BRASH Syndrome - A Classical Presentation of an Uncommon Syndrome.

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Background:
BRASH syndrome is a relatively new acronym for a constellation of symptoms including Bradycardia, Renal failure, AV nodal blockade, Shock, and Hyperkalemia, causing life-threatening bradycardia. In literature, Only handful of such cases of BRASH syndrome were found. Increasing incidence in the elderly population, along with the lack of prompt recognition and high rates of misdiagnosis of BRASH syndrome can escalate mortality.

Case Presentation:
- A 72-year-old female with Stage V CKD, Atrial fibrillation (on metoprolol/ apixaban), Diabetes, CAD, Hypertension, and Hypothyroidism presented to the ED with complaints of fatigue.
- She was noted to be bradycardic to 30bpm and was given 2 doses of Atropine by the EMS. Vitals on arrival were HR of 42bpm, hypotension with MAP <65mmHg.
- Significant labs included potassium of 6.8mEq/L (3-5.5mEq/L), Creatinine of 3.80mg/dL (baseline - 2mg/dL). Thyroid function tests were normal.
- EKG showed a junctional Bradycardic rhythm at 43bpm.

Diagnosis and Management:
- She was resuscitated with IV fluids and temporized for hyperkalemia with calcium gluconate, insulin-dextrose and treated with Lokelma.
- Metoprolol was immediately discontinued.
- She subsequently intubated, and admitted to the ICU.
- Hemodialysis was initiated for hyperkalemia unresponsive to medical therapy.
- Echocardiogram was normal with an Ejection Fraction of 50-55%. During her clinical course, bradycardia and hypotension resolved, and was successfully extubated.
- She was discharged on amlodipine for hypertension and close outpatient follow-up.
- She was recommended to avoid beta-blocker use in the future.

Discussion:
- The pathophysiology for the uncommon BRASH syndrome involves synergistic effects of nodal blockade, renal failure, and hyperkalemia. A vicious cycle is thus established with hyperkalemia exacerbating bradycardia and hypotension.
- The clinical presentation varies in severity, with profound bradycardia and hypotension being the most notable finding. If persistent, it causes hemodynamic instability, multi-organ failure, and eventually death.
- As the literature suggests, BRASH is relatively more common in fragile elderly patients and is often precipitated by medications causing bradycardia. ACE-i and ARBs are also risk factors for BRASH syndrome since they accelerate both renal dysfunction and hyperkalemia. Thus, the combination of ACE-i and beta-blockers may set patients up for developing BRASH syndrome, especially in the setting of hypoperfusion from dehydration, GI bleed, and CKD.
- In severe cases, transvenous pacing and renal replacement therapy may be needed as a life-saving intervention.
- The diagnosis can be difficult to make due to multiple critical components, with the focus being only on one component of the syndrome. Our patient had multiple risk factors such as CKD in addition to metoprolol use, which led to the occurrence of BRASH syndrome in her.

Conclusion:
Prompt recognition of BRASH syndrome and timely intervention can avoid fatal outcomes. In clinical practice, medications like beta blockers and ACE/ARBs or a combination of these, are widely used for cardiovascular mortality benefits, and should not be discontinued unless BRASH or a possibility of BRASH develops.

References: