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Case Report

A RARE CASE OF REVERSIBLE HEART BLOCK IN PATIENT WITH ACUTE KIDNEY INJURY AND MILD HYPERKALEMIA

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ABSTRACT

Hyperkalemia caused by acute renal failure has variable effect on the myocardial tissue depending upon its concentration in blood. This electrical abnormality varies from prolonged P-R interval to variable degree heart blocks. This is a case report of a 63 year old gentleman known case of diabetes and hypertension (treatment with bisoprolol) who presented to emergency department with complaints of breathlessness associated with chest pain since three days. Examination showed bradycardia, hypotension and bilateral pedal edema. Lab test showed mild hyperkalemia and elevated serum creatinine levels. ECG demonstrated features suggestive of complete heart block. In view of bradycardia, renal failure, AV nodal blocker therapy and hyperkalemia, he was diagnosed that the bradycardia is secondary to synergetic effect of renal failure, mild hyperkalemia and AV nodal blockers therapy. Patient was managed with intravenous fluids, vasopressors, hemodialysis and temporary pacemaker. High level of clinical vigilance and appropriate timely management can reduce mortality.

Key words: Bradycardia, Hyperkalemia, Hypotension, Renal failure.

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INTRODUCTION

Potassium is essential to regulate normal electrical activity of heart. Hyperkalemia is known to cause adverse clinical outcomes [1-4]. Hyperkalemia is defined as potassium level > 5.5 and severe hyperkalemia > 7 meq/l. Hyperkalemia is commonly seen in renal failure. Serum potassium >7 meq/l causes conduction abnormality such as prolonged QRS interval, AV nodal block, sinus bradycardia and or sine wave pattern [5,6]. Patients who are taking AV (Atrioventricular) nodal blockers, mild hyperkalemia can cause complete heart block during acute kidney.

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CASE REPORT

A 63 year old male, known case of diabetes and hypertension (treatment with bisoprolol) presented to our hospital with bilateral lower limb swelling and decreased urine output since two days, breathlessness and chest pain since few hours. History of percutaneous transluminal coronary angioplasty (PTCA) and stenting to left circumflex artery four months prior to the present symptoms. Patient was on aspirin, clopidogrel, atorvastatin and bisoprolol treatment. In the emergency room (ER) patient was conscious, afebrile with heart rate of 34 beats/minute, blood pressure 76/48 mmHg, Spo2 96 % on room air breathing and bilateral pitting type of pedal edema. Immediately ECG was done which revealed bradycardia with complete heart block (Figure-1). 2D ECHO suggestive of mild left ventricular

hypertrophy, mild pulmonary arterial hypertension ABG showed severe metabolic acidosis with high lactate levels. Labs revealed sodium -125 meq/l, potassium of 6.1meq/l, serum creatinine- 10.34mg/dl and blood urea-198 mg/dl. In view of bradycardia, renal failure, AV nodal blocker therapy (bisoprolol) and hyperkalemia he was diagnosed that the bradycardia is secondary to synergetic effect of renal failure, mild hyperkalemia and AV nodal blockers therapy. Patient was managed with boluses of and grade 1 left ventricular diastolic dysfunction. intravenous fluids, noradrenalin and hemodialysis. In spite of all these treatment the symptomatic hypotension persisted hence patient was shifted to cath lab and temporary pacemaker was inserted (Figure-2). After 3 cycles of hemodialysis and adequate fluid resuscitation his hemodynamics normalized. On third day the temporary pacemaker was removed and shifted to ward, later on 5th day patient was discharged home.

Fig 1. ECG showing bradycardia with complete heart block at the time of admission.

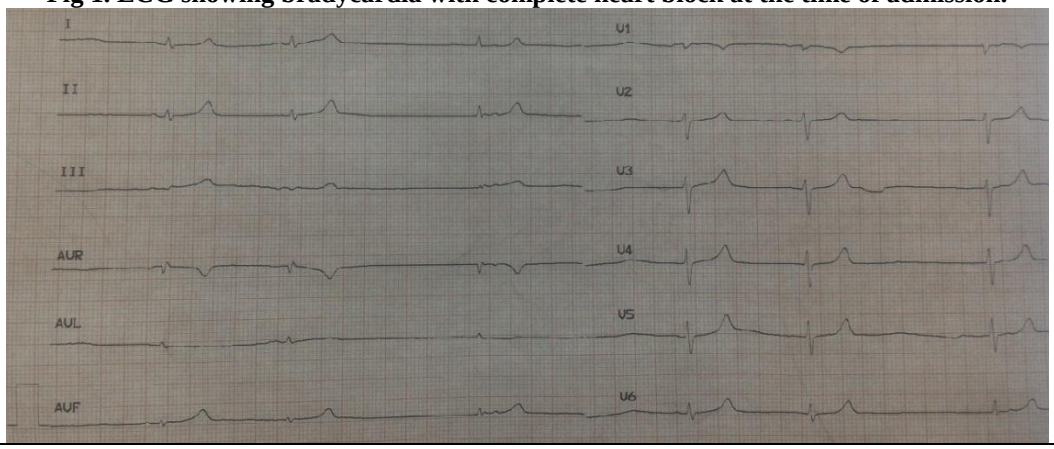
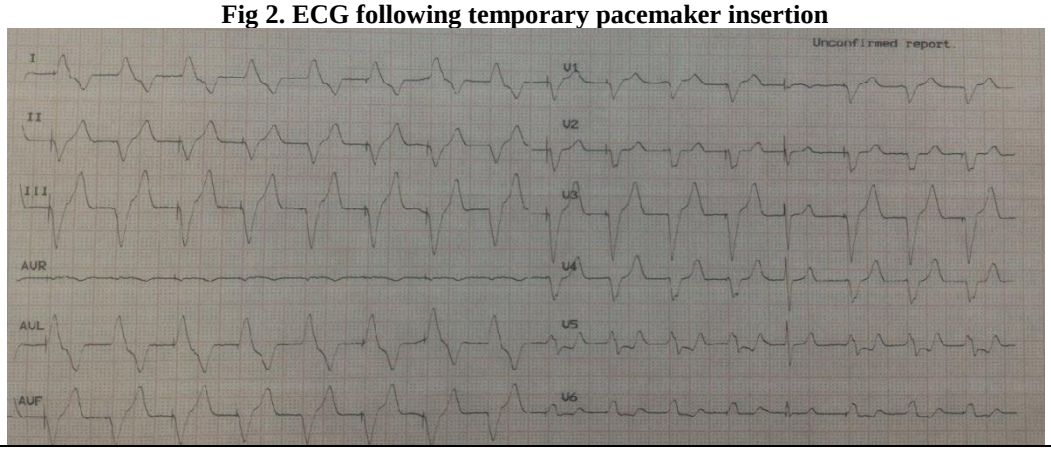


Fig 2. ECG following temporary pacemaker insertion



DISCUSSION

The synergetic components such as renal failure, cardiac disease and treatment with AV nodal blockers can cause bradycardia and complete heart block. These components can act as a tedious recipe for cardiac disaster [7]. This situation is not mainly due to hyperkalemia alone because in this situation, the potassium will be marginally elevated. These synergetic components we sometimes call in our ICU as? KRAB syndrome (Kalemia- Hyperkalemia, Renal failure, AV nodal blockers and Bradycardia) and should be suspected in patients on AV nodal blocker and recent derangement in renal functions. Hyperkalemia is known to cause conduction block in the heart. This is usually seen at higher concentration.

In our patient there was only marginal rise in serum potassium. The bradycardia was mainly due to accumulation of AV nodal blockers in the context of acute kidney injury.

Cardiac patients who are taking AV nodal blockers with borderline renal function are prone for this synergetic syndrome. These patients usually present with hypotension, chest pain, giddiness, breathlessness and decreased urine output. Management is usually supportive with fluid resuscitation, vasopressors, potassium lowering therapy (beta-2 agonist nebulisation [8], insulin

dextrose infusion [9] and sodium bicarbonate), cardiac membrane stabilizer (calcium gluconate) [10] exchange resin (sodium polystyrene sulfonate) [11] and hemodialysis [12]. Temporary pacemaker required in case of symptomatic hypotension and chest pain as in our case. This case report mainly highlights about a rare case of synergetic syndrome (KRAB syndrome). Early diagnosis and effective supportive treatment is all that isrequired for the successful management of these kinds of patients.

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DECLARATION OF INTEREST

None declared.

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