A case of unstable bradycardia requiring comprehensive management in the emergency department: BRASH syndrome

Sinan Genc, Omer Yusuf Erdurmus, Abdullah Erhan, Ahmet Burak Oguz, Ayca Koca, Muge Gunalp Eneyli, Onur Polat

Ankara University School of Medicine Department of Emergency Medicine, Ankara, Turkey

Abstract

Bradycardia, renal failure, Atrioventricular (AV) nodal Blocker Drug Use, Shock, and Hyperkalemia (BRASH) syndrome is a clinical condition frequently seen in emergency services but with low diagnostic awareness. In cases of the syndrome, its cause was determined to be the synergistic effect of hyperkalemia due to renal failure and the use of AV nodal blocker drugs. The common features of patients diagnosed with BRASH syndrome are moderately elevated potassium levels and symptomatic bradycardia with various ECG findings (such as junctional bradycardia, atrioventricular block, and sinus bradycardia). Detection of these findings is very important in the diagnosis process. In this case report, we aimed to reveal the important points in the diagnosis of BRASH syndrome, ECG findings, and treatment approach.

Correspondence: Omer Yusuf Erdurmus, Ankara University School of Medicine Department of Emergency Medicine, Samanpazarı-Ankara, Turkey.
Tel.: +903125083143; Fax: +903125083032
E-mail: yerdurmus@gmail.com

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Introduction

Bradycardia, renal failure, Atrioventricular (AV) nodal Blocker Drug Use, Shock, and Hyperkalemia (BRASH) syndrome was coined by Josh Farkas in 2016.[1] In this syndrome, effects of shock and bradycardia on the body caused by hyperkalemia, renal failure and accumulation of AV nodal blocker drug are seen.[2] Although case reports and case series about this syndrome are present in the literature, there are unexplored aspects of BRASH syndrome in the diagnosis and treatment stages.[3] In this case report, we present a patient who applied to the emergency department with bradycardia and shock findings and who was diagnosed as having and treated for BRASH syndrome.

The unique treatment of BRASH syndrome, which can show symptoms similar to those of isolated hyperkalemia and isolated AV nodal blocker intoxication, is due to the physiopathology of the syndrome.[2] It results from a vicious cycle between nodal blocker drugs, hyperkalemia, and renal failure. Although renal failure causes hyperkalemia, it can also cause an accumulation of nodal blockers. Hyperkalemia and nodal blocker drugs cause bradycardia and hypoperfusion with a synergistic effect, with the severity of the underlying renal failure progressing after the hypoperfusion.[1] However, the degree of hyperkalemia in BRASH is not related to the severity of the syndrome and may lead to bradycardia and various Ecg findings regardless of the level.[4]

Case Report

An 81-year-old male patient presented at emergency services with sweating, weakness, and syncope for 2 days. Coronary artery disease and hypertension were found in the patient's history. The patient was using metoprolol (50 mg twice daily), acetylsalicylic acid (100 mg once daily), and amlodipine (10 mg once daily) as medical therapy.

During examination, the patient was cooperative and oriented. He had a heart rate of 50 beats per minute, a body temperature of 36.2°C, a blood pressure of 77/42 mmHg, a respiratory rate of 16 breaths per minute, and a peripheral capillary oxygen saturation on room air of 94 percent. His Glasgow Coma Scale score was 15. No

focal neurologic deficit was observed. Cardiovascular and respiratory system examinations were unremarkable. The patient was placed in a bed with a monitor, and isorhythmic dissociation was seen on the ECG (Figure 1). There was no pathological feature in bedside ultrasonography.

In laboratory analysis, serum creatinine level was 2.09 mg/dL, blood urea nitrogen level was 38 mg/dL, potassium level was 7.7 mEq/L, troponin T level was 21.5 pg/mL, and lactate level was 1.8 mmol/L. However, the high level of Lactate Dehydrogenase (LDH) in the blood was re-evaluated with the thought that the blood products taken may have undergone hemolysis. Potassium level was determined as 5.7 mEq/L, and there was no significant change in other results. The basal serum creatinine value could not be determined in the patient's hospital registry system, and the value measured in the cardiology outpatient clinic one day prior was determined as 1.9 mg/dL. Upon arterial blood gas evaluation, a pH of 7.30, partial pressure of oxygen (PaO_2) of 75 mmHg, partial pressure of carbon dioxide ($PaCO_2$) of 32 mmHg, bicarbonate (HCO_3) levels of 19.2 mmol/L, base excess of −3.2 mmol/L, anion gap of 14 mEq/L, Na+ levels of 138 mmol/L, Cl– levels of 99 mmol/L, and blood glucose levels of 103 mmol/L were found. One liter of normal saline, 10 mL of intravenous calcium gluconate (10%), 10 units of insulin, 500 mL of 5 percent dextrose, and 20 mg of nebulized salbutamol were administered intravenously to the patient in 3 minutes. Following anti hyperkalemic therapy, the ECG returned to normal sinus rhythm, with a heart rate of 92 beats per minute (Figure 2). The patient's blood pressure was 105/68 mmHg. The patient, whose symptoms diminished after medical treatment and whose vital signs were stable, was then discharged. Written informed consent was obtained from the patient for the purpose of the case report.

Discussion

BRASH syndrome typically results from the synergistic effect between hyperkalemia and AV nodal blocker drugs, resulting in symptomatic bradycardia and shock.[1] When the cases in the literature were evaluated, the use of AV nodal blocker drugs such as verapamil, diltiazem, carvedilol, and metoprolol, which was also seen in our case, was more common in this syndrome.[3] Bradycardia may impair renal perfusion and thus cause renal failure. This cycle can progress to multi-organ failure.[5] In cases seen after our literature review, BRASH syndrome was detected mostly in people with reduced kidney function, as in our case. The hypotension seen in the syndrome results in decreased renal perfusion and worsening of hyperkalemia.[6]

Hyperkalemia results in various ECG findings after decreased cardiac conduction velocity (such as junctional bradycardia, atrioventricular block, and sinus bradycardia).[3] However, a direct correlation between the severity of hyperkalemia and that of the syndrome has not been established, and this situation is used in the differential diagnosis of BRASH syndrome.[7] ECG findings (peak T wave, prolongation of the PR interval, prolongation of the QT interval and QRS enlargement) that can be seen in isolated hyperkalemia may not be seen in BRASH syndrome, they alone are insufficient in diagnosing.[8] This syndrome can lead to bradycardia and various Ecg findings regardless of the degree of hyperkalemia.[4]

Patients in BRASH syndrome case reports generally presented at the emergency department with complaints of shortness of breath, discomfort, dizziness, syncope, fatigue, and weakness, and these symptoms are strongly thought to be caused by hypotension, shock, and bradycardia due to the mechanism of the syndrome.[3] Decompensated heart failure findings, such as pretibial edema, jugular venous blood flow, exertional dyspnea, orthopnea, cyanotic appearance, hypoxia, and a prolonged capillary refill time, are often seen upon physical examination.[9]

Early diagnosis and initiation of correct treatment by emergency physicians are crucial in BRASH syndrome.[10] It is important to determine this clinical situation, since atropine and pacemaker applications, included in the standard bradycardia algorithms, may not be effective in the treatment of patients with BRASH syndrome.[11] In the treatment steps, the AV nodal blocker drug should be stopped first.[1] In patients with high levels of hyperkalemia, IV calcium, which provides cardiac stabilization, should be initiated together with treatments that lower potassium levels (e.g., albuterol and IV insulin).[3] Facilitating the urinary excretion of potassium with potassium-losing diuretics, such as furosemide, may also be considered during treatment. In cases demonstrating resistance to medical treatment, hemodialysis is also among the treatment options to normalize the K+ levels of patients.[10] Caution

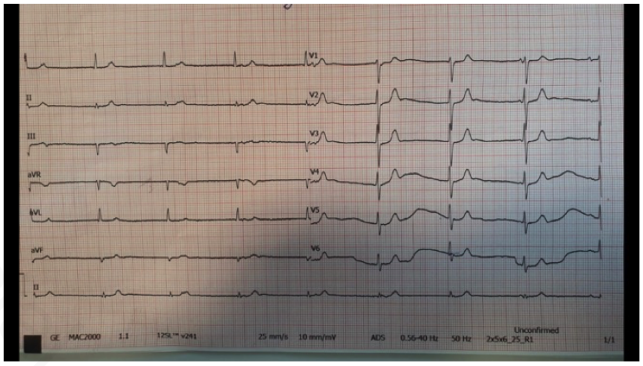

Figure 1. Isorhythmic dissociation as an ECG finding.

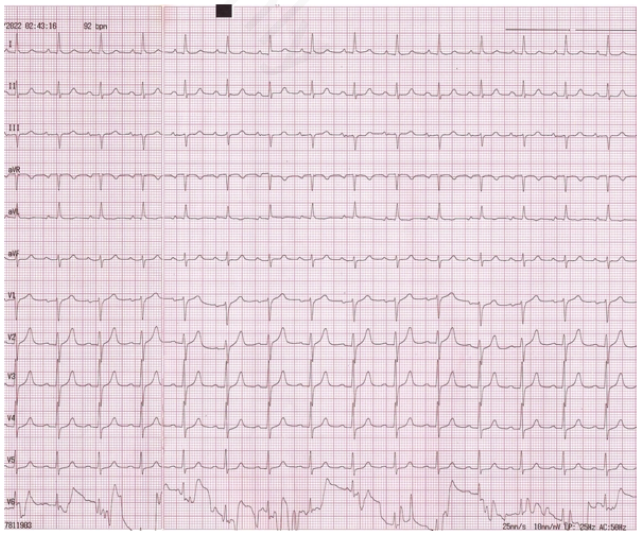

Figure 2. Normal sinus rhythm as an ECG finding.

should be exercised for patients with renal insufficiency, as excessive fluid overload may occur during medical treatment.[12] If the patient is not hemodynamically stable, adding catecholamine infusion (epinephrine, dopamine, etc.) to the treatment may contribute to both hemodynamic and adequate renal perfusion.[13]

Conclusions

In our case report, the importance of the correct diagnosis of BRASH syndrome and the correct treatment was emphasized. It is predicted that BRASH syndrome cases will be seen more frequently due to both the increasing use of drugs that are blamed in the etiology and the increasing elderly population. In BRASH syndrome, which can be diagnosed by a comprehensive evaluation together with the findings in the ECG and the patient's clinic, early initiation of the correct treatment is of vital importance for the patients, as seen in our case.

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