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BRASH syndrome in the elderly: A case report

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ABSTRACT

Background. Bradycardia, shock, renal failure, and hyperkalemia in patients with atrioventricular nodal block are rare in clinical practice. These features were not referred to as a syndrome until 2016, under the term BRASH syndrome. Early recognition of this condition and a systematic approach may reduce the need for invasive interventions and help decrease mortality.

Case presentation. A 79-year-old female patient diagnosed with chronic atrial fibrillation and heart failure presented to the emergency department with chest pain, followed by loss of consciousness and shock. Laboratory tests revealed hyperkalemia and acute kidney injury, and ECG showed atrial flutter with a very slow ventricular rate response. The patient was immediately treated with adrenaline, sodium bicarbonate, fluid replacement, and antibiotics. Her condition improved after four days, and she was discharged after sixteen days without cardiac pacing or renal replacement.

Conclusion. Physicians should maintain a high suspicion of BRASH syndrome when approaching a patient with bradycardia, hyperkalemia, and renal failure. Additionally, dehydration should always be considered when evaluating renal failure and infection in a diuretic-treated patient with heart failure. Invasive therapies can be avoided with appropriate management of BRASH syndrome.

Keywords: shock, bradycardia, hyperkalemia, renal insufficiency, AV nodal blocker

Abbreviations (in alphabetical order):

AV Block – atrioventricular nodal block
ECG – electrocardiogram
IVC – inferior vena cava
POCUS – point-of-care ultrasound

INTRODUCTION

BRASH syndrome is characterized by the presence of bradycardia, renal failure, atrioventricular (AV) nodal block, shock, and hyperkalemia [1,2]. Bradycardia caused by AV block can reduce cardiac output, leading to decreased renal perfusion and pre-renal failure. This, in turn, results in hyperkalemia, which exacerbates bradycardia, creating a vicious cycle that may lead to life-threatening multi-organ dysfunction. Although BRASH syndrome primarily affects elderly patients, it can also occur in younger individuals [3]. Prompt recognition of hyperkalemia and accurate diagnosis of pre-renal failure are critical in managing BRASH syndrome. Early intervention can reduce the need for cardiac pacing and renal replacement therapy, and may decrease mortality, particularly in resource-limited settings.

CASE REPORT

A 79-year-old female presented to the emergency department with chest pain followed by a loss of consciousness over the past three hours. Her medical history included hypertension, atrial fibrillation, and heart failure, which had been ongoing for five years.

She was taking bisoprolol 2.5 mg once daily, furosemide 40 mg twice daily, atorvastatin 20 mg once daily, and losartan 50 mg once daily. Her son reported that she had experienced poor appetite and an absence of urination for the past three days. On arrival, her ECG showed atrial flutter with a very slow ven-

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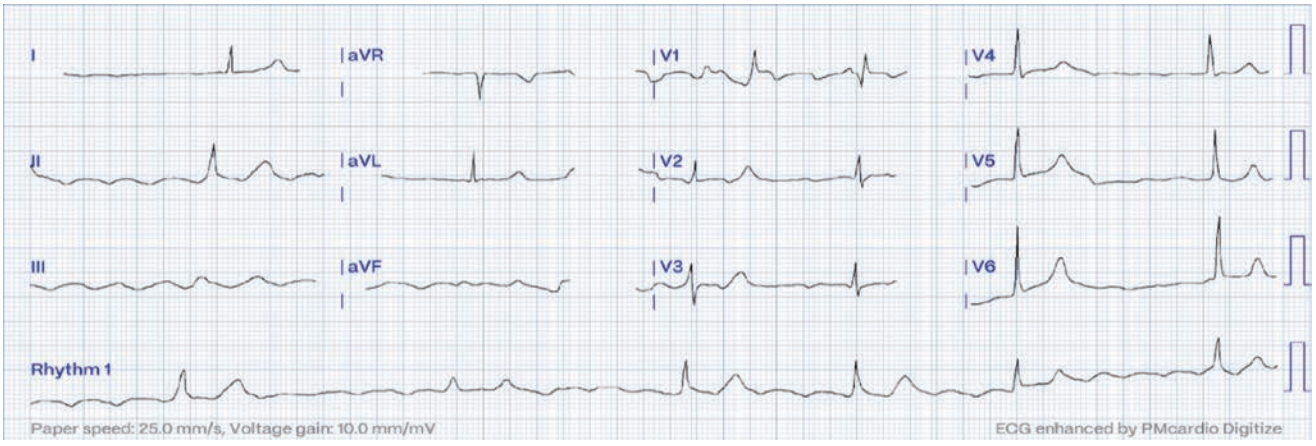

FIGURE 1. ECG at admission showed atrial flutter with a slow ventricular response. The tall T waves may indicate hyperkalemia.

tricular rate of 30 beats per minute (Figure 1), tall T waves, a blood pressure of 80/50 mmHg, a capillary refill time of four seconds, and oxygen saturation of 90%. Point-of-care ultrasound (POCUS) revealed an IVC diameter of 10–15 mm, no pericardial effusion, and an ejection fraction of 50% using the Simpson method.

The initial diagnosis was bradycardia with shock and suspected hyperkalemia in the setting of acute kidney injury. She was immediately treated with adrenaline (3 mg in 50 mL normal saline via infusion pump at 3 mL/h) to address shock and bradycardia, and with 4.2% sodium bicarbonate (2,500 mL per day) to rapidly expand intravascular volume and lower potassium levels. Laboratory tests were initiated. Her family declined the option of temporary pacemaker insertion.

After initial treatment, the patient regained consciousness and her hemodynamic status stabilized. She continued receiving adrenaline and intravenous fluids.

One hour later, laboratory results showed:

- Sodium: 119 mmol/L
- Potassium: 5.5 mmol/L
- Calcium: 2.2 mg/dL
- Urea: 37.5 mmol/L
- Creatinine: 353 µmol/L
- Albumin: 3.1 mg/dL
- Troponin: 17 pg/mL
- NT-proBNP: 21,000 pg/mL
- CRP: 168 mg/L
- WBC: 22.6 × 10^9/L (Neutrophils 87%)
- RBC: 3.6 × 10^{12}/L (Hemoglobin 104 g/L)
- pH: 4.9
- pCO_2: 26 mmHg
- pO_2: 68 mmHg
- SaO_2: 95% (FiO_2 30%)

No urinalysis was performed. The diagnosis of BRASH syndrome (bradycardia, renal failure, AV nodal block, shock, hyperkalemia) was confirmed, along with hyponatremia and community-acquired pneumonia.

After five hours in the emergency department, she was transferred to the Cardiology Department. On examination: temperature 39°C, respiratory rate 26/min with crackles in the right lung, SpO_2 92% (room air), blood pressure 100/70 mmHg, and an irregular heart rate of approximately 70 bpm. She continued receiving 4.2% sodium bicarbonate 500 mL IV, 5% glucose 500 mL IV, and adrenaline (3 mg/50 mL saline at 1 mL/h via infusion pump).

Her CHA_2DS_2-VASc score for stroke risk in atrial fibrillation was 4. Acenocoumarol 1 mg (¼ tablet) was initiated. Broad-spectrum antibiotics—cefoperazone/sulbactam and moxifloxacin—were started, considering her impaired renal function and a CURB-65 score of 4. A urinary catheter was placed, and the patient was advised to drink 2,000 mL of water per day. Her urine output reached 1,000 mL/day, and fluid balance remained positive.

Follow-up labs confirmed renal failure and hyponatremia:

- Urea: 37.6 mmol/L
- Creatinine: 301 µmol/L
- Sodium: 126 mmol/L
- Potassium: 4.6 mmol/L
- Chloride: 101 mmol/L.

Day 2 (Figure 2):

- Urea: 12 mmol/L
- Creatinine: 119 µmol/L
- Sodium: 127 mmol/L
- Potassium: 3.3 mmol/L
- Chloride: 100 mmol/L.

Blood pressure was 100/70 mmHg, and ECG showed atrial fibrillation with a ventricular rate of 90 bpm. Adrenaline was discontinued.

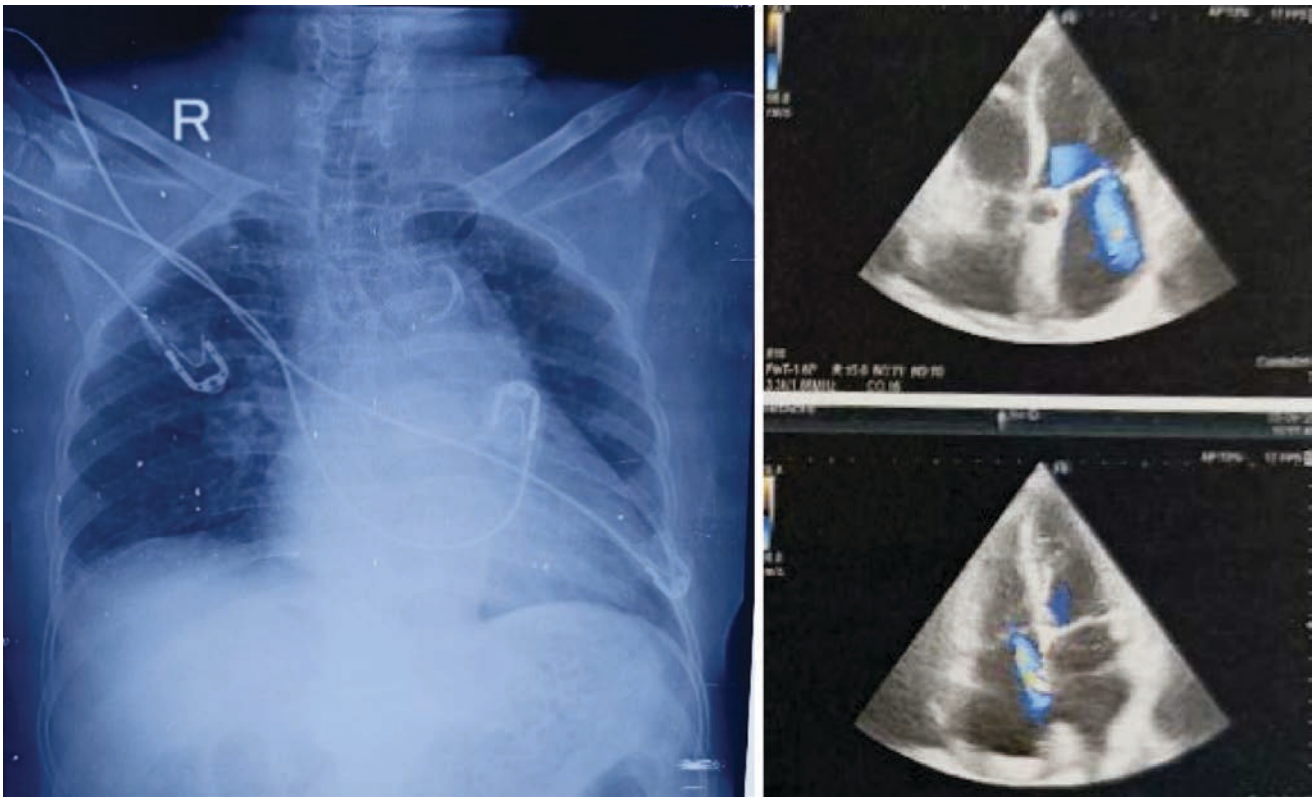

FIGURE 2. Chest X-ray and echocardiography on day 2. The anteroposterior chest X-ray showed an enlarged cardiac silhouette without pleural effusion. Echocardiography demonstrated a left ventricular ejection fraction of 50% (Simpson method), dilation of both the right and left atria, mitral regurgitation, tricuspid valve regurgitation, and a pulmonary artery pressure (PAPS) of 48 mmHg.

Day 4:

- Urea: 5.7 mmol/L
- Creatinine: 80 µmol/L
- Sodium: 131 mmol/L
- Potassium: 3.4 mmol/L
- Chloride: 100 mmol/L

NT-proBNP had decreased significantly to 3,176 pg/mL.

By day six, the patient was stable.

The discharge diagnoses were BRASH syndrome, community-acquired pneumonia, and chronic atrial fibrillation with moderate ventricular response (Figure 3).

DISCUSSION

We report a case of BRASH syndrome in an elderly patient with heart failure who was being treated with beta-blockers, ACE inhibitors, and diuretics. She presented to the emergency department with bradycardia and subsequent shock.

The ECG at admission revealed tall T waves with prominent F waves. QRS complexes were narrow, with no Q waves, no ST elevation, and a prolonged

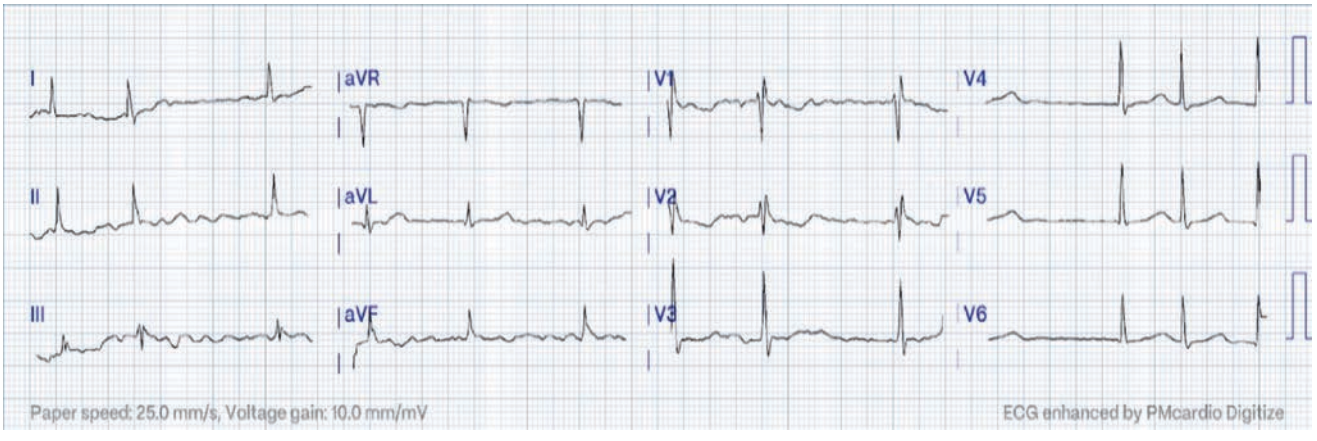

FIGURE 3. ECG at discharge. The electrocardiogram showed atrial fibrillation with a ventricular response rate of 70 beats per minute.

QT interval. These findings are consistent with atrial flutter with a slow ventricular response [4] and suggestive of hyperkalemia. Recognizing tall T waves on ECG in the setting of bradycardia is crucial for the early diagnosis of BRASH syndrome. Differentiating atrial flutter from sick sinus syndrome is also important, as atrial flutter necessitates anticoagulation. A slow ventricular response in BRASH syndrome should prompt consideration of differential diagnoses, including AV nodal blocker overdose and hyperkalemia.

In this case, AV nodal blocker overdose was unlikely, given the patient's long-term use of bisoprolol at a low dose of 2.5 mg once daily. This dose rarely causes significant bradycardia, and the patient's consistent medication adherence supports the exclusion of overdose [5].

Hyperkalemia usually causes ECG changes, with levels between 5.5 and 6.5 mmol/L typically producing tall T waves, and levels above 8 mmol/L leading to arrhythmias, sine wave patterns, or asystole [6]. In BRASH syndrome, however, bradycardia can occur at lower potassium levels due to the synergistic effect of beta-blockers [7]. In this patient, the serum potassium level was 5.5 mmol/L—mild hyperkalemia, yet significant in the context of concurrent beta-blockade and renal dysfunction.

The combination of hyperkalemia, acute kidney injury, and even a low dose of AV nodal blocker contributed to worsening bradycardia. Point-of-care ultrasound (POCUS) showed an IVC diameter of 10–15 mm, no pericardial effusion, and an ejection fraction of 50% by the Simpson method. This suggested that shock was caused primarily by bradycardia and dehydration, likely due to diuretic use, poor oral intake, and infection. This case underscores the importance of considering intravascular volume depletion in elderly patients with infection and cognitive impairment [8].

Alongside volume resuscitation, discontinuing beta-blockers and ACE inhibitors was essential to improve cardiac output and renal perfusion in this context of bradycardia, hyperkalemia, and dehydration.

In this patient, hyperkalemia combined with hyponatremia was quickly treated with sodium bicarbonate. This solution shifts potassium from the blood to the cells and provides a sufficient amount of sodium to correct hyponatremia [9]. Additionally, sodium bicarbonate has been shown to improve creatinine clearance in patients with acute kidney injury [10]. At the same time, sodium bicarbonate provides fluid to expand circulating volume. In summary, it was the most appropriate treatment in this clinical scenario, as it addressed four key issues simultaneously: (1) reducing serum potassium, (2) increasing serum sodium, (3) correcting dehydration, and (4) improving renal function. This highlights an important difference from other reported cases of BRASH syndrome, in which sodium bicarbonate was not administered promptly upon suspicion of hyperkalemia. Sodium bicarbonate is an effective, inexpensive, and safe intervention. We propose that, despite its age, sodium bicarbonate remains a valuable therapeutic option and should be considered a first-line treatment for BRASH syndrome, especially when hyperkalemia is accompanied by hyponatremia.

According to the AHA, treatment options for bradycardia-induced shock include adrenaline, dopamine, and transvenous pacing. In this case, adrenaline was preferred due to its rapid onset, positive chronotropic effect, and ability to improve blood pressure and renal perfusion [11,12]. Dopamine, by contrast, has a slower onset and requires careful titration, with higher doses risking vasoconstriction and arrhythmias.

Importantly, adrenaline also helps shift potassium into cells. Standard algorithms for hyperkalemia and bradycardia—such as calcium gluconate and atropine—may be ineffective in BRASH syndrome. Calcium offers only temporary stabilization of myocardial membranes and does not treat bradycardia, while atropine is often ineffective because BRASH-related bradycardia is not vagally mediated [5]. Delays caused by reliance on these less effective interventions can postpone definitive therapy with adrenaline.

BRASH syndrome's pathophysiology differs from isolated hyperkalemia or AV nodal blockade and therefore requires a tailored therapeutic strategy. In this case, the combination of sodium bicarbonate and adrenaline was effective in restoring renal function and normalizing potassium levels.

Prompt recognition and treatment prevented the need for transvenous pacing. The patient stabilized quickly and was discharged on day 14. Beta-blockers and ACE inhibitors were temporarily withheld.

In the month following discharge, the patient was treated with rivaroxaban 20 mg once daily, pantoprazole 40 mg, losartan 50 mg, and a low-salt diet. Due to her history of dehydration, special care was taken with diuretic use. At the first follow-up, her blood pressure was 135/80 mmHg, heart rate was 80–100 bpm, and renal function remained stable. To minimize the risk of BRASH recurrence, we plan to replace furosemide with a sodium–glucose cotransporter-2 (SGLT2) inhibitor and spironolactone at the next follow-up if shortness of breath recurs.

CONCLUSION

BRASH syndrome is a life-threatening condition characterized by bradycardia, atrioventricular (AV)

nodal block, renal insufficiency, shock, and hyperkalemia. It is crucial for healthcare providers to understand the pathophysiology of this syndrome and maintain a high level of suspicion in patients with bradycardia and mild hyperkalemia, especially those on AV nodal blocking agents. This case highlights two key learning points: (1) Elderly patients with infections and poor appetite are at high risk of dehydration, and the use of diuretics can trigger or exacerbate BRASH syndrome. Therefore, assessing volume status and identifying factors that reduce circulating volume are critical for preventing further deterioration and improving patient outcomes; (2) The effective use of bicarbonate and adrenaline can lead to rapid recovery from BRASH syndrome without the need for pacing.

Ethical approval:
Not applicable. Written informed consent for the publication of this case report was obtained from the patient.

Availability of data and materials:
The data presented in this case report are available upon reasonable request.

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Conflict of interests:
The authors declare no conflicts of interest related to this manuscript.

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