A Surprising Spiral of Spironolactone into BRASH

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Case Description:

We present a 66-year-old male with a history of untreated chronic hepatitis C, decompensated cirrhosis, chronic kidney disease stage 3a, and peripheral artery disease who presented to the emergency department with generalized body aches and shortness of breath for the last 5 days. On arrival, he was found to be hypotensive (81/46 mmHg) and bradycardic with a heart rate of 43 bpm. Laboratory studies were notable for severe hyperkalemia (serum potassium 8.2 mmol/L) and an acute kidney injury with a creatinine elevation to 2.46 mg/dL from his baseline of 1.1 mg/dL. Electrocardiogram demonstrated sinus arrest with junctional escape complexes. The patient was admitted to the medical intensive care unit (MICU) and underwent emergent dialysis with rapid improvement and subsequently stepped down from the intensive care unit on day 2 of hospitalization. He was diagnosed with BRASH syndrome.

Discussion:

BRASH syndrome is an acronym for bradycardia, renal failure, AV nodal blockade, shock, and hyperkalemia. It has classically been described in the setting of AV nodal blocking agents and typically more prevalent in patients with underlying cardiac or renal impairments. The pathophysiology involves AV nodal blockers directly leading to bradycardia which leads to a reduced cardiac output and reduced renal perfusion. This leads to an acute kidney injury leading to hyperkalemia causing further bradycardia. This cycle continues to repeat itself leading to multi-organ failure and shock. Its severity can also be attenuated by angiotensin-converting enzyme (ACE) inhibitors and angiotensin blockers (ARBs). In some cases, BRASH can also arise in the setting of dehydration or overdiuresis leading to acute kidney injury and hyperkalemia. In our patient, the precipitating factor was most consistent with spironolactone-induced hyperkalemia secondary to a pre-existing acute kidney injury rather than the typical beta-blocker use. This case emphasizes the importance of routine follow-up for patients with advanced liver disease that optimal medical therapy may typically include beta-blockers, loop diuretics, and mineralocorticoid receptor antagonists which can all in theory precipitate this syndrome.