Transitnt Trifascicular Block in Severe Hyperkalemia

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Abstract

Hyperkalemia is a commonly encountered electrolyte abnormality that can significantly alter normal cardiac conduction. Potentially lethal dysrhythmias associated with hyperkalemia include complete heart block and Mobitz Type II second-degree AV block. We report a case of trifascicular block, due to hyperkalemia. The patient’s symptoms and electrocardiogram (ECG) evidence of trifascicular block resolved with lowering of serum potassium levels, with subsequent ECG showing left anterior hemiblock. This paper highlights an infrequently reported dysrhythmia associated with hyperkalemia that emergency physicians should be familiar with.

Introduction

Hyperkalemia is a life-threatening electrolyte abnormality requiring urgent management.¹ Serum potassium levels greater than 5.5 mmol/L is considered hyperkalemia. Hyperkalemia is often asymptomatic until plasma potassium concentration is above 6.5-7 mmol/L when it results in fatal arrhythmias, hence its called silent killer. Hyperkalemia is associated with significant disturbances in cardiac conduction, ranging from QT interval shortening, to PR interval lengthening and QRS widening.² Reversible fascicular blocks, as well as bundle branch blocks or intraventricular conduction delay can be seen. Moreover, hyperkalemia is known to cause potentially lethal dysrhythmias including ventricular tachycardia, ventricular fibrillation, idioventricular rhythms, and asystole.¹ ³ ⁵

Case Report

A 65 year old female was brought to Emergency Department of MLB Government Medical College, Jhansi, with complaints of retrosternal chest pain, diaphoresis and syncopal attacks. Patient was a known case of diabetes mellitus and systemic hypertension. Right nephrectomy had been performed 24 years back. Patient was not on any treatment at the time of presentation. Patient’s blood pressure was 166/92 mm of Hg and pulse rate 30/min. Patient was given injection atropine and was immediately transferred to Coronary Care Unit. On examination in Coronary Care Unit patient’s blood pressure was 160/90 mm of Hg, pulse rate 72/minute, cardiac auscultation revealed no finding and all other systems were within normal limit. Patient’s initial electrocardiogram showed heart rate of 78/min, PR interval of 255 millisecond, QRS duration of 146 millisecond and mean QRS axis to be 266°. ECG was interpreted as trifascicular block i.e. right bundle branch block (RBBB), left anterior fascicular block (LAFB) and first degree atrioventricular (AV) block. Patient’s Random Blood sugar was 350 mg%. Patient experienced two episodes of Stokes Adams attack following which isoprenaline drip was started. General management of acute coronary syndrome was also started. ABG showed metabolic acidosis with compensatory respiratory alkalosis and hyperkalemia (pH 7.349, bicarbonate 10.6 mmol/L, pCO₂ 19.2 mm of Hg, S. Na+ 131 mmol/L and S. K+ 7.2 mmol/L). Complete blood count showed TLC of 15200/mm³, hemoglobin of 6.2 g%, DLC P₃, Lₑ, and hematocrit of 20.5%. Serum creatinine was 4 mg% and blood urea 96.6 mg%. Anti-hyperkalemic measures (calcium gluconate, insulin, kayexalate, and albuterol nebulization) were urgently commenced and then patient underwent hemodialysis. Three units of blood were transfused. On second day her hyperkalemia and acidosis recovered (S. K⁺ 4.2 mmol/L, pH 7.5, pCO₂ 36.6 mm of Hg, HCO₃⁻ 28.9 mmol/L). Her ECG on second day showed heart rate 85/min, PR interval of 137 millisecond, QRS duration of 102 millisecond with mean QRS axis -53°. ECG was interpreted as LAFB. Thus RBBB and first degree AV block reversed after correction of hyperkalemia. To rule out coronary artery disease (CAD) CPK-MB and Troponin-I were done. CPK-MB was 1.2 IU/L and Troponin-I was 0.1 ng/ml (CPK-MB was normal and Troponin-I only slightly raised but in renal failure threshold is raised and upto 0.15 ng/ml is considered to be normal). 2-D echo was normal with LVEF 66%. She was discharged on day 5 of admission.

Discussion

This case demonstrates an atypical presentation of hyperkalemia-induced trifascicular block that resolved to LAFB with lowering of the serum potassium levels. Although electrophysiologic studies at the AV node and His-Purkinje system would be needed to confirm our theory that hyperkalemia induced the ECG pattern of trifascicular block in this patient, the absence of any other explanation, such as medication overdose, myopericarditis, rheumatic fever, or acute myocardial ischemia, suggests that hyperkalemia was the underlying etiology. The resolution of trifascicular block to LAFB with aggressive treatment of hyperkalemia also suggests that this electrolyte disturbance produced the higher-grade AV block.
The AV node is known to be susceptible to hyperkalemia, producing the classic prolonged PR interval and QRS widening seen often in the setting of hyperkalemia.

In our patient, in the absence of acute myocardial infarction, we propose that trifascicular block resulted due to the patient’s AV node and Purkinje fibres susceptibility to hyperkalemia.

Ohmae et al reported a case of RBBB with left axis deviation which resolved with correction of hyperkalemia. Bashour et al reported twelve patients who exhibited electrocardiographic evidence of fascicular block during hyperkalemia. Isolated left posterior hemiblock occurred in four, isolated left anterior hemiblock in two, right bundle branch block with left anterior hemiblock in two, right bundle branch block with left posterior hemiblock in one, left bundle branch block with abnormal left axis deviation in two and advanced atrioventricular block in one. In all seven patients with sinus rhythm the P-R interval shortened after correction of hyperkalemia. Electrophysiologic studies using His bundle recording and atrial pacing in one patient revealed intraatrial conduction delay and marked prolongation of conduction time in the His-Purkinje system. Weidner et al described a case of RBBB in hyperkalemia which reversed after correction of hyperkalemia.

In conclusion, hyperkalemia is a commonly encountered electrolyte abnormality that can produce life-threatening derangements in cardiac conduction. The ED physician should be aware of the range of dysrhythmias attributed to hyperkalemia—including trifascicular block—and should promptly correct hyperkalemia to minimize mortality and morbidity.

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References