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A Rare Case Study of BRASH Syndrome

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Keywords

Bradycardia, Renal Failure, Av Nodal Blocking Agents, Shock, Hyperkalemia

Abstract

BRASH syndrome in a combination of clinical findings resulting in patients with baseline renal failure on long term AV nodal blocking agents. Our case is a patient on long term AV nodal blocker who came with complaints of light headedness and further was diagnosed as BRASH syndrome. Initially the patient's will be started on intravenous fluids, administration of calcium gluconate, intravenous dextrose and insulin.

1. Introduction

Bradycardia [1], renal failure [3], AV nodal blockers, Shock and Hyperkalaemia [2,4,6] is a collection of findings of a synergistic cycle, often fatal, occuring in renal failure patients taking AV nodal blockers. It is often under diagnosed and not recognised, easily confusing with other etiology of the presenting clinical symptoms.

2. Case Report

A 60 years old female was brought to the ER with complaints of giddiness and light headedness for 1 day, and a near syncope episode. On the way to ER she had of heightened complaints of the same, but no history of loss of consciousness. There was no history suggestive of trauma, loss of consciousness, blacking out of vison, seizures [7]. No history of nausea, vomiting, ear pain and headache. She was a known case of Type 2 DM, Systemic hypertension, CAD and hyperlipidaemia and was on regular medications.

The patient was drowsy, oriented to time, place and person and was afebrile. She was moderately built and nourished. On monitoring the vitals, the heart rate was irregular and 39 beats per minute, Blood pressure was 90/50mmhg, reduced breath sounds on both sides and the respiratory rate was 25 per minute, spO2 was 99% at 6L of oxygen. Other systemic examinations were insignificant.

Investigations showed raised BNP values 1650pg/ml, serum creatinine was 2.3mg/dl, BUN 33mg/dl, serum potassium was 6.5mEq/l, lactate was 5.4mmol/l.

Her history revealed the long term use of T CARVEDILOL, and renal failure [3], on examination bradycardia was revealed, investigation showed Hyperkalaemia [2,4,6] leading to the primary diagnosis of BRASH syndrome.

The patient was started on IV fluid normal saline 1L bolus and Calcium gluconate. The patient improved clinically with the treatment.

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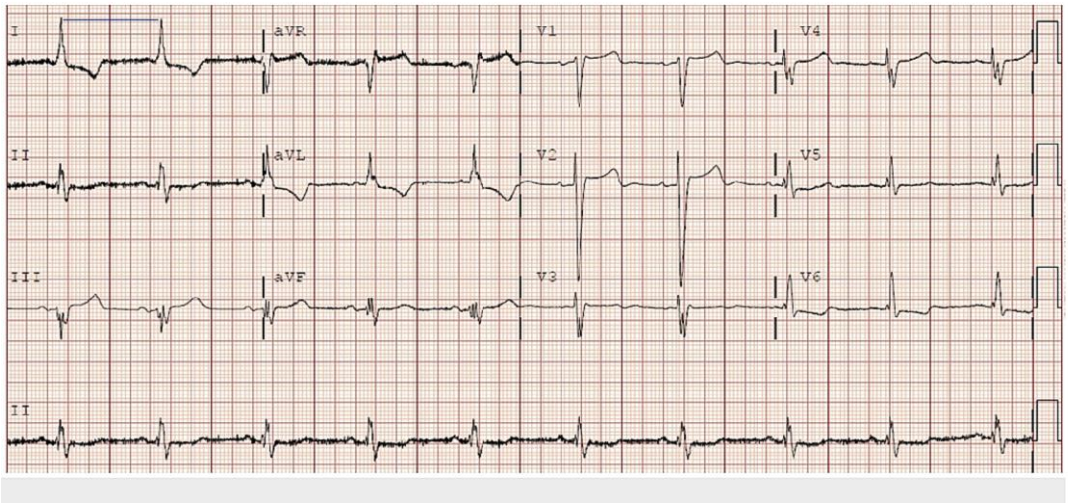

Figure 1 ECG changes

3. Discussion

BRASH Syndrome is usually seen in elderly patients with cardiovascular pathology with chronic usage of chronotropic drugs or renal dysfunction with reduced reserve, triggered by hypovolemia leading to synergistic bradycardia, and Hyperkalaemia. However in the setting of this syndrome l, Hyperkalaemia is not as extreme as in other usual cases. And also this syndrome is to be kept in mind before prescribing long term AV blockers to patients with pre-existing comorbidities. Despite numerous cases presenting with similar clinical signs and symptoms, this syndrome can be uniquely picked from the lot with careful observation and monitoring. The clinical symptoms can vary on a wide range of asymptomatic brady cardia to multiple organ failure. The basic treatment is to treat each component of the syndrome; fluid administration in case of hypovolemia [5], intracellular potassium shifting initiated with calcium gluconate for stabilization of membrane along with intravenous dextrose and insulin administration.

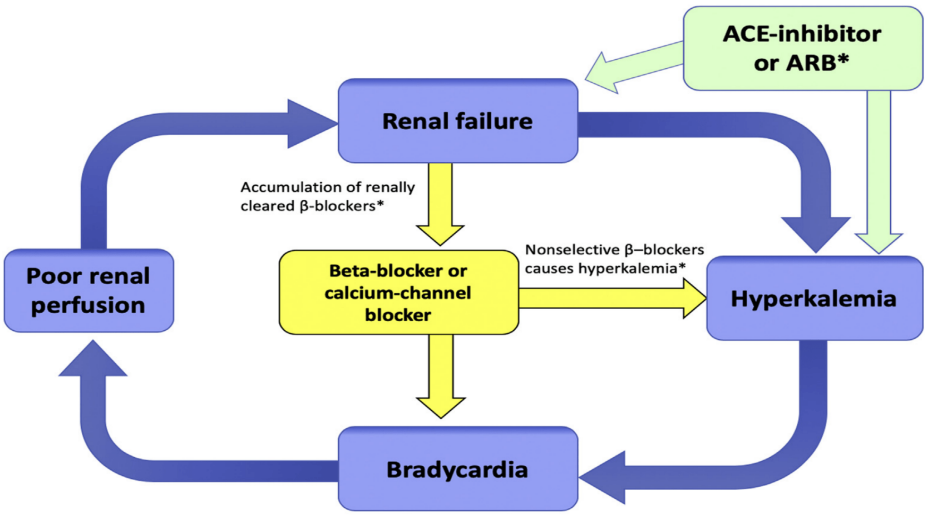

Figure 2: Mechanism of BRASH SYNDROME

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4. Conclusion

It is vividly known that BRASH syndrome in chronic chronotropic drug usage presents with bradycardia and Hyperkalaemia [6] and sometimes hypotension. After evaluation of the patient, initial treatment should be focused on fluid correction and potassium level correction. Careful prescription and follow up of patients should be ensured.

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Ethical Consent
Patients included in the study had provided informed consent

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Conflict of Interest
The authors declare that was no conflict of interest

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