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Case Report

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Bradycardia, renal failure, atrioventricular nodal blockade, shock, and hyperkalemia: An important syndrome to recognize

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Abstract:

BRASH syndrome is a syndrome characterized by bradycardia, renal failure, usage of atrioventricular (AV) nodal blocker, shock, and hyperkalemia (BRASH). It is more common among patients with multiple comorbidities such as cardiac disease, kidney dysfunction, and hypertension requiring AV nodal blockers. Cardiac conduction abnormalities are frequently caused by severe hyperkalemia. However, it may also occur in mild-to-moderate hyperkalemia with concomitant use of AV nodal blockers due to the synergistic effects between these two factors in the presence of renal insufficiency. It is essential for the physician to identify BRASH syndrome as the treatment may differ from standard advanced cardiovascular life support (ACLS) protocol. We report the two cases of patient who presented with BRASH syndrome who failed to respond to standard ACLS protocol.

Keywords:

atrioventricular nodal blocker, bradycardia, hyperkalemia, renal failure, shock

Introduction

The term BRASH syndrome came to light in 2016 when Josh Farkas described the syndrome in an acronym representing bradycardia, renal failure, atrioventricular (AV) nodal blocking agents, shock, and hyperkalemia (BRASH) in Pulmcrit.[1] It is described as a vicious cycle of bradycardia and shock which are caused by the synergistic effect of hyperkalemia and accumulation of AV nodal blocker secondary to renal failure. The features of this syndrome were described in multiple literatures previously, but there was a lack of recognition of it as a distinct syndrome.[2-4] BRASH syndrome is often seen in patients with underlying cardiovascular disease and chronic renal failure that are put on long-term management with AV nodal-blocking agents. Among the precipitating factors identified are acute illnesses such as sepsis, hypovolemia, up-titration of home medications such as potassium sparing diuretics, and any other causes of acute kidney injury. It is important to recognize this clinical entity as standard advanced cardiovascular life support (ACLS) treatment may fail in reversing the bradycardia and shock seen in these patients.

Case Reports

Case 1

A 62-year-old female with diabetes mellitus, hypertension, and dyslipidemia presented to the emergency department with multiple

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episodes of syncopal attack. Further history revealed that she had the multiple episodes of vomiting and diarrhea prior to that. She denied chest pain, shortness of breath, or any other symptoms. She was on oral telmisartan 40 mg OD, atenolol 50 mg OD, diltiazem 60 mg TDS, frusemide 60 mg OD, metformin 1 g BD, aspirin 75 mg OD, and atorvastatin 20 mg QHS. She was compliant to her medications and there was no history of drug overdose. Upon presentation, the patient was drowsy with cold peripheries and poor pulse volume. Blood pressure was 105/41 mmHg, and heart rate was 40 beats/min (bpm). Other examination findings were unremarkable. Electrocardiogram (ECG) showed junctional bradycardia with a heart rate of 40 bpm [Figure 1]. Renal profile showed potassium: 6.3 mmol/l (normal value: 3.3–5.1 mmol/l), urea : 10.9 mmol/l (normal value: 1.7–8.3 mmol/l), and creatinine : 175 μmol/l (normal value: 60–120 μmol/l). Intravenous (IV) bolus of 20 ml/kg of normal saline was started, and two doses of IV atropine 0.5 mg were given but there was no improvement. We proceeded with giving 10 ml of IV calcium gluconate 10%, 50 ml of dextrose 50% and 10 unit of IV insulin infusion. She required catecholamine infusion with dopamine up to a dose of 20 μg/kg per minute and adrenaline of 5 μg/minute. After completing the first dextrose/insulin infusion, potassium from blood gas returned as 5.6 mmol/l. We proceeded with second round of dextrose/insulin infusion, and she showed marked improvements with a return to sinus rhythm and heart rate of 75 [Figure 2] subsequently. Catecholamine infusion was weaned off gradually. Repeated serum potassium returned to 3.5 mmol/l.

Case 2

The second case involved a 44-year-old lady with underlying chronic kidney disease, hypertension, and diabetes mellitus presented to us with an episode of syncopal attack. No seizure activity was observed. She denied chest pain, shortness of breath, diarrhea, or vomiting. Medication history revealed that she was on oral diltiazem 30 mg TDS, Furosemide 80 mg TDS, metoprolol 100 mg BD, felodipine 10 mg BD, spironolactone 25 mg BD, aspirin 75 mg OD, vidagliptin 50 mg OD, ferrous fumarate 200 mg BD, and Vitamin C 5 mg OD. Upon arrival, the patient was drowsy with GCS of E3V5M6, blood pressure of 88/52 mmHg, heart rate of 48 bpm, and saturation of 96% under room air. Pulse volume was poor, capillary refill time was 3 s, and peripheries were cold. Lungs examination was normal, and cardiovascular examination revealed no murmur. Neurological examination was unremarkable. ECG noted junctional escape rhythm with intermittent drop beat [Figure 3]. IV atropine was given up to 1.5 mg, and patient was put on dopamine infusion up to 20 μg/kg/min. In view of no improvements, patient was intubated and sedated for transcutaneous pacing (TCP). At this point, potassium result returned as 5.5 mmol/l (normal value: 3.3–5.1 mmol/l), and we proceeded with giving 10 ml of IV Calcium gluconate 10% with 50 ml of IV dextrose 50% and 10 unit of IV insulin infusion. Other significant investigations were increasing urea of 21.2 mmol/l from the baseline of 16.2 mmol/l (normal value: 1.7–8.3 mmol/l) and creatinine of 257 μmol/l from baseline of 132 μmol/l (normal value: 60–120 μmol/l). Cardiac enzymes, other electrolytes, liver function test, and full blood counts were all normal. The repeated potassium post treatment was 4.3 mmol/l and ECG returned to sinus rhythm [Figure 4]. Dopamine and TCP were weaned off rapidly, and patient was extubated the next day. Written informed consent were taken from both patients.

Discussion

The proposed pathophysiology of the above two cases is the ability of hyperkalemia to synergize the effect of AV nodal blockers leading to reduced chronotropy

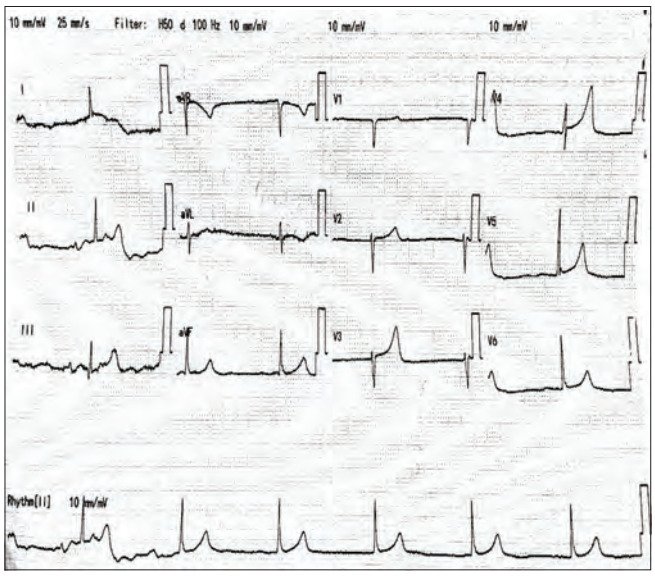

Figure 1: Electrocardiogram shows junctional bradycardia. Potassium level of 6.3 mmol/l

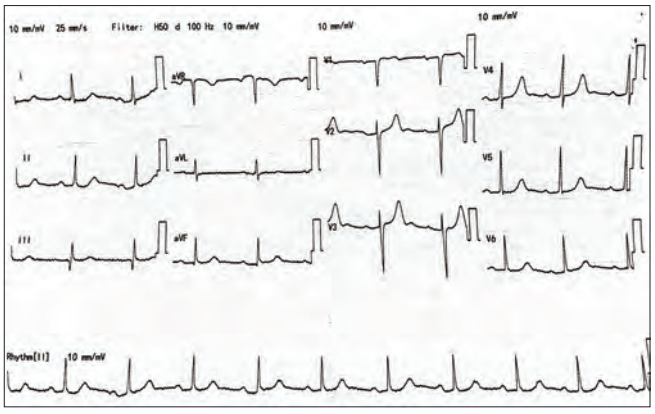

Figure 2: Electrocardiogram shows reversion to sinus rhythm post treatment. Potassium level of 3.5 mmol/l

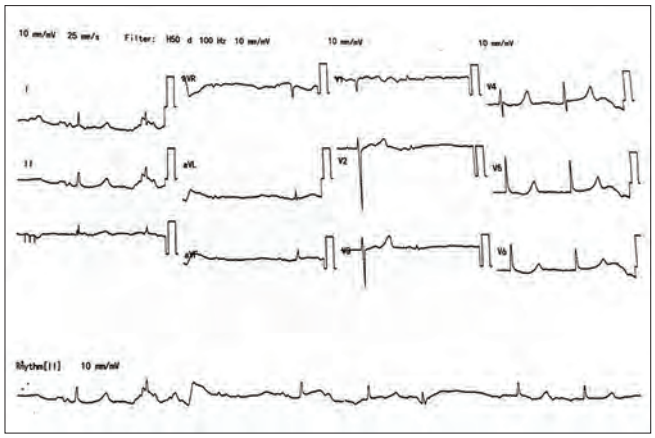

Figure 3: Electrocardiogram shows junctional escape rhythm with intermittent drop beat. Potassium level of 5.5 mmol/l

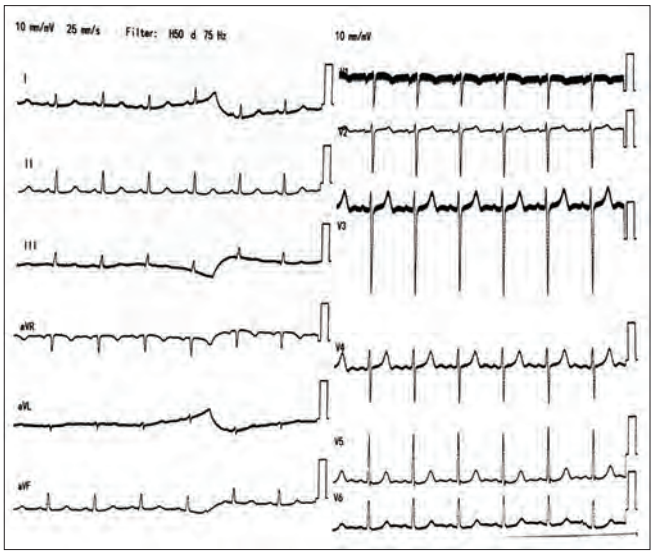

Figure 4: Electrocardiogram shows reversion to sinus rhythm posttreatment. Potassium level of 4.3 mmol/l

and hypotension. This leads to hypoperfusion and renal failure which may precipitate hyperkalemia and accumulation of drugs in the body forming a vicious cycle.[5] The effect of hyperkalemia can be reflected on ECG and the changes depend on the rate of rise and the absolute potassium level. The correlation between potassium level and ECG changes can be variable but most patients showed ECG abnormalities at serum potassium of 6.7 mmol/l.[6] Sinus arrest with absence of P-wave on ECG is a rare hyperkalemic changes because the fibers of sinoatrial node are more resistant to hyperkalemia as compared to atrial muscles. There may be numerous hyperkalemic ECG changes in moderate hyperkalemia (potassium 6–6.5 mmol/l) such as peaked T-wave, broad QRS and prolong PR interval but sinus arrest usually occur during severe hyperkalemia when the potassium level is nearing 8–9 mmol/l.[7] In BRASH syndrome, bradycardia occurs in mild or moderate hyperkalemia due to the synergistic effect between AV nodal blocker and hyperkalemia.[1] This is supported by a study by Hegazi *et al*.[8] showing that verapamil may induce junctional bradycardia in the presence of even mild hyperkalemia. Other distinguishing features between pure hyperkalemia and BRASH are the presence of junctional bradycardia in the absence of other hyperkalemic electrocardiographic signs.[3] Bonvini *et al*.[2] also showed that moderate hyperkalemia can present with sinus arrest without any other electrocardiographic signs in the presence of calcium channel blocker and beta blocker.

It is important to identify this clinical entity as standard ACLS algorithm involving giving atropine and cardiac pacing may not be effective in treating patients with BRASH syndrome. The treatment should aim at treating hyperkalemia, identify and treat the cause of kidney injury and occasionally treating beta-blocker or calcium channel blocker toxicity. Hyperkalemia should be treated with IV calcium for cardiac membrane stabilization and IV insulin and glucose to shift potassium intracellularly. Other modalities such as albuterol may be useful in treating bradycardia as well. As BRASH patient frequently presented with hypovolemia, fluid replacement is paramount for these patients. Care has to be taken for patients who are in anuric renal failure as they can be volume overload. Kaliuresis with potassium-wasting diuretics such as furosemide can be considered to facilitate urinary excretion of potassium. Emergency dialysis should be given if above measure fails. Catecholamine infusion should be started if the patient is persistently unstable hemodynamically.[1,5] This is important to maintain adequate renal perfusion while waiting for the accumulated drugs to be excreted.[9] Among the choices of catecholamine are epinephrine, dopamine, and isoproterenol. Treatment of beta-blocker or calcium channel intoxication with high-dose insulin and glucagon may be necessary occasionally.

Conclusion

These two cases highlight the importance of diagnosing BRASH syndrome, especially in populations with multiple comorbidities. Understanding the pathophysiology of the disease can assist the attending physician to treat these patients more effectively. The treatment of BRASH syndrome differs from standard ACLS algorithm, and it may lead to overutilization of cardiac pacing if we follow the ACLS algorithm blindly.

Author contribution statement

WCK contributed to clinical management of the patients, writing of the manuscript, approval of the final draft of the manuscript and corresponding author.

MJJ contributed to support of the literature, revision and approval of the final draft of the manuscript.

Conflicts of interest
None declared.

Consent to participate
Written informed consent was obtained from the patient for her/his anonymized information to be published.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images, and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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