

SEVERE HYPERKALEMIA PRESENTING AS BRADYCARDIA WITH FEATURES SUSPICIOUS FOR AN UNDERLYING MYOCARDIAL INFARCTION: A NEAR-MISS GREAT MIMICKER

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CASE PRESENTATION

A 64 years old man known with dislipidemia, hypertension, diabetes mellitus and cardiomyopathy presented to the emergency department (ED) following a fall due to postural dizziness. In the review of systems he reported a history of retrosternal chest discomfort without classical radiation as seen in acute coronary events. He had also a persistent epigastric discomfort without nausea or vomiting.

He had been closely attending to scheduled follow up visits with his primary care provider (PCP) and he was stable on medications that included captopril, hydrochlorothiazide, aldactone and oral hypoglycemics (glimepiride and metformin). A small dose of carvedilol had been newly added at his most recent visit to his PCP.

His evaluation at the ED revealed a restless and disorientated man with central obesity. He had a flapping tremor with no lateralizing signs. His vital signs were recorded as BP 100/34 mmHg, HR 38 - 44, RR 19, SpO₂

94% RA, Temperature 36.7 degrees Celsius and a random blood glucose of 6.3 mmol/L (dextrostick on middle finger). He was noted to have a variable bradycardia that "somewhat" initially improved with supplemental oxygen via a face mask. His cardiovascular assessment revealed a pericardial rub and few bi-basal crackles without other signs of overt fluid overload. While in the ED, his systolic blood pressure remained stable at baseline.

A bedside echocardiogram showed a global hypokinesis (no isolated regional wall motion abnormality) with a LVEF 25 - 30%, moderate mitral regurgitation, small tricuspid regurgitation and a small pericardial effusion of 1.1 cm in anterior diameter.

The abdominal ultrasound revealed small sized kidneys with poor cortico-medullary differentiation. There was no features of hydronephrosis.

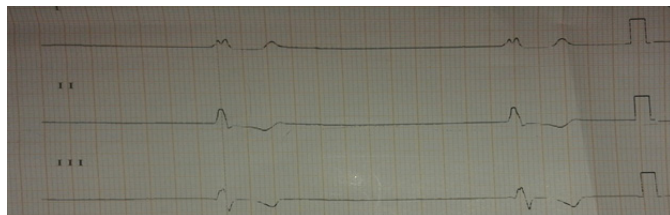


Figure 1: Electrocardiogram at presentation in ED

Severe bradycardia, at 25 bpm, wide complex QRS rhythm as shown on the leads I, II and III. A comprehensive review of the other leads showed an ST depression in II, III and aVF, a subtle LBBB pattern in V5 and V6, and tall teinted T waves seen in some precordial leads (Figure 1).

The initial assessment and history of illness were concerning for the possibility of acute myocardial infarction (right coronary artery vascular territory) leading to arrhythmia. The main differential diagnosis was thought to be severe renal failure leading to overt uremic state (pericarditis and encephalopathy) and hyperkalemic ECG changes.

The patient was admitted to the high dependence unit for commencement of acute MI protocol and continuous monitoring pending to obtain his preliminary laboratory work-up results including a full electrolytes' panel. While on the HDU he suffered a cardiac arrest that was completely reversed with a timely resuscitation.

In the following few hours his laboratory work-up revealed a normal serum blood glucose level, and the two-sets of cardiac enzymes (four hours apart) were negative. His chemistry panel was significant for serum potassium of 8.6 mmol/L with a GFR of 13 mls/min and a severe metabolic acidosis.

In normal ED working routine, a preliminary acid-base balance and electrolytes' panel is run on the Arterial Blood Gas analysis machine which was unfortunately broken at the time the patient came in, hence the delay in diagnosing hyperkalemia until the patient suffered from cardiac arrest.

Immediate measures included medical management of hyperkalemia with myocardial instability and preparations for urgent hemodialysis. The electrocardiography results showed: wide complex rhythm, at 66 bpm, first degree AVB and mild ST depression is still noted in the rhythm lead II (Figure 2), and Narrow complex and regular rhythm at 60 bpm, first degree AVB, features of left ventricular hypertrophy (voltage criteria) and repolarization abnormality (Figure 3).

The patient was discharged in a stable condition to continue his renal replacement therapy (hemodialysis) in ambulatory setting.

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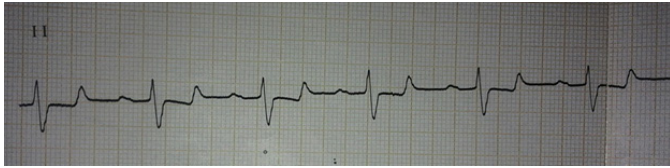


Figure 2: Electrocardiogram after repetitive K-shifting medications and cardiomyocytes' membrane stabilizer

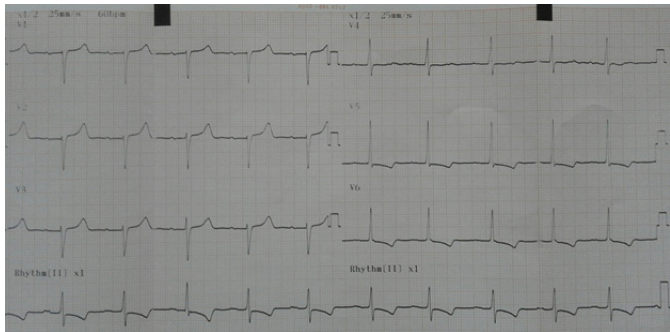


Figure 3: Electrocardiogram after 3 sessions of hemodialysis and normalization of serum potassium levels

DISCUSSION

Among all the serum electrolytes, potassium is known to play a big role in action potential of the myocardium (Sah et al., 2003). The abnormal serum potassium levels (either high or low) often lead to myocardial instability and fatal arrhythmias. The classical manifestation of hyperkalemia on electrocardiogram include the tall tained T waves, flattening of the P waves, widening of the QRS complex and the typical sine wave that can be seen in extreme high levels of serum potassium (Parham, Mehdirdar, Biermann, & Fredman, 2006).

The pseudo-infarction pattern and conduction abnormalities or blocks at any level (sinal, AV junction, ventricular or bundle branch) have been also cited in many reports, and they should be recognized as potential manifestations of abnormal levels of serum potassium (Bellazzini & Meyer, 2010).

The treatment of serum potassium abnormality is a medical emergency that if left untreated or recognized too late, it can lead to severe arrhythmias and death (Medford-Davis & Rafique, 2014).

The modalities of treatment are based on reducing the potassium intake (strict K-deprived diet), impairing its absorption with oral binding resins i.e kayexelate salts, reducing the circulating levels i.e potassium shifting from bloodstream to the intracellular compartment, and promoting its elimination from the body (i.e using loop diuretics or hemodialysis).

The widely available treatment in our emergent settings includes immediate measures that shift the potassium from blood circulation to the intracellular compartment such as nebulization with beta-agonists i.e salbutamol and/or intravenous infusion of dextrose 50% mixed with soluble insulin. All the medications that potentially contribute to potassium retention in the body such as aldactone should promptly be discontinued.

The administration of calcium gluconate is indicated in those patients with electrocardiographic changes and it is repeated as needed to stabilize the myocardium (Mushiyakh et al., 2012).

It is encouraged to obtain a nephrology consult as soon as possible, as most cases that are refractory to medical treatment aiming at lowering lethal levels of serum potassium would end up requiring urgent hemodialysis. Occasionally, especially in those patients with a metabolic acidosis, the administration of oral or intravenous sodium bicarbonate might still be used to buffer the severely acidic internal milieu (Sterns, Grieff, & Bernstein, 2016).

CLINICAL RELEVANCE AND RECOMMENDATION

This case highlights the need for proper channeling of patients with grossly abnormal vital signs including severe bradycardia under investigation and re-emphasizes on the crucial need for multidisciplinary approach in the management of such complex cases. It also bring about the recognition of a fully functioning laboratory that timely report on urgent tests such as electrolytes' panel, as an irreplaceable tool for the clinician that handles patients who are likely to decompensate in between evaluation steps.

Would this case present to a facility unequipped with highly monitored wards or those whereby the laboratory testing capacity is still a logistical nightmare, it is very likely that the outcome would have been quickly fatal.

With the ongoing roll up of medical equipments across the district referral and provincial hospitals, we recommend to all the medical personnel to get more familiar with electrocardiography; a tool that is often thought to be utilized only by the cardiologist. It can often provide hints on background diseases that constitute the reason of visiting the emergency department, and if abnormal, might have immediate therapeutic implications that are indeed life-saving.

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