International Scholars Journals

African Journal of Medical Case Reports ISSN 2756-3316 Vol. 14 (2), pp. 001-003, February, 2026. Available online at www.internationalscholarsjournals.org © International Scholars Journals



*Case Report*

# Hyperkalemia-Induced Transient Trifascicular Block: A Case Report

**AGARWAL Navnit, SINGH Anurag, GABA Ripudaman*, SHUKLA Ranjeet, AGARWAL Mandavi and JAISWAL Pankaj**

Department of Medicine, MLB Medical College, Jhansi Uttar Pradesh, 284128, India.

Accepted 17 November, 2025

**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 atrioventricular (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.**

**Key words:** Hyperkalemia, trifascicular block, right bundle branch block, left anterior fascicular block, first degree AV block, isoprenaline, hemodialysis, emergency.

## INTRODUCTION

Hyperkalemia is life threatening electrolyte abnormality requiring urgent management. Serum potassium levels greater than 5.5 mmol/l is considered hyperkalemia. Hyperkalemia is a common and potentially life- threatening electrolyte abnormality (Sood et al., 2007). Hyperkalemia is often asymptomatic until plasma potassium concentration is above 6.5-7 mmol/L when it results in fatal arrhythmias, hence it's called silent killer. While the incidence of hyperkalemia in the general population is not known, it is approximated that this electrolyte disturbance occurs in 1 to 10% of hospitalized patients annually and carries a mortality rate of 1 per 1,000 patients (Sood et al., 2007). Hyperkalemia can lead to hyporeflexia and gradual paralysis. Hyperkalemia is associated with significant disturbances in cardiac conduction, ranging from QT interval shortening, to PR interval lengthening and QRS widening (Bashour et al., 1975). 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 (Alfonzo et al., 2006; Sood et al., 2007).

## CASE REPORT

A 65 year old female was brought to emergency department of Maharani Laxmi Bai (MLB) Government Medical College, Jhansi, India with complaints of retrosternal chest pain, diaphoresis and syncopal attacks. Patient has a known case of diabetes mellitus and systemic hypertension. Right nephrectomy had been performed 24 years back since patient had multiple renal calculi with pyonephrosis (right kidney). Patient was not on any treatment at the time of presentation. In emergency department patient's blood pressure was 166/92 mm of Hg and pulse rate was 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,

*Corresponding author. E-mail: ripu_2cool@yahoo.com. Tel: 09695980274.

**Abbreviations: AV,** Atrioventricular; **ECG,** electrocardiogram; **MLB,** maharani laxmi bai; **RBBB,** right bundle branch block; **LAFB,** left anterior fascicular block; **ABG,** arterial blood gas; **TLC,** total leucocyte count; **DLC,** differential leucocyte count; **CPK- MB,** creatine phosphokinase.

pulse rate 72 min, cardiac auscultation revealed no finding and all other systems were within normal limit. Patient's initial ECG showed heart rate of 78 min, PR interval of 255 millisec, QRS duration of 146 millisec and mean QRS axis to be 266. ECG was interpreted as trifascicular block. that is, right bundle branch block (RBBB), left anterior fascicular block (LAFB) and first degree 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. Arterial blood gas (ABG) analysis 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).

Patient's complete blood count showed total leucocyte count (TLC) of 15200/$mm^3$ , hemoglobin of 6.2 g%, differential leucocyte count (DLC) $P_{90}L_7E_3$ and hematocrit of 20.5%. Patient's 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 blood were transfused. On second day her hyperkalemia and acidosis recovered (S. $K^+$ 4.2 mmol/L, pH 7.5, $pCO_2$ 36.6 mm of Hg, $HCO_3$ 28.9 mmol/L). Her ECG on second day showed heart rate 85/min, PR interval of 137millisec, QRS duration of 102 millisec with mean QRS axis -53. ECG was interpreted as LAFB. Thus RBBB and first degree AV block reversed after correction of hyperkalemia. Uremia as a cause of transient trifascicular block was ruled out because blood urea and serum creatinine levels remained stable following hemodialysis where as the electrocardiographic changes reverted. Patient's serum creatinine was 4 mg% and blood urea
96.6 mg% before dialysis. Patient's serum creatinine was 3.94 mg% and blood urea 89.8 mg% after dialysis. To rule out coronary artery disease creatine phosphokinase (CPK-MB) and troponin-I were ordered. 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 up to .15 ng/ml is considered to be normal). To further rule out CAD, a 2-Dimensional echocardiography was done which revealed normal study (no regional wall motion abnormality, chamber size normal, left ventricular systolic function normal, left ventricular ejection fraction 66%). She was discharged on $5^{th}$ day of her hospital admission and advised regular follow up.

## 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 eitiology. 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's and Purkinje fibres susceptibility to hyperkalemia. Ohmae and Rabkin (1981) reported a case of RBBB with left axis deviation which resolved with correction of hyperkalemia. Bashour et al. (1975) 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, RBBB with left anterior hemiblock in two, RBBB with left posterior hemiblock in one, left bundle branch block with abnormal left axis deviation in two and advanced AV block in one. In all seven patients with sinus rhythm the PR interval shortened after correction of hyperkalemia. Electrophysiologic studies using His bundle recording and atrial pacing in one patient revealed intra-arterial conduction delay and marked prolongation of conduction time in the His-Purkinje system. Dittrich et al. (1986) reported that initial high T waves and shortened intervals give way to prolongation of conduction and lethal dysrhythmias as the serum potassium level rises. Weidner et al. (1978) described a case of RBBB in hyperkalemia which reversed after correction of hyperkalemia.

## CONCLUSION

Hyperkalemia is a commonly encountered electrolyte abnormality that can produce life threatening derangements in cardiac conduction. The emergency department 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.

## ACKNOWLEDGEMENT

We acknowledge Professor Dr. P. K. Jain, Prof. and Head, Department of Medicine, MLB Medical College, Jhansi, UP-India for helping us in publication of this article and providing adequate facilities for working on this study. The authors are highly thankful to the Residents of Postgraduate Department of Medicine, MLB

Medical College, Jhansi, UP-India, for their ever tiring hard work for working on this study.

**REFERENCES**

Alfonzo AVM, Isles C, Geddes C, Deighan C (2006). Potassium disorders—clinical spectrum and emergency management. Resuscitation, 70(1): 10-25.

Bashour T, Hsu I, Gorfinkel HJ, Wickramesekaran R, Rios JC (1975). Atrioventricular and intraventricular conduction in hyperkalemia. Am. J. Cardiol., 35(2): 199-203.

Dittrich KL, Walls RM (1986). Hyperkalemia: ECG manifestations and clinical considerations. J. Emerg. Med., 4(6): 449-455.

Ohmae M, Rabkin SW (1981). Hyperkalemia-induced bundle branch block and complete heart block. Clin. Cardiol., 4(1): 43-46.

Sood MM, Sood AR, Richardson R (2007). Emergency management and commonly encountered outpatient scenarios in patients with hyperkalemia. Mayo Clinic Proceedings, 82(12): 1553-1561.

Weidner NJ, Gaum WE, Chou TC, Kaplan S (1978). Hyperkalemia-electrocardiographic abnormalities. J. Pediatr., 93(3): 462-464.