**Decoding Severe Bradycardia: Antihypertensive Interplay in BRASH Syndrome**

Authors1

Siti Fatimah Kather Hussain1, Tan Chun Chau1, Barathan Manoguran1, Varrsha Manoharan1

1Emergency and Trauma Department, Hospital Seberang Jaya, Pulau Pinang, Malaysia

Introduction

BRASH syndrome, a critical complication in patients on AV nodal blockers, combines Bradycardia-Renal Failure-AV Nodal Blockade-Shock-Hyperkalemia. Prompt recognition and intervention are crucial due to its potential for high morbidity and mortality. Hyperkalemia should suspected in bradycardic patients with renal impairment on AV nodal blockers.

Case description

A 76-year-old Malay man with diabetes, hypertension, dyslipidemia, and chronic kidney disease presented with severe bradycardia, heart rate (HR) of 30 bpm during a routine clinic visit. He was on atenolol 100mg OD (beta-blockers), diltiazem 30mg BD (calcium-channel blocker), and perindopril 8mg OD (ACE inhibitor). Upon assessment in the emergency department, the patient was conscious and asymptomatic. However, his blood pressure (BP) was unrecordable, with a HR of 26-30 bpm. An electrocardiogram noted sinus bradycardia without a tall-tented T wave. Blood investigation revealed compensated metabolic acidosis, hyperkalemia (6.2mmol/L), and elevated creatinine (160mmol/L). The patient was resuscitated with IV crystalloid, IV atropine, nebulized salbutamol, and an IV lytic cocktail, leading to normalization of BP, HR (50-60 bpm), and potassium level (4.9mmol/L). He started on kalimate powder and oral sodium bicarbonate, discontinuation of beta blocker, and ACE inhibitors. He was admitted for five days while his BP was well-controlled with amlodipine and diltiazem.

Discussion

The BRASH syndrome can arise from hyperkalemia and AV nodal blockers, exacerbated by dehydration or medication titration. ACE inhibitors combines with other antihypertensives can increase side effect of hyperkalemia, hypotension, and renal failure. ECG changes may not align with typical hyperkalemia patterns. Initial resuscitation necessitates addressing hyperkalemia and bradycardia concurrently, with aggressive hyperkalemia therapy and fluid resuscitation. Sodium bicarbonate aids in managing uremic acidosis and hyperkalemia, potentially avoiding urgent dialysis. Severe cases may require renal replacement therapy, while reversal of AV nodal blockade through various medications (lipid emulsion, glucagon, or high-dose insulin infusion) or transcutaneous pacing.

Conclusion

The BRASH syndrome is often overlooked but poses a life-threatening risk. Effective management entails promptly addressing all syndrome components. In this case, timely interventions targeting hyperkalemia and bradycardia prevented the need for invasive treatments. Withhold the ACE inhibitors can mitigate the risk of recurrent BRASH syndrome.

Keywords: Bradycardia, hyperkalemia, ACE Inhibitor

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