

## Case Report



# Reversible complete atrioventricular block in patient with mild hyperkalemia

## Abstract

Complete atrioventricular (AV) block or third degree AV block is a type of bradyarrhythmia that can be diagnosed as mortality if it is not noticed and treated. There are several causes of complete AV block and one of them is severe hyperkalemia. A 84 year-old woman known to type II diabetes mellitus, applied to emergency department with nausea, vomiting, fatigue, conscious semi-closed. Hypotension and complete AV block were detected. Lab findings showed metabolic acidosis and mild hyperkalemia (K:6,1 mmol/L). Fluid replacement, noradrenalin infusion,  $\beta_2$  agonist and insuline infusion administered and her cardiac rythm returned sinus rythme without pacemaker or hemodialysis. Generally, hyperkalemia related complete AV block occurs in severe hyperkalemia (K>7 mmol/L) and a temporary pacemaker and/or hemodialysis are required for treatment. In this case report, serum potassium level was lower than 7 mmol/L but complete AV block occurred and she was treated successfully without the temporary pacemaker or hemodialysis.

**Keywords:** complete atrioventricular block, mild hyperkalemia, temporary pacemaker, intensive care unit

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 Behiye Deniz Kosovali,<sup>1</sup> Huseyin Yildiz<sup>2</sup>
<sup>1</sup>Department of Intensive Care Unit, Malatya Education and Research Hospital, Turkey

<sup>2</sup>Department of Internal Medicine, Malatya Education and Research Hospital, Turkey

**Correspondence:** Behiye Deniz Kosovali, Department of Intensive Care Unit, Malatya Education and Research Hospital, 44330 Malatya, Turkey, Tel +90 422 444 56 34, Fax +90 422 232 53 43, Email kosovalideniz@yahoo.com

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## Introduction

Complete atrioventricular (AV) block or third degree AV block is a type of bradyarrhythmia that can be diagnosed as mortality if it is not noticed and treated. Complete AV block results from various pathologic states. The causes of AV block are as drugs, ischemic or infarctive causes, degenerative diseases, metabolic causes, infectious cause, rheumatic diseases, infiltrative processes, neuromuscular disorders, toxins and congenital causes. Common metabolic causes of AV block are as hypoxemia, hyperkalemia, hypothyroidism.

Potassium (K) is an extracellular ion with an important role in the electrophysiologic regulation of myocardial function. Different concentrations of high intracellular and low extracellular potassium ions, the opposite of which is true for sodium ion, are maintained by sodium potassium adenosine triphosphatase (Na-K ATPase) pumps, giving rise to a resting membrane potential of -90 mV across the myocytes membrane. As a result, any change in extracellular potassium concentration may have a significant effect on myocyte electrophysiologic gain.<sup>1</sup> Serum potassium level greater than 5,5 mmol/L is hyperkalemia and severe hyperkalemia is defined as potassium level >7 mmol/L. Serum potassium >7 mmol/L causes conduction abnormality such as prolonged QRS interval, AV nodal block, sinus bradycardia and or sine wave pattern.<sup>2,3</sup> Moreover, hyperkalemia is known to cause potentially lethal dysrhythmias including ventricular tachycardia, ventricular fibrillation, idioventricular rhythms, and asystole.<sup>3</sup> Complete AV block generally occurs with severe hyperkalemia. We present a case who was treated with mild hyperkalemia related complete AV block without the temporary pacemaker.

## Case presentation

A 84 year-old woman known to type II diabetes mellitus (DM) and had irregular insuline use. She applied to emergency department with nausea, vomiting, fatigue, conscious semi-closed and general condition disorder. On arrival to the intensive care unite (ICU), she was lethargic and dry mucous membranes. Her initial vital signs showed a normal temperature, pulse 35 beats/minute, respiratory rate 20 breaths/minute, pulse oximetry 85% (5-6 L/minute mask oxygen), and blood

pressure 90/40 mmHg, mean arterial pressure (MAP) 50 mmHg. Examinations of the head, neck, chest, and abdomen were normal, showed no meningeal signs and Glaskow Coma Score (GKS) 10. Only bradyarrhythmia was defined, no additional sound or murmur heard on cardiovascular auscultation. Her glucose level, determined by a finger stick, was 305 mg/dL and arterial blood gas (ABG) analysis showed pH:7,18 partial oxygen pressure (PaO<sub>2</sub>) 58, partial carbon dioxide pressure (PaCO<sub>2</sub>) 45mmHg, bicarbonate (HCO<sub>3</sub>) 15mmol/L, lactate 3,6 mmol/L, base excess (BE) -10, saturation (SaO<sub>2</sub>) 88%. The patient was given supplemental oxygen, IV fluids replacement, and placed on a cardiac monitor. An immediate electrocardiogram (ECG) was done which obtained bradycardia with complete AV block at rate of 53 beat/min (Figure 1). Despite administration of approximately one liter normal saline the patient remained hypotensive, with a blood pressure of 85/55 mmHg and noradrenalin infusion (0,5 µg/kg/dk) was added. On her blood sample results included leukocyte (WBC):7190/mm3, hematocrit (Htc):34%, platelet (Plt):197000/mm3, glucose:272 mg/dL, urea:59 mg/dL, creatinine:1,11 mg/dL, sodium (Na):129 mmol/L, potassium (K):6,1 mmol/L, chlorine (Cl):100 mmol/L, troponin I <0,1 ng/mL (0-0,3 ng/mL normal range), C reactive protein (CRP):0,48 mg/dL (0-0,5mg/dL normal range). Immediately 10 mL 10% calcium gluconate was administered intravenously (IV) over 5 minutes, inhaler  $\beta_2$  agonist agent (salbutamol) and dextrose 5% mixed with 10 IU insuline IV were started for treatment of hyperkalemia. Transthoracic echocardiography (ECHO) showed ejection fraction 50%, bradycardia and no abnormal function. Consultant of cardiology was planned to applied the temporary pacemaker. The patient was prepared for procedure, approximately two hours later, her MAP was greater than 70mmHg, GCS improved 15 and control ABG analysis showed pH:7,26 PaCO<sub>2</sub>:31mmHg PaO<sub>2</sub>:105mmHg HCO<sub>3</sub>:17 mmol/L, lactate:1,8 mmol/L, BE:-8 SaO<sub>2</sub>:97%, K:5,5 mmol/L, Na:130 mmol/L. At the same time on cardiac monitor we identified the patient's cardiac rhythm returned sinus rhythm and ECG showed sinus rhythm, too (Figure 2). The temporary pacemaker procedure was waived. The patient's clinical symptoms disappeared and laboratory findings recovered within 24 hours. Last lab results were urea:46 mg/dL, creatinine:0,86 mg/dL, Na:136 mmol/L, K:4,2 mmol/L, Cl:105 mmol/L, pH:7,35, HCO<sub>3</sub>:21mmol/L, PaO<sub>2</sub>:75mmHg, PaCO<sub>2</sub>:41 mmHg, SaO<sub>2</sub>:96%. She was discharged from ICU to general ward.



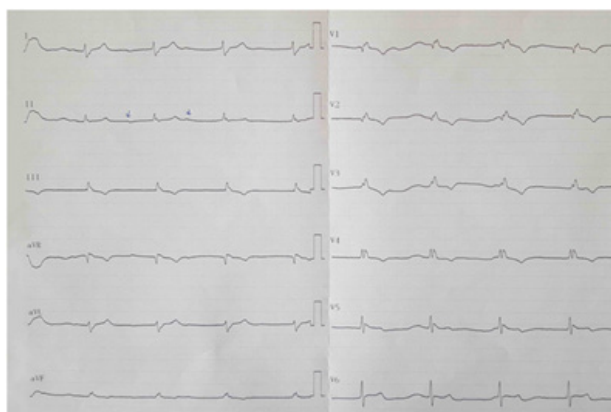


Figure 1

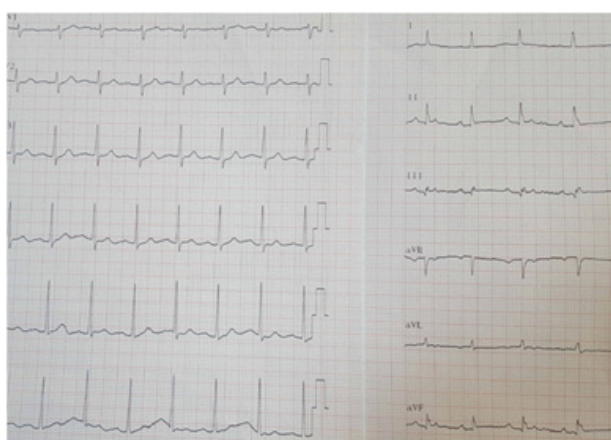


Figure 2

## Discussion

Elderly patients also often develop AV node and His-Purkinje disease. It is common for patients to present to the emergency department with symptoms of fatigue, exercise intolerance, dizziness, near syncope, or syncope.<sup>4</sup> Our case was applied to emergency department similar symptoms and admitted to intensive care unit. Drugs such as digoxin or calcium channel blockers, can be caused hyperkalemia or hyperkalemia related complete AV block. The patient's companion informed us that she didn't take any medication except for insulin.

Atrioventricular (AV) block is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomical or functional impairment in the conduction system. This disturbance of impulse transmission can be transient or permanent and may manifest as conduction delay in the AV node (first degree), intermittent failure of conduction from the atria to the ventricles (second degree), or complete (third degree) AV block.<sup>5</sup> Third-degree AV block is present when no P waves are conducted. The atrial rate is dissociated from the ventricular rate.<sup>4</sup>

Severe hyperkalemia is an important cause of complete AV block. Although hyperkalemia is defined as a serum potassium concentration  $>5$  mmol/L, moderate (6-7 mmol/L) and severe ( $>7$  mmol/L) hyperkalemia are life-threatening and require immediate therapy. Hyperkalemia also may manifest as a conduction block at different levels, including bundle branch block or AV block; conduction defects in the specialized intraventricular conduction system are common in

hyperkalemia and result in aberrant ECG patterns.<sup>6</sup> Electrocardiogram (ECG) findings in hyperkalemia progress from tall, "peaked" T waves and a shortened QT interval to a lengthening of PR interval and loss of P waves, and then to widening of the QRS complex, culminating in a "sine wave" morphology and death if not treated.<sup>7</sup> The correlation between the degree of ECG changes and the serum potassium level is poor; AV block is typically associated with a serum potassium level  $>6,3$  mmol/L.<sup>8</sup> Our patient's serum potassium level was 6,1 mmol/L and wide abnormal QRS complexes in all leads and complete AV block with a heart rate of 53 beats/min were observed on her ECG. It is aimed to place potassium into the cell in the treatment of the hyperkalemia, so IV,  $\beta_2$  agonist agents, IV insulin treatments are admitted. Calcium gluconate protects against to cardiac adverse effects and exchange resin is used for exchange sodium to potassium in gastrointestinal system. Hemodialysis is required if there are life-threatening hyperkalemia, chronic renal failure, rhabdomyolysis, untreatable or persistent hyperkalemia.<sup>9</sup> Calcium gluconate, inhaler  $\beta_2$  agonist, IV insulin, fluid replacement administered but hemodialysis was not required.

Several case reports have published about hyperkalemia related complete AV block in the literature. But these cases' serum potassium levels are greater than 6.3 mmol/L or serum potassium levels are lower than 6,3 mmol/L and renal failure accompany to hyperkalemia.<sup>2,7</sup> In spite of standard hyperkalemia treatment hemodialysis and pacemaker were required in the treatment of most patients. In presented case, despite serum potassium level was lower than 6,3 mmol/L and renal function parameter was normal, complete AV block occurred. The other interesting sign in our case, she was treated without the temporary pacemaker or hemodialysis. The elderly patients can be more sensitive in mild hyperkalemia than younger patients and clinical findings can occur earlier.

## Conclusion

Hyperkalemia can be life-threatening, especially elderly patients and to be aware of cardiac complications. Examination findings, especially abnormal ECG findings and presence of bradyarrhythmia should be suspected hyperkalemia and the hyperkalemia treatment is started immediately. Quick recognition and treatment is the main factor. Thereby patients should be treated without invasive procedures, such as hemodialysis or pacemaker.

## Acknowledgments

None.

## Conflicts of interest

None declared.

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