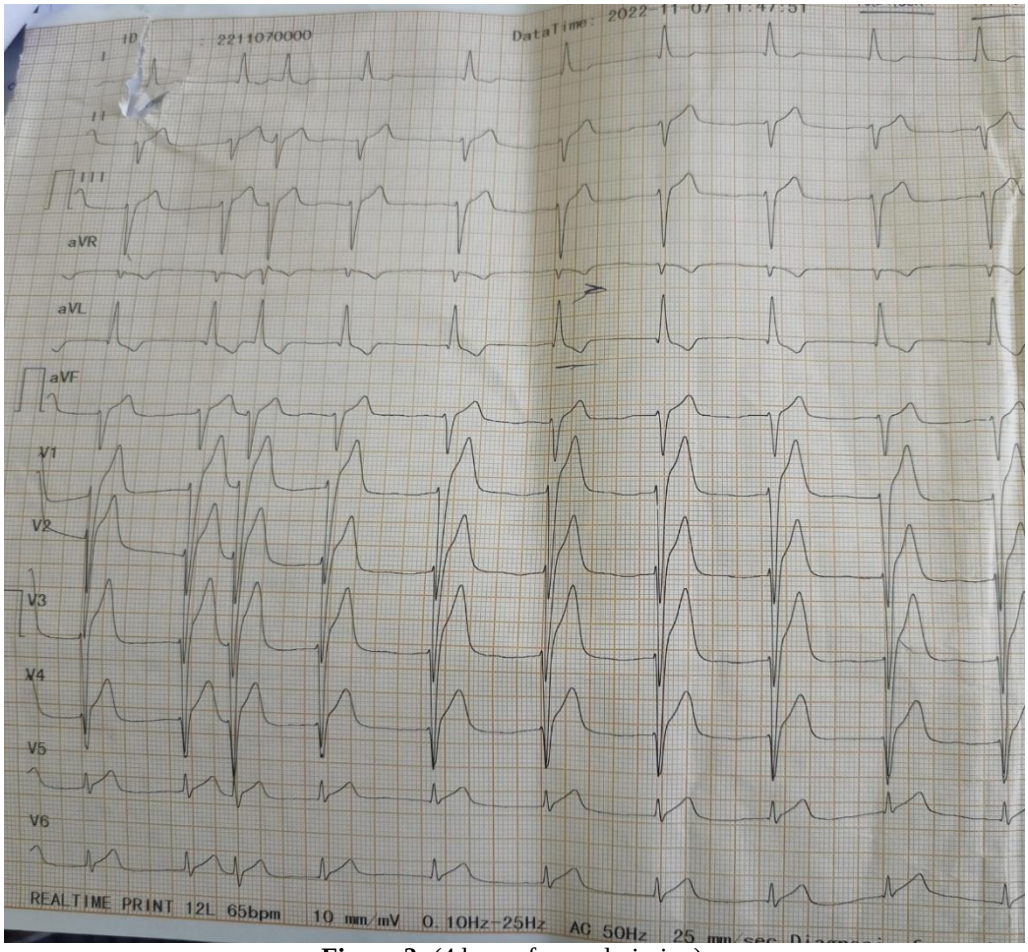

Figure 2: (4 hours from admission)

CKMB after 4 hours of admission was 23U/L. The patient was on repeated cycles of salbutamol, insulin dextrose and potassium binding resins. After 12 hours the patient suddenly went in to Ventricular tachycardia. This was the ECG.

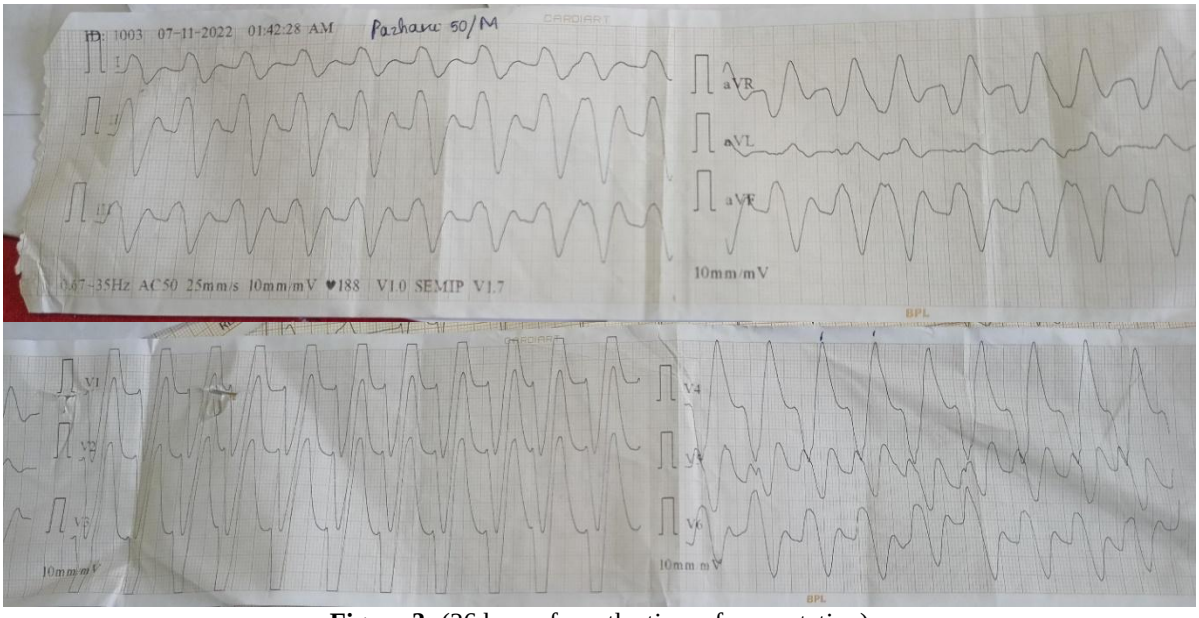

Figure 3: (26 hours from the time of presentation)

The patient was revived using DC shock and he was put on mechanical ventilation. The patient went into cardiogenic shock to which he was started on dual inotropes. Though the patient was treated with all the measures to reduce hyperkalemia, he went into repeated Ventricular tachycardia, despite of all adequate resuscitation, he could not be revived.

3. Discussion

Pseudoinfarction in Hyperkalemia:

Sometimes the ECG changes of hyperkalemia can mimic acute ST elevation myocardial infarction. This must be differentiated from serial ECG monitoring in which

hyperkalemic ECG changes don't follow the typical MI ECG pattern [1]. Hyperkalemia can produce several characteristic electrocardiographic (ECG) changes. It rarely produces ST - segment elevation simulating an acute myocardial infarction (AMI) [2]. "Pseudo - infarction" is a rare manifestation of hyperkalemia; with very few cases documented in the literature until date.

The mechanism is related to shortening phase 3 repolarization where potassium efflux is the predominant ionic shift. A potassium current channel located on the cell membrane is responsible for most of the potassium efflux during phase 2 and phase 3 of cardiac action potential and increases potassium efflux when extracellular potassium is higher [3]. This leads to shortening of phase 2 and phase 3 of action potential in the setting of Hyperkalemia and therefore shortening of repolarization.

The pathological Q waves as well as ST/T changes resembling acute injury has been attributed to the altered QRS forces secondary to the interventricular conduction abnormality due to hyperkalemia. Kyunhunet al have reported 4 cases of acute renal failure with hyperkalemia and ST/T changes mimicking myocardial infarction, but in all these patients lowering the serum potassium levels by hemodialysis was associated with the return of ECG changes to normal. However invasive coronary angiography in these patients who mimic myocardial infarction can be done to rule out any coronary artery stenosis. Aproximately 28 cases of Hyperkalemia mimicking acute myocardial infarction have been reported so far [4]. one review stated approximately 80% of cases have anteriseptalpseudo in farction pattern with q waves present in V1&V2 and ST elevation in avR [5]. Pattern of isolated inferior as well as anterolateral wall MI pattern have also been reported

In summary we describe the presentation of a gentleman who presented with acute flaccid paralysis secondary to malignant Hyperkalemia whose ECG changes mimicked acute anterior myocardial Infarction which was ruled out by bedside Echo and serial cardiac enzyme monitoring. However the definite exclusion of coronary occlusion can be ruled out only by invasive angiography which was not done in this patient owing to his serious illness.

On presentation he has electrocardiographic evidence of an acute myocardial infarction which clouded recognition of Hyperkalemia and delayed the necessary emergency treatment. This Hyperkalemic pseudo infarction is rare overall. However awareness about this rare presentation of a common electrolyte abnormality may help expedite recognition and treatment of a life threatening disorder.

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