A VICIOUS CYCLE: BRASH SYNDROME

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Category: Case Vignettes

Introduction: BRASH syndrome consists of bradycardia, renal dysfunction, the use of AV nodal blocking agents, shock, and hyperkalemia. This is an under-recognized condition that can result in multi-organ failure if not accurately diagnosed and treated.

Case: An 88-year-old female presented to the emergency department from a skilled nursing facility after being found bradycardic with a heart rate (HR) in the 30s. The patient has a history of stage IIIb chronic kidney disease, paroxysmal atrial fibrillation, hypertension, dyslipidemia, and Bell's Palsy. In the ED, she was noted to be hypothermic to 92.1 F, bradycardic with HR in the 30s, hyperkalemic with potassium of 7.2, and noted to have an AKI (baseline creatinine was around 1.5, and presented with a creatinine of 2.1). She was given calcium chloride, insulin with dextrose, sodium bicarbonate, Kayexalate, and glucagon. Her home medications Carvedilol and Amlodipine were held. She had minor improvement of her bradycardia with HR reaching the 50s, however the next day her bradycardia worsened back to the 30s. She was also noted to be encephalopathic and was intubated at this time for airway protection and given atropine with improvement in heart rate. She was subsequently extubated after one day and noted to have improvement of her bradycardia with heart rates sustaining in the 50s and 60s without any further interventions after treatment of hyperkalemia and discontinuation of home medication Carvedilol. All AV nodal blocking agents were held throughout the remainder of her admission, and she was discharged with kidney function back to baseline and heart rate in the 70s.

Discussion: BRASH syndrome is a relatively new diagnosis, with the acronym BRASH first coined in 2016. It is most often recognized in elderly patients with chronic kidney disease. The synergistic mechanism between hyperkalemia, worsening renal function, and accumulating AV nodal blocking agents is what results in bradycardia and hypotension/shock seen with BRASH. Although AV nodal blocking agents are held when the patient is initially bradycardic, renal failure will result in prolonged time to metabolize and excrete these agents, resulting in persistent bradycardia. The bradycardia and shock results in hypoperfusion which then worsens the renal failure and exacerbates the hyperkalemia, which leads to a cycle of clinical deterioration if intervention is not taken. In BRASH syndrome, it is essential to recognize that relatively mild hyperkalemia may result in severe bradycardia and possible heart block due to increased accumulation of AV nodal blocking agents.

Conclusion: This case highlights the importance of recognizing possible iatrogenic consequences of AV nodal blocking agents in patients with chronic kidney disease and keeping BRASH in our differential.