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BRASH Syndrome Case Report and Brief Review

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Abstract

BRASH syndrome is an acronym that stands for bradycardia, renal failure, AV node blocker, shock and hyperkalemia. The syndrome is precipitated by synergism from AV nodal blocking agents and hyperkalemia.(1). The combination of the AV nodal blockade and renal failure leads to a cycle of severe bradycardia and hyperkalemia. We will discuss a case of Brash syndrome with a 68 year old female who presented with generalized weakness and was found to be hypotensive, bradycardia, in acute renal failure with hyperkalemia likely related to metoprolol and amlodipine use.

Case Presentation

A 68 year old female with past medical history of atrial fibrillation not on anticoagulation secondary to recent GI bleed, status post watchman device on dual antiplatelet therapy, heart failure with preserved ejection fraction, type 2 diabetes, on metoprolol 200 mg as well as 10mg amlodipine, presented for complaints of generalized fatigue, weakness, slowed speech and nausea worsening over several days. She stated her home health aid noted her to be bradycardic and hypoglycemic and encouraged her to get evaluated.

Her vital signs on initial presentation included heart rate (HR) of 34 beats per minute, respiratory rate (RR) 18, blood pressure (BP) 88/57, temperature 98.1, and pulse oximeter (SpO2) 91% on room air, put on 2L nasal cannula and improved to 96%.

Physical examination revealed an ill appearing female, oriented to self, location and time but with sluggish speech. She was, however able to follow commands and was nonfocal on examination. Her heart rate revealed bradycardia with an irregular rhythm, lungs were clear bilaterally. She had chronically swollen lower extremities without evidence of cellulitis and strong distal pulses. Physical examination was otherwise negative.

The patients point of care blood glucose was 52 at which time she was given 25g dextrose. Her EKG read atrial fibrillation with slow ventricular response with rate 34 beats per minute. She had inferior T wave inversions but not other signs of ischemia. The patient had electrolytes revealing potassium 6.2 and magnesium 2.3. Her creatinine was 4.74 (previous 1.6 two months prior), BUN 122, and GFR 9. She had hemoglobin 10 and TSH 21.29. The patient had a CT head and chest x-ray done, both of which were unremarkable.

The patient was medically treated for hyperkalemia with insulin with glucose, lasix 40 mg, calcium gluconate, and sodium bicarbonate. Her blood pressure improved however heart rate remained low throughout duration in the emergency department. The patient was started on an epinephrine drip at 0.03 to maintain heart rate and admitted to the ICU. During her ICU stay her creatinine did not improve initially and patient had a vascath placed and underwent 2 rounds of hemodialysis (HD). Her electrolytes and creatinine did ultimately improve and patient was discharged to subacute rehab after 11 day hospital stay, no longer requiring HD.

Discussion

The most common cause of BRASH syndrome are AV nodal blockers, metoprolol and amlodipine which is thought to be due to renal clearance of these medications which causes an inverse elevation in concentration as the GFR decreases.(5)

The synergistic effect is due to the bradycardia causing reduced cardiac output leading to poor renal perfusion, worsening the AKI and hyperkalemia. The cycle typical starts secondary to dehydration caused by recent illness or medication changes causing a pre-renal AKI. There have been some studies suggesting temporal association, worse in the summer months thought to be secondary to high likelihood of developing dehydration.

Given that BRASH is a constellation of abnormalities, not a cause of a symptom, management should be geared toward intervening on the life threatening components of the syndrome, bradycardia and hyperkalemia usually being the most immediately life threatening.

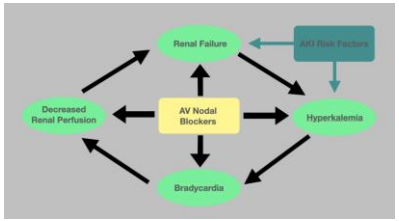
When evaluating these patient, a prompt EKG should be performed as well as CMP. Given the cardiotoxic effects of hyperkalemia, calcium is frequently given, especially in cases of hypocalcemia. Calcium gluconate is the preferred method, though calcium chloride is more potent, containing three times the amount compared to calcium gluconate, however, patients typically do not tolerate calcium chloride was well. The standard dose of calcium gluconate is 10 mL of 10% solution or 1 gram IV given over 5-10 minutes, while the dose of calcium chloride is 1gram IV over 1-2 minutes. (3). These patients will typically also get insulin, which aids in shifting potassium back into the cell, though this should be given with glucose. Nebulized albuterol also acts similarly by shifting potassium intracellularly and has added benefit of beta-2 blockade causing tachycardia.

IV hydration in these patients is essential given that dehydration is a very common inciting cause.

The most common acid base disturbance is a non-anion gapped metabolic acidosis from uremia.(1). The BICAE-ICU trial discussed use of isotonic bicarb, 150 mEq/L sodium bicarbonate in D5W which did not show statistical significance related to mortality but did improve morbidity as it decreased the need for emergent dialysis from 52% to 35%.(2)..

If hypotension does not improve with fluids and other management options, vasopressors should be utilized. The vasopressor of choice are epinephrine infusion or isoproterenol. (4).

Frequently patients condition will improve once one is able to block the synergic effect by fixing the hyperkalemia and hydrating the patient however there are instances that patients develop need for hemodialysis, as in our case and due to the worsening of cardiogenic shock may require transvenous pacing. If left untreated for prolonged time hyperkalemia would continue worsening, ultimately resulting in cardiac arrest .



Conclusion

We discussed a case of BRASH syndrome in a patient who presented with bradycardia, renal failure, metoprolol use, shock, hyperkalemia and hypoglycemia. When treating these patients it is essential to treat life threatening abnormalities as one typically would. Complications of the syndrome include the need for hemodialysis, cardiogenic shock, transvenous pacing, and cardiac arrest so prompt diagnosis and treatment must be made.

Citations

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