

## Case Report:

### Left bundle branch block: a rare ECG manifestation of hyperkalemia

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#### ABSTRACT

A 20-year-old female patient with chronic kidney disease was brought to our emergency medical department with symptoms of pain in chest and abdomen, vomitings. Laboratory testing revealed serum potassium 7.7 mEq/L, serum creatinine 9.1 mg/dL. Electrocardiogram (ECG) showed left bundle branch block (LBBB) pattern with left axis deviation, tall T waves and ST elevation. Among ECG alterations in hyperkalemia, LBBB is rare and is being reported in our case.

**Key words:** *Electrocardiogram, Hyperkalemia, Left bundle branch block*

Venkata Madhav M, Anvesh G, Sessaiah KV, Eswar G, Mohammad A. Left bundle branch block: a rare ECG manifestation of hyperkalemia J Clin Sci Res 2015;4:159-63. DOI: <http://dx.doi.org/10.15380/2277-5706.JCSR.13.075>.

#### INTRODUCTION

Extracellular potassium concentration is normally maintained between 4.0-4.5 mEq/L. Hyperkalemia, defined as serum potassium greater than 5.0 mEq/L, is a common metabolic disturbance with potentially lifethreatening consequences. While the incidence of hyperkalemia in the general population is not well documented, it is 1% to 10% in hospitalized patients, with a mortality ratio of 1 per 1000 patients.<sup>1</sup> Electrocardiogram (ECG) may provide the first evidence of hyperkalemia, but patient-to-patient variability is high. Even though most common ECG findings in hyperkalemia include peaked T waves and widened QRS complexes, hyperkalemia can cause several characteristic abnormalities which are often progressive. Poor correlation has been observed between serum potassium levels and any specific ECG finding and the sensitivity of ECG for diagnosis of hyperkalemia is poor.<sup>1-3</sup> At the same time, any ECG change in the clinical background of hyperkalemia should

alert the clinician for carrying out the work-up for diagnostic confirmation. We report the occurrence of LBBB caused by hyperkalemia in a young lady with Chronic kidney disease (CKD).

#### CASE REPORT

A 20-year-old female patient was brought to our emergency department with complaints of pain abdomen, vomiting, and retrosternal chest pain of acute onset. Abdominal pain and chest pain were dull aching in character and were not related to any known precipitating factors. She was known to have CKD and was on maintenance haemodialysis since 3 months. Her past history, birth and developmental history, childhood history were unremarkable. Patient denied taking any medications known to precipitate her clinical condition. She was not known to have hypertension, diabetes mellitus.

General and systemic physical examination was unremarkable. Laboratory values were hemoglobin 8.3g/dL; total leukocyte count

Received: 12 December 2013; Revised manuscript received: 03 July 2014; Accepted: 16 August 2014

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13,100 cells/mm<sup>3</sup>; neutrophils 80%. Blood urea nitrogen 40 mg/dL; serum creatinine 9.1 mg/dL; random plasma glucose 120 mg/dL; serum sodium 146 mEq/L; and serum potassium 7.7 mEq/L. Antistreptolysin O titers, troponin I levels, and calcium were within normal range. Arterial blood gas analysis showed pH 7.20; PaO<sub>2</sub> 109 mm Hg; PaCO<sub>2</sub> 55 mm Hg; bicarbonate 16.2 mEq/L. ECG showed LBBB with left axis deviation, secondary ST-T changes and tall T waves in chest leads (Figure 1). A 2D-echocardiogram was unremarkable. Chest radiograph showed mild cardiomegaly with normal lung fields.

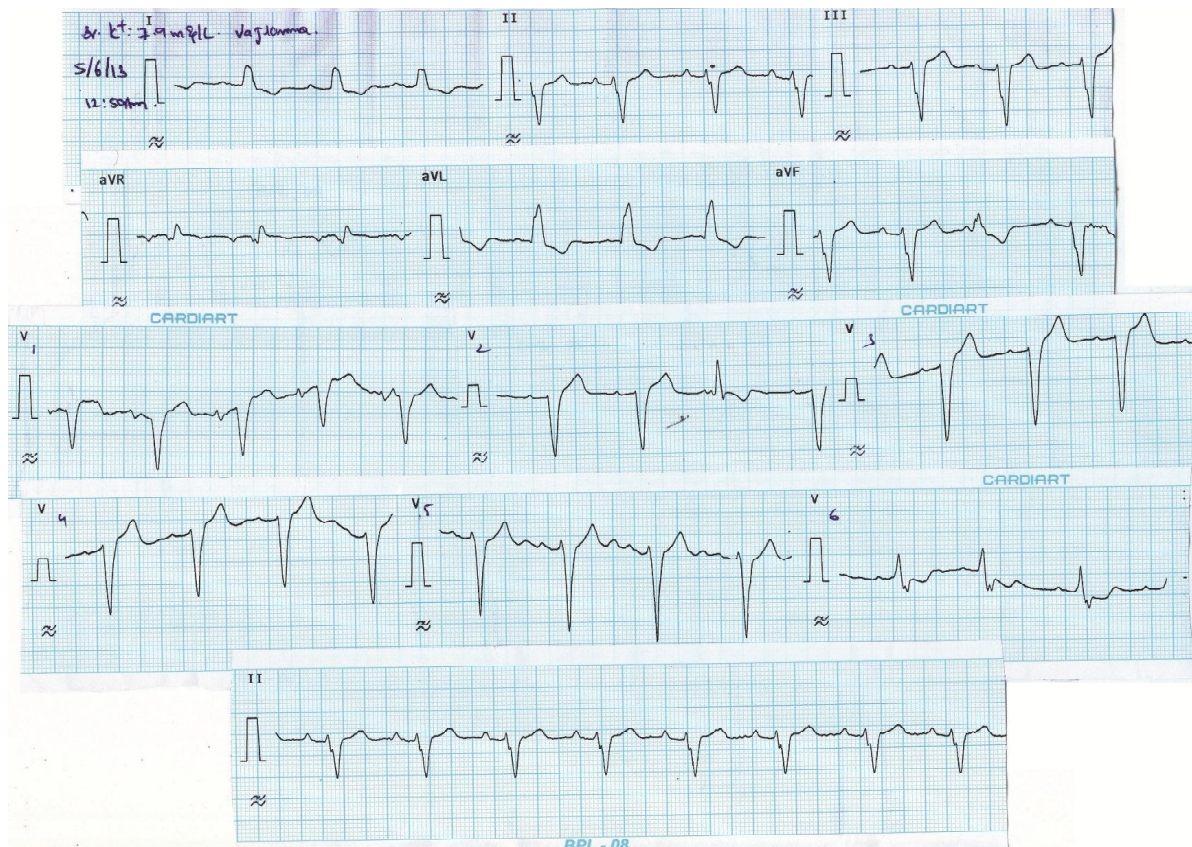
She was initially treated with intravenous calcium gluconate, insulin with glucose, salbutamol nebulization, and sodium polystyrene sulfonate. Drug treatment did not normalize her serum potassium levels. Haemodialysis was initiated, with which her serum potassium levels as well as ECG changes become normal (Figure 2).

In the clinical background of CKD and absence of known precipitating factors for increased serum potassium levels, her hyperkalemia was attributed to CKD. With normal cardiac evaluation and absence of risk factors for coronary arterial disease, her ECG findings, were attributed to be due to hyperkalemia.

## DISCUSSION

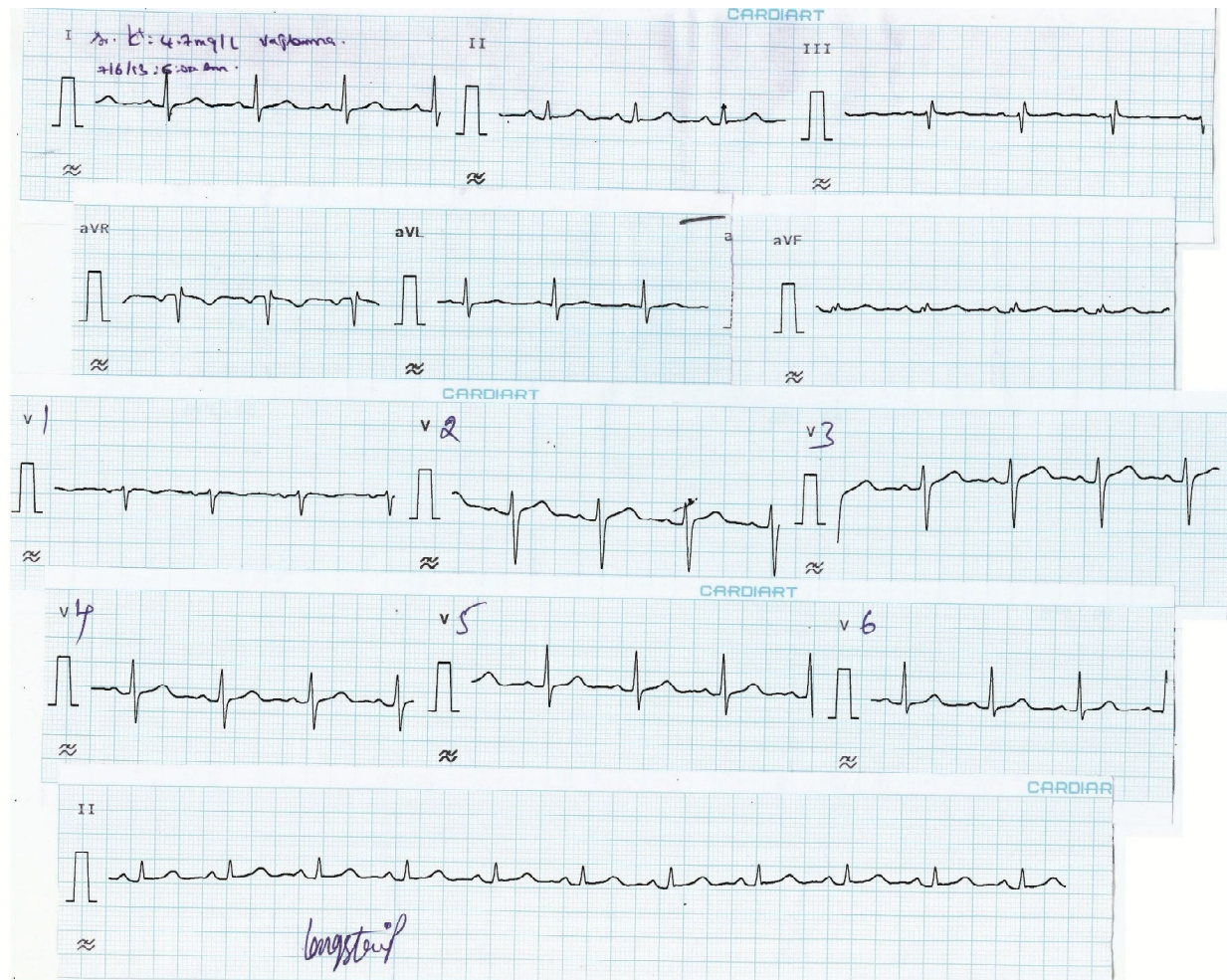
Under normal circumstances renal excretion accounts for approximately 90% of daily potassium elimination. As renal function declines, a greater fraction of the filtered load is excreted and serum potassium levels do not rise until renal function declines to less than 25% of normal.<sup>2</sup>

Hyperkalemia decreases the resting membrane potential (RMP), the magnitude of the action potential, and maximum rate of rise of phase 0 in the cardiac muscle. Hyperkalemia produces a biphasic effect on arterio-venous (AV) conduction system, with mild hyperkalemia



**Figure 1:** Pre-treatment electrocardiogram showing changes of hyperkalemia





**Figure 2:** Post-treatment electrocardiogram showing normalization

accelerating the AV conduction, and severe hyperkalemia ( $\geq 7.5$  mEq/L) depressing it.<sup>3</sup> A high concentration of extracellular potassium slows impulse conduction through all cardiac tissue, which accounts for a number of ECG findings.

Disproportional effects of varying levels of hyperkalemia on the resting membrane potential and the threshold potential (TP) explains the initial increase in excitability and conduction velocity followed by their decrease as the potassium level increases further. Hyperkalemia is associated with increased membrane permeability to potassium, which accelerates repolarization and shortens the action potential (AP) duration. Hyperkalemia preferentially shortens the plateau of the Purkinje fibres, thereby decreasing the

dispersion of repolarization in the ventricle. It also slows the diastolic depolarization of the Purkinje fibers.<sup>4</sup> The effect of hyperkalemia depends on the tissue involved, with the atrial myocardium being the most sensitive, the ventricular myocardium less sensitive, and the specialized tissue (sinoatrial node and His bundle) the least sensitive. Atypical bundle branch blocks, intraventricular conduction delays, ventricular tachycardia, ventricular fibrillation and idioventricular rhythm are more commonly seen in severe hyperkalemia (serum potassium  $> 10$  mEq/L). Bundle branch blocks in hyperkalemia are atypical in the sense that they involve the initial and terminal forces of QRS complex. Shifts in the QRS axis indicate disproportionate conduction delays in the left bundle fascicles.<sup>4</sup>

As hyperkalemia progresses, depolarization merges with repolarization, expressed in the ECG with QT shortening and apparent ST segment elevation simulating acute injury. This disappears with haemodialysis dialyzable current of injury.

The disproportionate conduction delay in the bundle branch system occurs more often in the right than in the left, unlike in our patient who had LBBB pattern.<sup>5</sup> The hyperkalemic ECG changes range from peaked T waves, loss of P waves, prolonged QRS complex, ST elevation, escape beats and escape rhythm to sine wave, ventricular fibrillation, asystole, axis deviations, bundle branch blocks, and fascicular blocks.<sup>6</sup> Sinus node depression, and malignant ventricular arrhythmias can also occur. Usual order of appearance of ECG changes is tall T waves, QRS widening, loss of P wave, ST elevation with “pseudo infarction” pattern, sine wave and eventual asystole.<sup>7</sup>

In a study of 27 patients with ECG changes of hyperkalemia, elevated serum potassium levels were found in 22 patients (82%); On the other hand in 39 patients with hyperkalemia, ECG showed agreement in only 21 (54%).<sup>8</sup> In a retrospective study, ECG changes were seen in 43% of patients with serum  $K^+$  ranging from 6.0-6.8 mEq/L, and in only 55% of patients with value of 6.8 mEq/L or greater.<sup>1</sup> Another study documented that ECG changes were associated with serum  $K^+$  6.8-7.6 mEq/L, and found consistent above levels of 7.8 mEq/L.<sup>9</sup> In one more study<sup>7</sup> of 292 patients with hyperkalemia, only 40 abnormal ECGs were found. In this study<sup>7</sup> peaked T waves, most frequent ECG change were seen in only two patients with mild (5.0-6.9 mEq/L) hyperkalemia and prolonged PR interval was seen in all ranges of serum  $K^+$  levels.

In general, a correlation can be observed between increasing abnormality of ECG pattern and increasing serum potassium concentrations.

However, patient-to-patient variability is high. ECG changes might be subtle or even absent, further complicating the diagnosis. Thus ECG findings might not be sensitive in detecting mild and moderate hyperkalemia, as documented in many studies.

It was documented that there was a lack of confirmity of ECG manifestations with serum postassium levels and this disparity was attributed to the presence of ECG alterations from other causes or to concomitant abnormalities of other electrolytes.<sup>8</sup> In nephrectomized animals or in the presence of renal disease with acidosis, small increases in the serum potassium concentration may quickly alter the  $K_e / K_i$  (extracellular potassium/ intracellular potassium) ratio resulting in early and typical changes of hyperkalemia. But in the presence of alkalosis with intact renal regulatory mechanism, a disturbance of the  $K_e/K_i$  ratio may not occur until there is marked depletion of the body potassium and ECG changes can be expected to be delayed. Thus, it is conceivable that the effects of the electrolyte derangement on the ECG may depend to a significant degree on the presence or absence of renal regulation of serum electrolytes.<sup>8</sup>

Among hyperkalemic changes, LBBB was reported as rare finding and was associated with severe hyperkalemia ( $>8.0$  mEq/L). In a study<sup>10</sup> of atrioventricular and intraventricular conduction in hyperkalemia (n=12) the authors reported isolated left posterior hemi-block (LPHB) in 4; isolated left anterior hemi-block (LAHB) in 2; RBBB with LAHB in 2; RBBB with LPHB in 1; LBBB with abnormal left axis deviation in 2; and advanced atrio-ventricular block in 1 patient.

Two cases with hyperkalemic ECG changes with RBBB with left axis deviation in one case and complete heart block in second case, which disappeared with treatment of hyperkalemia have been reported.<sup>5</sup> Hyperkalemia appears to

potentiate subclinical conduction abnormalities especially in the His-Purkinje system.

ECG changes of hyperkalemia are frequently consistent at higher levels of serum  $K^+$  ( $\geq 7.8$  mEq/L) and often progressive.<sup>11</sup> Among hyperkalemic ECG changes, conduction blocks are infrequent and also LBBB is rare compared to RBBB. LBBB, which was a rare report in literature, was documented in our patient, in addition to the other ECG findings.

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