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Extreme Bradycardia with Variable Block in Severe Hyperkalemia: A Forgotten Culprit in Brady-Arrhythmia

Han Naung Tun[1,2]* and Syed Haseeb Raza[3]

[1]Coronary Care Unit, Heart and Vascular Centre, Victoria Hospital, Yangon, Myanmar
[2]National Representative of Heart Failure Specialist of Tomorrow (HoT) for Myanmar in European Heart Failure Association and Clinical and Research Working Group of Cardiac Cellular Electrophysiology, European Society of Cardiology, Sophia Antipolis, France
[3]Department of Cardiac Electrophysiology, National Institute of Cardiovascular Diseases, Karachi, Pakistan

Abstract

Bradycardia is commonly encountered in emergency department. Hyperkalemia may sometime cause bradycardia with block and also synergize with AV node blockers to cause bradycardia and hypoperfusion. We report a 53 years old male with history of hypertension, congestive heart failure and coronary artery disease was admitted to hospital for sudden onset of breathlessness. He underwent Percutaneous Coronary Intervention (PCI) to Left Anterior Descending (LAD) artery and Left Circumflex (LCx) artery one year ago and taking Aspilet 80 mg for daily, Clopidogrel 75 mg daily, Ramipril 5 mg daily, Atorvastatin 20 mg daily, Metoprolol 25 mg daily, Spironolactone 25 mg daily and Frusemide 40 mg daily. Significant physical examination was remarkable for a temperature 97.5'F, blood pressure of 110/70 mmHg, heart rate of 40 beats per minute, oxygen saturation was 99% on air and both lung were full with audible crepitation by auscultation. He was given atropine 0.6 mg bolus and transcutanaeous pacing with unimproved heart rate and then a transvenous pacing was immediately placed before the blood investigation results were returned. His relevant laboratory values were significant for a potassium of 7.99 mmol/L(ref range: 3.5-5.2 mmo/l), creatinine of 458 micmol/L (ref range: 59-104 micmol/L), Urea of 33.9 mmol/L (ref range: 2.7-8.0 mmol/l), random blood glucose of 233mg/dl, sodium 126.8 mmol/L (ref range 135-145 mmol/L), anion gap of 13.5 mmol/? (ref range: 3.6-11.0 mmo/L) and bicarbonate of 15.6 mmil/L (ref range: 22-29 mmol/L). He was given calcium glucoronate, insulin with dextrose, kaexylate, nebulizer salbutamol with significant improvement in his potassium levels to 4.6 in 24 hours. In Cardiac intensive care unit his heart rate was improved and the transvenous pacemaker was turned off the next day.

Keywords: Hyperkalemia • Bradycardia • Pacemaker • Heart block

Introduction

Bradycardia is commonly encountered in emergency department. Hyperkalemia may cause bradycardia with block and may also synergize with AV node blockers to cause bradycardia and hypoperfusion [1]. Potassium is vital for regulating the normal electrical activity of the heart. Increased extracellular potassium reduces myocardial excitability, with depression of both pacemaking and conducting tissues [2]. Progressively worsening hyperkalaemia leads to suppression of impulse generation by the SA node and reduced conduction by the AV node and His-Purkinje system, resulting in bradycardia and conduction blocks and ultimately cardiac arrest [3].

Case Presentation

A 53-year-old man known to have coronary heart disease, congestive heart failure, type 2 diabetes mellitus and hypertension was taking Aspilet 80 mg for daily, Clopidogrel 75 mg daily, Ramipril 5 mg daily, Atorvastatin 20 mg daily, Metoprolol 25 mg daily, Spironolactone 25 mg daily and Frusemide 40 mg daily. His Underwent Percutaneous Coronary Intervention (PCI) to Left Anterior Descending (LAD) artery and Left Circumflex (LCx) artery one year ago. He presented to the emergency department in acute shortness of. He had no documented fever, no history of trauma, no gastroenterological symptoms and no other recent complaints before this event but his family members said that he had muscle pain before 3 days and had intramuscular injection of pain killer. On arrival to the emergency department, he was dyspnoic with lightheadedness. Her initial vital signs showed a temperature of 97.5'F, blood pressure of 110/70 mmHg, heart rate of 40 beats per minute, oxygen saturation was 99% on air and both lung were audibled crepitation by auscultation. His glucose level, determined by a finger stick, was 233 mg/dl. The patient was given IV atropine 0.6 mg bolus, started on intravenous frusemide 80 mg and nebulizer salbutamol 5 mg, and placed on a cardiac monitor. An immediate ECG was obtained Figure 1; showing marked bradycardia with ventricular response at rate of 45 bpm with variable block was noted in lead II and III.

Despite given IV Atropine, the heart rate remained extreme bradycardia with ventricular rate of around 40 bpm. So transcutaneous pacing pad was placed on chest shown in Figures 2 and 3 and sent to cardiac intensive care unit. In the cardiac care unit, immediate transvenous pacing (VVI mode) was implanted into right ventricle as his heart rate was still unimproved on transcutaneous pacing. Blood samples were sent for immediate determination of Arterial Blood Gas (ABG) concentrations, complete blood count, urea and electrolytes, liver function, and cardiac enzymes. His relevant laboratory values were significant for a potassium of 7.99 mmol/L (ref range: 3.5-5.2 mmo/l), creatinine of 458 mic-mol/L (ref range: 59-104 mic-mol/L), Urea of 33.9 mmol/L (ref range: 2.7-8.0 mmol/l), random blood glucose of 233 mg/dl, sodium 126.8 mmol/L (ref range 135-145 mmol/L), anion gap of 13.5 mmol/? (ref range: 3.6-11.0 mmo/L) and bicarbonate of 15.6 mmil/L (ref range: 22-29 mmol/L). Other blood tests were normal. The patient was immediately started on 10 mg salbutamol by nebulization and 10% calcium glucoronate of 10 ml was administered intravenously (IV) over 5 minutes. Dextrose 50% (D50) mixed with 10 IU regular insulin IV, 50 ml of 8.4% bicarbonate IV slowly, and 30 gm K-exelate were also administered. His heart rate rose to 98/min under

***Address for Correspondence:** Tun HN, Coronary Care Unit, Heart and Vascular Centre, Victoria Hospital, Yangon, Myanmar, E-mail: annasxhan@gmail.com*

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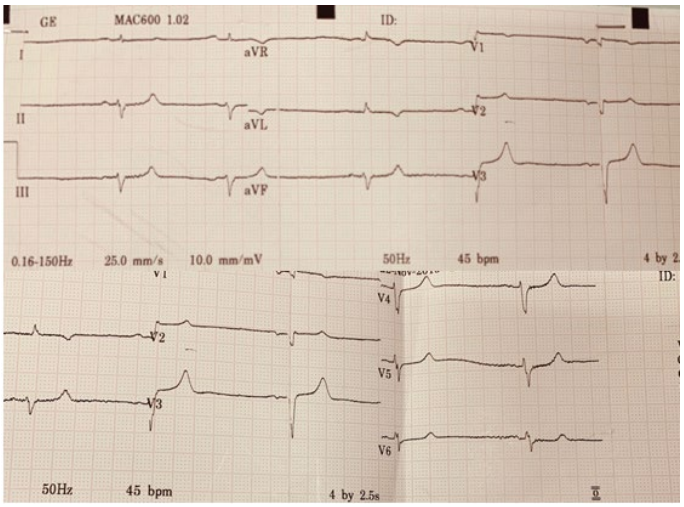

Figure 1. ECG on admission showing severe bradycardia with variable block.

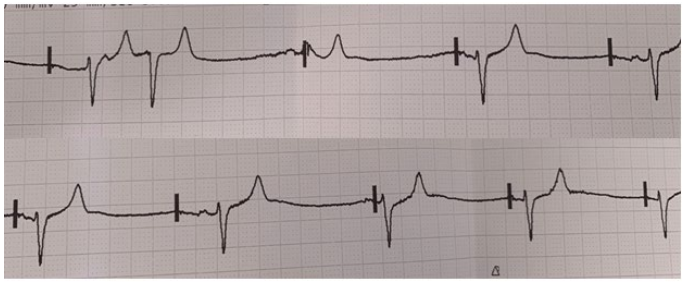

Figure 2. ECG on admission showing bradycardia with failure to capture on transcutaenous pacing.

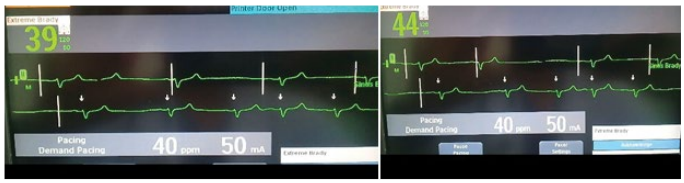

Figure 3. ECG on monitoring after transcutenous pacing in emergency department.

VVI mode temporary pacing (Figure 4). After 24 days of potassium correction to K level (6.5 mmol/L) and The Temporary Pacemaker (TPM) was turned off. The rechecked ECG after removal of TPM was shown in Figure 5.

Discussion

Hyperkalemia is a dangerous electrolyte disorder that can lead to serious hemodynamic and neurologic complications. When serum potassium levels exceeding 8.5 mEq/L can cause respiratory paralysis or cardiac arrest.4 In general, hyperkalemia is seen when the increased potassium intake, decreased potassium excretion, or a shift of potassium from the intracellular to the extracellular space. Serum potassium more than 7.0 mEq/L is associated with abnormal cardiac electrical conduction and bradycardia with prolonged QRS interval, bizarre QRS morphology, high-grade AV block, slow junctional and ventricular arrhythmias, any kind of conduction block such as bundle branch blocks and fascicular blocks [4]. Sinus bradycardia or slow Atrial Fibrillation (AF) and development of a sine wave appearance (a pre-terminal rhythm) may also be seen in the electrocardiographic findings [5]. In cardiac cellular electrophysiology, higher serum potassium is usually seen as a flattening of the part of the action potential due to the reduction of cardiac pacemaker action potential in the concentration gradient (outflow) of potassium in repolarizaton state that leads to reduced heart rate [6].

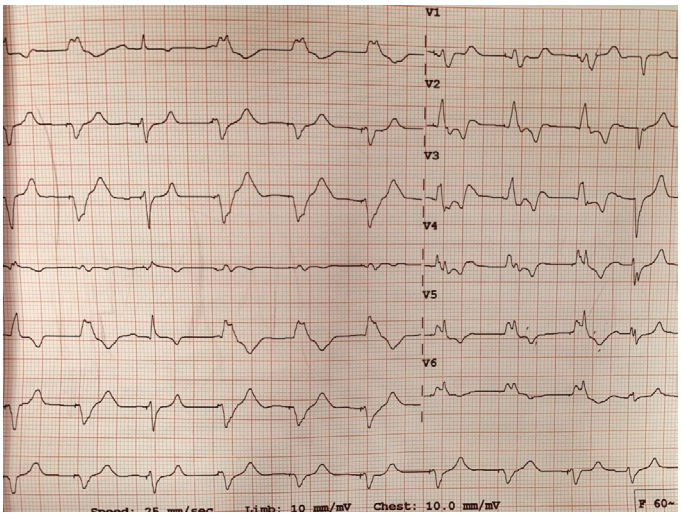

Figure 4. ECG after temporary pacemaker implantation.

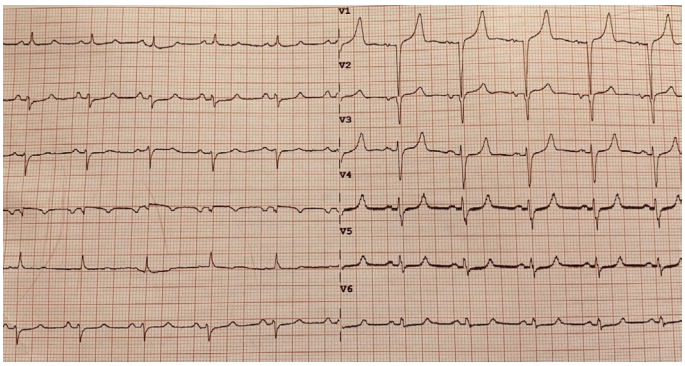

Figure 5. ECG shows sinus rhythm with tall and tent T seen in V1, V3 and V4 leads after removal of temporary transvenous pacing.

If patients are found to be with bradycardiac, clinicians and medics should not miss life threatening hyperkalemic bradyarrhythmia due to electrolytes imbalance that may cause profound bradycardia or with different forms of heart block. The clue to the correct diagnosis is the broad QRS complex with absence of P waves. Hyperkalaemic related second and third degree Atrioventricular (AV) block may be been seen sometime but they are uncommon because the P wave usually disappears before such advanced AV block occurs. If the ECG is available before the serum potassium and is consistent with life threatening hyperkalaemia, then it would seem sensible to give calcium glucoronate speculatively while waiting for the biochemistry results. However, the prevalence of severe hyperkalemia accompanying symptomatic bradycardia has only been explored in a few case reports. Some authors have reported “BRASH” syndrome that can be seen due to a vicious cycle in the setting of medications, hyperkalemia, and renal failure [7,8]. Renal failure causes hyperkalemia which may cause the accumulation of some AV node blockers (e.g. Atenolol, Nadolol). Hyperkalemia synergizes with AV node blockers to cause bradycardia and hypoperfusion. Hypoperfusion, in turn, causes worsening of the renal failure [7,8]. In this case, our patient was seemed as hyperkalemia with low Estimated Glomerular Filtration Rate (eGFR) that was caused by acute kidney injury, and on beta-blocker medication but not in hypotensive shock state.

Conclusion

Hyperkalemic related bradyarrhythmia is not uncommon cardiac conduction disorder. Clinicians should be knowledgeable about the electrocardiogram changes and manifestations of electrolyte imbalance to provide effective initial management for hyperkalemia induced bradyarrhythmia in emergency care

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