Too slow and too high

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Abstract
A 77-year-old woman on metoprolol and lisinopril presented to an emergency department with giddiness after vomiting for few hours. She was found to have low blood pressure and bradycardia 38 beats per minute due to atrioventricular nodal blockade. Her bradycardia was refractory to atropine and dopamine infusion; but improved with calcium gluconate. She was found to have acute kidney injury and hyperkalemia at 6.4 mEq/L. This is a case of Bradycardia, Renal Failure, Atrioventricular-Nodal Blockers, Shock, and Hyperkalemia (BRASH) syndrome, precipitated by dehydration and perpetuated by atrioventricular blockade, illustrating the degree of bradycardia and electrocardiographic changes being out of proportion to the potassium level. BRASH syndrome should be recognized and intervened early in the course to avoid the patient entering a vicious cycle that could be rapidly fatal.

Keywords
Electrocardiography, renal failure, hyperkalemia

Case presentation
A 77-year-old woman presented to a local emergency department with giddiness. Her past medical history included atrial fibrillation on metoprolol for rate control, hypertension on lisinopril, and impaired fasting glucose not on medication. She had poor oral intake for several days and subsequently developed vomiting plus retrosternal chest discomfort for several hours, thus called the ambulance. She reported good compliance to her prescribed medications.

Her first blood pressure at triage was 76/30 mmHg and heart rate 38 beats per minute (bpm). She appeared tired but remained conscious and orientated. Fluid resuscitation was started immediately, and an electrocardiogram (ECG) was done (Figure 1).

Question 1
How do you interpret this ECG?

Answer 1
Atrial flutter waves could be seen, with a regular 6:1 conduction leading to bradycardia of approximately 36 bpm.

The T waves were prominent, with an amplitude comparable or larger than that of the QRS complex in some leads. This is highly suggestive of hyperkalemia.

The emergency physician (EP) first attempted to treat the bradycardia with atropine and followed by dopamine infusion in a moderately high dose, but both medications failed to increase the heart rate. Subsequently, in view of the high suspicion of hyperkalemia and the patient being in shock from high degree heart block, a dose of calcium gluconate was administered, which within minutes improved the patient’s hemodynamics with the prominent T waves resolved (Figure 2) and dopamine weaned off. The blood tests coming back later showed acute kidney injury (AKI) with creatinine level at around 300 µmol/L (baseline level was normal) and potassium level at 6.4 milliequivalents per
liter (mEq/L). The patient was admitted to the cardiac care unit (CCU) for close monitoring, in which her vital signs remained stable. Her AKI resolved with rehydration, chest discomfort subsided, and her troponin levels did not ascend.

**Question 2**
What were the predisposing and precipitating factors for the patient to present with this arrhythmia?

**Answer 2**
The predisposing factors included being on beta blocker and angiotensin-converting enzyme inhibitor. The AKI was most likely precipitated by dehydration from vomiting.

**Discussion**
This is a typical presentation of Bradycardia, Renal Failure, Atroventricular (AV)-Nodal Blockers, Shock,

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**Figure 1.** The patient’s first ECG.

**Figure 2.** ECG after administration of calcium gluconate. Note that the prominent T waves in Figure 1 had resolved.
and Hyperkalemia (BRASH) syndrome, which is a name coined only recently.2–4 “BRASH” is the acronym of what constitute the syndrome, namely, Bradycardia, Renal failure, AV blockers, Shock and Hyperkalemia.2 This condition is not rarely encountered in the emergency department. However, the lack of awareness of how this particular constellation can create a vicious cycle makes this syndrome under-represented in the literature. Its pathophysiology was hypothesized to that once a certain degree of hypoperfusion was caused, most often precipitated by dehydration or sepsis, the AV blockers limit the patient’s heart rate and thereby further exacerbate the hypoperfusion.3,4 Hyperkalemia can lead to bradycardia or heart block by increasing membrane repolarization reserve and prolonging effective refractory period.1 Consequently, the cardiac tissue is less responsive to catecholamines, and in BRASH syndrome, this effect is further potentiated by AV blockers.3,4 The degree of hemodynamic compromise and ECG changes are often out of proportion to the patient’s potassium level,5,6 as in the above case where one may not expect such dramatic ECG findings and profound bradycardia with a potassium level of 6.4 mEq/L. BRASH syndrome, in its more severe form, can definitely lead to refractory shock and therefore should be recognized and intervened early in the course. Management includes stopping the offending agents and treating hyperkalemia and the underlying cause, for example, by rehydration. In cases with refractory shock or bradycardia, transvenous pacing and inotrope support may be needed.7 In conclusion, EPs should be competent in recognizing classical ECG features of hyperkalemia and should be aware that patients on AV blockers are particularly vulnerable to dehydration which could rapidly lead to renal injury and hemodynamic compromise from the consequent electrolyte disturbance.

Author contribution
All authors contributed equally to this manuscript.

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Human rights
The data and images obtained were in accordance with the principles outlined in the Declaration of Helsinki.

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