

The Great Masquerader Complete Heart Block (CHB) With Crusader Hyperkalemia in an Elderly: The Lessons to Reckon

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Abstract: Cardiac impulse conduction involves the passage of impulse from sinoatrial(SA) node to atrioventricular(AV) node, and then through Bundle of His, Bundle branches, Purkinje fibers, and finally Ventricles. Potassium, an extracellular ion plays an important role in the electrophysiologic function of the myocardium. A high serum potassium level impairs the impulse conduction in purkinje fibers and ventricles more than that in the AV node. Therefore, although complete AV block (Complete Heart Block –CHB) can occur, it is a rare initial presentation. Here we present an elderly lady of 78 years with Diabetes mellitus and Hypertension comorbidities presenting with giddiness of 2 days duration and diagnosed to have CHB with hyperkalemia, which responded remarkably to conservative treatment. However post treatment electrocardiogram (ECG) depicted Right bundle branch block (RBBB) pattern and the puzzle for such finding was resolved. The present case thus offers a clear clinical message impacting the medical practice.

Keywords: Complete heart block (CHB), Complete AV block, Right bundle branch block (RBBB), Hyperkalemia.

1. Introduction

Potassium is an extracellular ion with an important role in the electrophysiological regulation of myocardial function. High intracellular & low extracellular potassium concentration is maintained by sodium potassium ATPase pumps, giving rise to a resting membrane potential of -90mV across the myocyte membranes. Any change in extracellular potassium concentration may have a significant effect on myocyte electro physiologic gain [1].

Hyperkalaemia is defined as a plasma potassium (K^+) level of 5.5 mEq/L. It occurs in up to 10% of hospitalized patients; severe hyperkalemia (>6.0 mEq/L) occurs in approximately 1%, with a significantly increased risk of mortality. Hyperkalemia is a medical emergency because of its effects on the heart. Cardiac arrhythmias associated with hyperkalemia include sinus bradycardia, sinus arrest, slow idioventricular rhythms, ventricular tachycardia, ventricular fibrillation, and asystole. Mild increases in extracellular potassium affect the repolarization phase of the cardiac action potential, resulting in changes in T-wave morphology; further increase in plasma K^+ concentration depresses intracardiac conduction, with progressive prolongation of the PR and QRS intervals. Severe hyperkalemia results in loss of the P wave and a progressive widening of the QRS complex; development of a sine-wave sinoventricular rhythm suggests impending ventricular fibrillation or asystole [2].

Hyperkalemia produces a gradual depression of the excitability, conduction velocity of the specialized pacemaker cells and conducting tissues throughout the heart. High serum potassium levels are thought to impair the conduction in the Purkinje fibers and ventricles more than in the AV node, although complete AV block (CHB) can occur [3].

Our present case highlights the occurrence of CHB even at mildly elevated serum potassium levels as initial presentation

and further highlights the diagnostic dilemma. Few therapeutic wisdom/pearls are forthcoming from the present case.

2. Methodology (Case Report)

The patient was a 78-year-old female who was admitted into our department with complaints of fatigue and episodes of lightheadedness of 2 days duration. She had a positive history of diabetes mellitus for the previous 10 years and systemic hypertension for 8 years. She also had a history of hospital admission previously for diabetic foot and was detected to have diabetic nephropathy, diabetic retinopathy. Insulin, Amlodipine, Telmisartan, Atenolol, Hydrochlorthiazide were the drugs that she used regularly. On present admission into the hospital, she had a respiratory rate of 16/min, a pulse rate of 40/min, a blood pressure of 160/90 mmHg, and her blood sugar was 180 mg/dL. Her ECG revealed complete heart block heart block (Fig 1). Laboratory examination revealed serum potassium of 6.4 mEq/L, and creatinine of 1.8 mg/dL



Figure 1: Complete Heart Block (on Admission).

Because of obvious Hyperkalemia and its apparent cardiac conductive effects, 10 mL of 10% calcium gluconate was administered immediately as it is known to protect cardiomyocytes. Infusion of 50% glucose with insulin was also instituted as it is the fast acting drug that shifts

potassium in to cells. Repeat ECG done showed Right bundle branch block (RBBB) pattern (Fig-2), even though serum potassium had returned to normal levels.

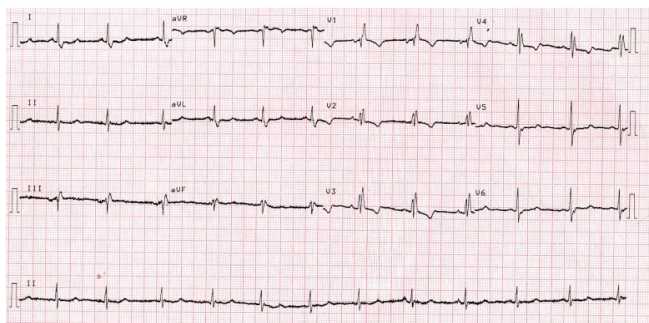


Figure 2: RBBB Pattern (Post Treatment)

Echo was done which was normal. She was managed in the ICU for 3 days with normal serum potassium levels on serial monitoring. Mean while patients ECG done several months prior to the present admission was traced which showed similar RBBB pattern (Fig 3).

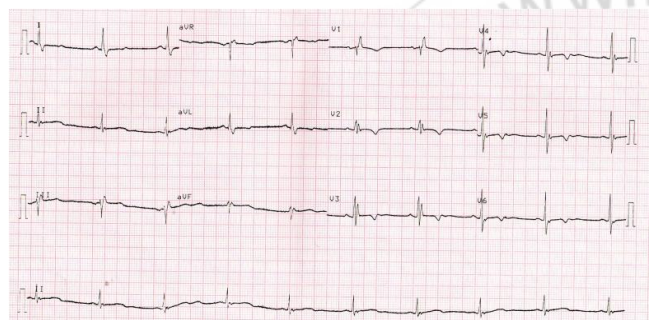


Figure 3: Old ECG Showing RBBB pattern(Traced later).

She was discharged 3 days later normokalemic. She was followed up for 2 months and she remained normokalemic without recurrence of complete heart block(CHB), although the right bundle branch block persisted.

3. Discussion

Hyperkalemia is a common acute life-threatening emergency. The usual causes include renal insufficiency or failure, drug-induced such as angiotensin converting enzyme inhibitors, or use of potassium sparing diuretics, insulin deficiency or resistance, and hemolysis[4]. Hyperkalemia is a common cause of cardiac arrhythmias seen in clinical practice. Electrocardiographic abnormalities noted in hyperkalemia can be summarized[4] as in Table 1.

Table 1:

Serum potassium level	Expected ECG abnormality
Mild hyperkalaemia 5.5 - 6.5 mmol/L	Tall peaked T waves with narrow base
Moderate hyperkalaemia 6.5 - 8.0 mmol/L	Peaked T waves Prolonged PR interval Decreased amplitude of P waves Widening of QRS complex
Severe hyperkalaemia > 8.0 mmol/L	Absence of P wave Progressive widening of QRS complex Eventual "sine-wave" pattern VF Asystole

Complete heart block (CHB) even though rare, can be seen at presentation in hyperkalemia[5]. The same has been observed in our present case as CHB was noted but surprisingly in the presence of only mild hyperkalemia (serum potassium 6.4mEq/L) which is a diagnostic challenge.

An electrocardiographic presentation of Left or Right bundle branch block pattern usually reflects an increase in the serum potassium level greater than 6.5mEq/L, due to worsening of intraventricular conduction delay. However in our present case RBBB pattern was noted post hyperkalemia treatment. But this paradox could be explained on the basis of patients previous pre existing RBBB.

Thus both the above observations in our present case further reiterates the fact that level of serum potassium does not correlate with the degree and progression of ECG changes, which is an important noticeable clinical lesson and it is also worthwhile to make an effort to trace the patients old ECGs in such grave emergency situations.

4. Conclusions

From the present case we conclude that - All patients with ECG changes suggestive of complete heart block (CHB) as an initial presentation also should be evaluated for serum potassium levels to identify underlying hyperkalemia even though it is rare. -Co morbidities such as Diabetes mellitus & Hypertension in elderly, further reiterate the need for serum potassium estimation in cases of complete heart block as initial presentation.

Lessons to Reckon: Prompt and efficient emergency management for CHB even in elderly (78 Years) is highly fulfilling and rewarding. -It is worthwhile to make an all out effort to trace patients old ECG even during grave situations like CHB, mainly to ascertain the cardiac stability and for rendering reassurance. Thus the present case highlights a rare presentation and offers a clear clinical message for all clinicians impacting the medical practice.

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