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BRASH Syndrome: A Deadly Pentad of Symptoms

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Abstract:

Here we present the case of a male in his 70's who presented with diarrhea and a syncopal episode who we diagnosed with BRASH syndrome. The acronym BRASH is a novel way to describe the constellation of symptoms that result in unfortunate synergistic effects that can result in profound bradycardia. In the setting of hyperkalemia and concurrent use of atrioventricular nodal blockers, there is potential for a worsening bradycardia and inevitable cardiovascular collapse if not identified.

Case Presentation:

A male in his 70's presented to the emergency department with a chief complaint of diarrhea which had been occurring for one day. The patient described the diarrhea as watery. He stated that he got up to go to the bathroom and had a syncopal episode, falling forward, an abrasion noted to the patient's left face. He denied nausea, vomiting, fever, chills, body aches, or recent antibiotic use. Per chart review, patient was taking amiloride, carvedilol, and losartan. He stated he was compliant with all medications.

On arrival to the emergency department, his vital signs were as follows: blood pressure 88/53 mmHg, heart rate of 101 beats per minute (bpm), respiratory rate of 10 breaths, per minute, temperature of 98°F orally, and a pulse oximeter of 90% oxygen saturation on room air. Patient was placed on 4 liters supplemental oxygen via nasal cannula with improvement to 95% saturation.

Physical exam revealed a nontoxic, well appearing male, in no acute distress. Patient's lungs were clear to auscultation bilaterally, no wheezing appreciated. Despite initial vital signs, patient was found to be bradycardic with an irregularly irregular rhythm. Abdomen was soft, non-distended, without peritoneal signs, with diffuse mild tenderness to palpation. Patient was alert and oriented, answering questions appropriately.

An EKG was ordered and resulted showing a heart rate of 35 bpm. The pattern on the EKG [Figure 1] was consistent with third degree heart block. A code was called, normal saline was started via an IV line, and 0.5 milligrams (mg) atropine was given. This intervention had no effect, prompting physicians to order 50 micrograms of fentanyl prior to transcutaneous pacing. While adjusting settings of the transcutaneous pacing, laboratory results began to come back. A complete blood count was significant for an elevated white blood cell count of 23,500 white blood cells per microliter. Potassium was elevated at 7.5 milliequivalents per liter. Creatinine was elevated at 2.10 milligrams per deciliter. Calcium chloride, insulin, dextrose, and a continuous albuterol nebulizer were ordered as immediate treatment for hyperkalemia.

Cardiology was consulted, who recommended first correcting potassium. They stated that if this did not improve the patient's profound bradycardia and hypotension, transcutaneous pacing could be trialed. Reevaluation after receiving correctional medication for hyperkalemia showed the patient's heart rate significantly improved, to the point where the patient was pacing himself normally. A repeat EKG [Figure 2] showed sinus tachycardia with first degree AV block. The patient's mental status status never changed throughout ED course. An ICU consult was placed and the patient was admitted for suspected BRASH syndrome.

BRASH is a description of the symptoms, representing bradycardia, renal failure, atrioventricular nodal blockers, shock, and hyperkalemia (1). Potential triggers exacerbating the condition include increased age, months of warmer weather, and any risk of dehydration (2).

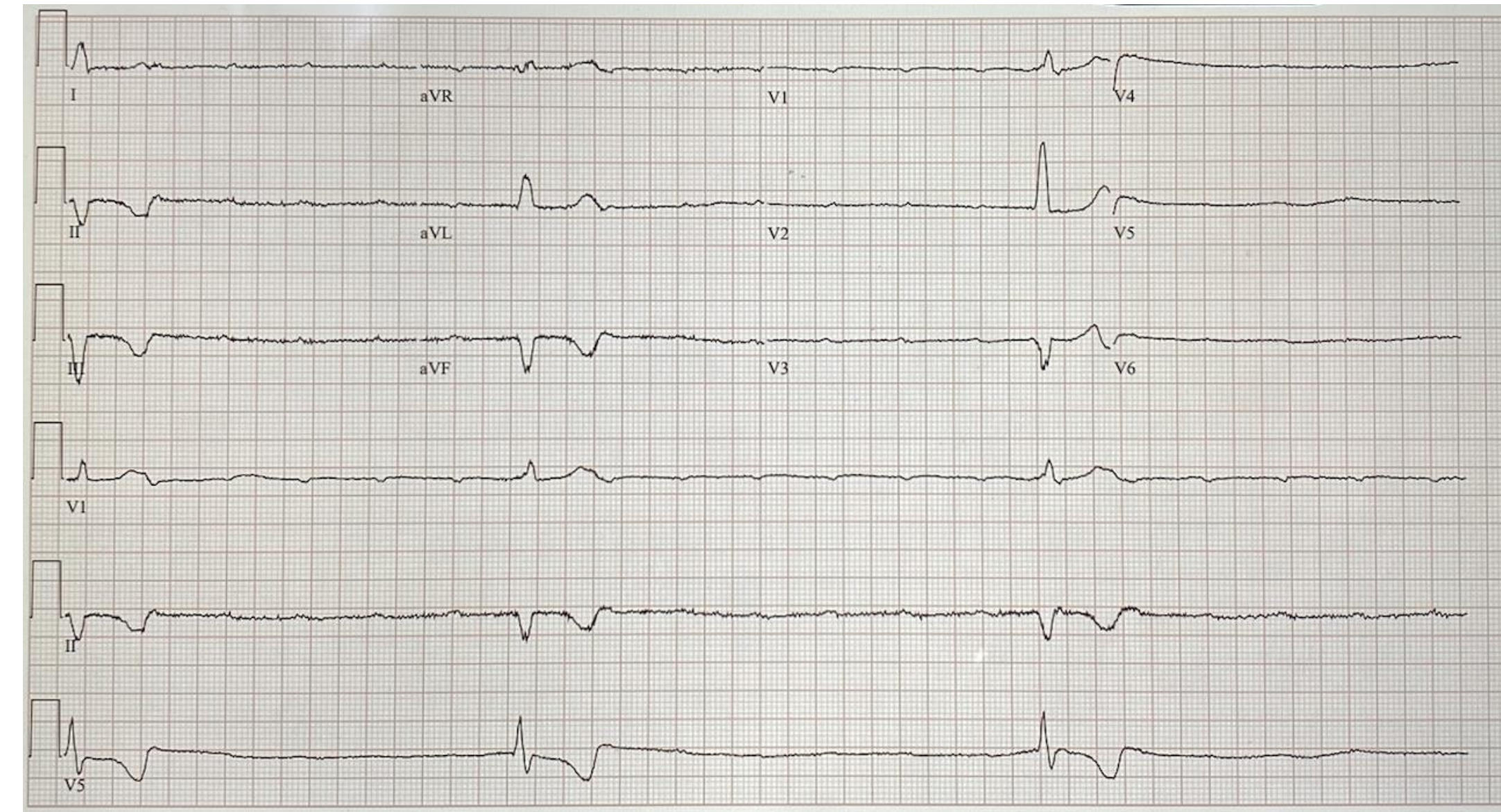


Figure 1: Initial EKG of patient, consistent with complete heart block, idioventricular escape rhythm

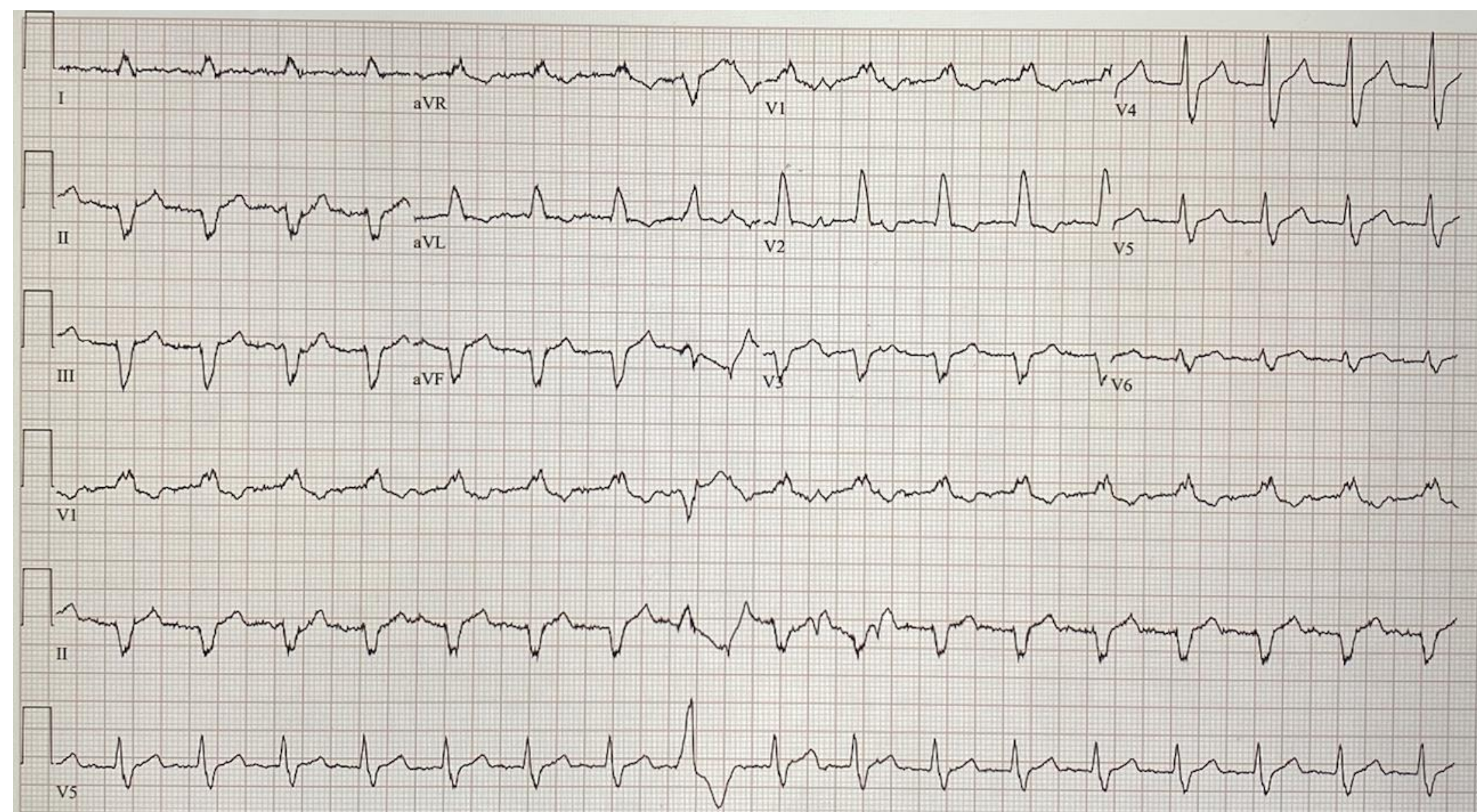


Figure 2: Repeat EKG following transcutaneous pacing cessation and correction of hyperkalemia

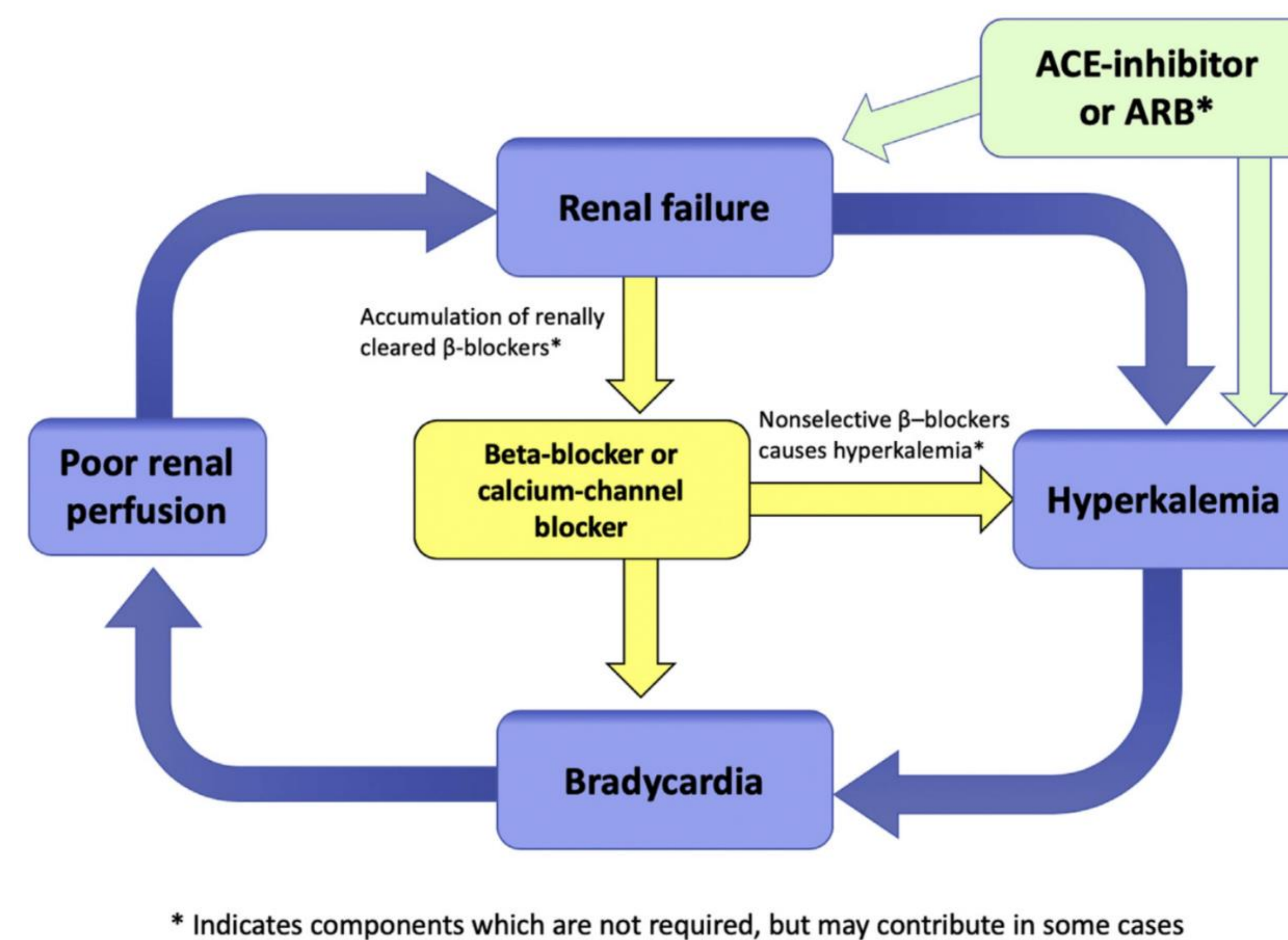


Figure 3: The physiology of BRASH syndrome demonstrating the synergism of different factors

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Discussion:

There are numerous case reports of different combinations of medication's leading to suspected BRASH syndrome. All cases involved some type of AV node blocker. Many also included diuretics. Given the aforementioned case, the patient was at increased risk, given that he was on a potassium sparing diuretic, a beta blocker, and an angiotensin receptor blocker..

Elderly patients are considered high risk for hyperkalemia, due to worsening renal function, volume depletion with increased age, and overall increase in co-morbidities (3). Erden et. al mentions mentions an similar case, involving an elderly woman, approximately the same age as the patient in this case, who experienced a syncopal episode caused by hyperkalemia, in the setting of an angiotensin-converting enzyme inhibitor and spironolactone (4). It is difficult to determine the cause of the syncopal episode that the patient mentioned on arrival to the emergency room. It may have been made multifactorial in nature, as he was severely dehydrated and bradycardic.

Chart review showed that once the patient admitted to the intensive care unit, he remained normotensive with reassuring vital signs during his inpatient stay. Subsequent notes from providers and ancillary staff demonstrated that the patient experienced one episode of hypotension that was resolved with a fluid bolus. Cardiology was consulted and advised against a permanent pacemaker. The patient was discharged home with close follow up.

Conclusions:

BRASH syndrome should be identified as quickly as possible to avoid further cardiovascular collapse. As seen in Figure 1, the initial electrocardiogram (EKG) of the patient showed a ventricular escape rhythm rather than the typical findings of a hyperkalemic patient (5). When we think of hyperkalemia represented on an EKG, we consider QRS widening in addition to peaked T waves, which are not present. Figure 2 is consistent with resolution of the idioventricular escape, showing sinus tachycardia with premature ventricular complexes present.

The overall prognosis of the syndrome, once identified, has been recorded as excellent if done so in a timely manner (6). In our case, the patient did extremely well after correction of hyperkalemia, in addition to fluid boluses. Transcutaneous pacing was performed while attempting to lower serum potassium. Documented cases also show that transvenous pacing and emergent dialysis can be beneficial in the treatment and improve morbidity/mortality of patients with severe metabolic derangement (1).

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