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Bradycardia, Renal failure, AV node blocker, Shock, Hyperkalemia (BRASH syndrome): Don't ignore it

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ABSTRACT

BRASH syndrome is characterised by bradycardia, renal failure, hyperkalemia and use of an AV nodal blocker (AVNB). These symptoms form a vicious cycle seen in a patient having reduced glomerular filtration rate who are on AVNB. There is reduced excretion of AVNB in a patient with low glomerular filtration rate along with hyperkalemia due to renal failure. All of these factors precipitate the cycle of BRASH Syndrome. Here we present a case of 50 year old male presenting with bradycardia, renal failure and hyperkalemia and on history evaluation revealed to be on calcium channel blockers for hypertension precipitating BRASH Syndrome.

Keywords: BRASH, Bradycardia, CKD, hyperkalemia, hypertension

1. INTRODUCTION

Chronic kidney disease (CKD) is a major global public health problem defined by the sustained presence of either kidney damage (albuminuria) or reduced kidney function (estimated glomerular filtration rate [eGFR] <60ml/min/1.73 m^2). Chronic kidney disease (CKD) is one of the serious diseases that spread widely. It is characterized by the gradual reduction of renal functions. This may lead to high risk of cardiovascular diseases and the end stage of renal disease (ESRD). CKD is believed to affect 10% to 15% of the population and is estimated to contribute to 5 to 10 million deaths annually. More than 14% of the general population was affected by CKD. The main two causes of CKD are elevated blood pressure and diabetes mellitus (DM). Although there has been increase in care provided to patients who have chronic diseases of the kidney, expectancy of their lives still remains to be reduced across the spectrum of chronic kidney disease and the weight of chronic kidney disease on the shoulder of heath care facilities throughout the world remains to be significant. Increased potassium levels are a frequent complication encountered in patients with chronic kidney disease. Hyperkalemia is amongst the most significant metabolic complications because it can cause electrophysiological disorders with severe clinical repercussions that may lead to death (Bonvini et al., 2006). Life threatening arrhythmias though can be

precipitated at any potassium level but hyperkalemia associated arrhythmias are more often to be seen at potassium levels above 6.5meq/l (lee et al., 1986).

BRASH Syndrome is an often unrecognized entity which has recently been described in few case reports. It is an important aspect that BRASH syndrome presents with profound bradycardia which is not in proportion to their potassium levels or there is use of an AV nodal blocker agent. Also, these patients are refractory to chronotropic agents and hence this is a crucial syndrome requiring an alert mind and prompt diagnosis. Here we report a Case of a 50-year-old male with chronic kidney disease with maintenance hemodialysis reported with bradycardia and on investigations turned out to be a case of BRASH Syndrome.

2. CASE PRESENTATION

A 50-year-old male patient who was a known case of chronic kidney disease on maintenance hemodialysis sine 6 months visited the hospital for routine dialysis. The patient was a known case of hypertension since 6 months and was on calcium channel blocker as nifedipine for the same. There was no history of diabetes mellitus, bronchial asthma, cough, cold or fever. The patient had no history of chest pain, palpitations, loose stools, pain in the abdomen, nausea and vomiting. On examination patient was in bradycardia with heart rate of 56 beats per minute, regular in rhythm. Blood pressure was 80/50 mm hg in right arm supine position Rest of the general examination was unremarkable.

On systemic examination there was Bilateral Crepitation in both infra mammary region, normal heart sounds, and soft abdomen with no organomegaly. Arterial blood gas (ABG) analysis showed 7.317 pH, 29mmHg PCO2, and 142 mmHg PO2. His laboratory investigations revealed:-Hb-12.3gm/dl, mcv-84fl, platelet count-231000/dl, White Blood cell count-9800/dl, Creatinine=10.8, urea-136, potasssium-6.4 meq/l, sodium-134meq/l, Total protein-6.6gm/dl, albumin-3.2gm/dl, globulin-3.4gm/dl, alkaline phosphatase-112/dl, alt-42, ast-37, Total Bilirubin-1.4mg/dl.

ECG showed in figure 1and 2 revealed sine wave pattern characteristic of hyperkalemia and normal waves after correction of potassium respectively. XRAY of chest figure 3 is suggestive of minimal infiltrates. Patient was immediately taken for Sustained Low Efficacy Daily Dialysis (SLEDD) through his AV Fistula. After four hour of dialysis his pulse increased to 98 beats per minute. Patient improved clinically through series of hemodialysis and was ultimately discharged in stable condition and is doing well on follow up with maintenance hemodialysis.

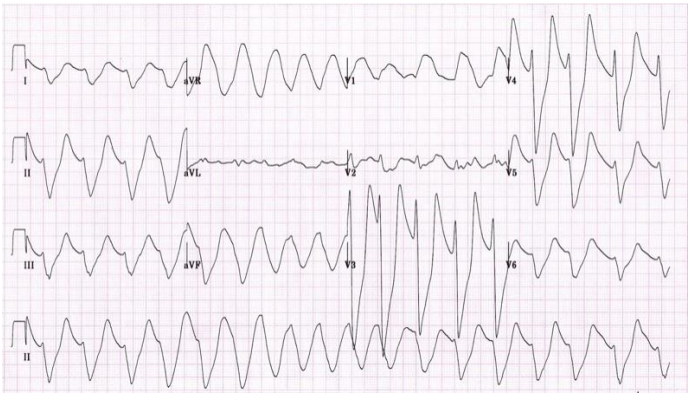

Figure 1 Sine wave before potassium correction

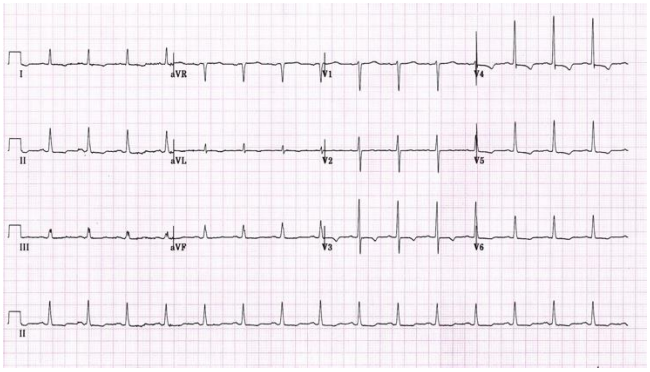

Figure 2 ECG after Potassium Correction

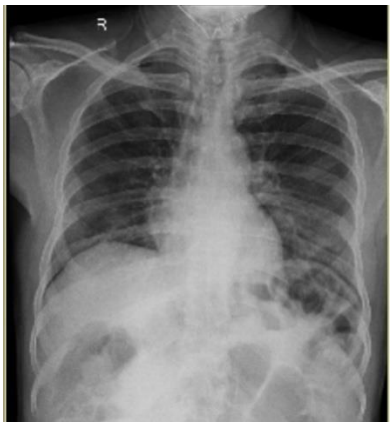

Figure 3 Chest X ray of the case showing mild infiltration

3. DISCUSSION

Hyperkalemia is frequently complicated in patients with advanced chronic kidney disease (CKD) because kidney is the prime source of potassium excretion (Ahmad et al., 2017). Decline in excretion of potassium is proportional to the reduced glomerular filtration rate and patients who is diabetic, elderly, having congestive cardiac failure and on medications such as renin angiotensin aldosterone system (RAAS) blockades are more likely to manifest with hyperkalemia (Kumar et al., 2013). Firstly, hyperkalemia management includes the initiation of diet with reduced potassium and following up of patients for compliance to this diet.

It is of utmost significance to enquire about the medications the patient is using as even temporary stoppage of reduction of dose of any precipitating medications can help in reducing potassium levels. Lastly, the use of agents which bind to potassium is useful in both acute as well as chronic hyperkalemia (Chande et al., 2021). With an increasing load of chronic kidney disease patients with multiple co morbidities who require a AV node blocker agent on prescription list an emerging entity known as BRASH syndrome has gained importance. BRASH if unrecognized can be fatal and hence required an early diagnosis and management. Importantly the hyperkalemia in our patient was disproportionate to the clinical findings. This emphasizes on the effect of AV node blocking agent leading to a marked bradycardia. Thus resulting in marked decrease in cardiac output; leading to shock with further worsening of renal functions thus making a vicious cycle of BRASH syndrome. A very few cases have been reported of BRASH syndrome till date however we argument that it a very under recognized condition due to lack of awareness in the treating physicians.

BRASH can also be seen in patients who do not have chronic kidney disease but develop acute kidney injury and are on AV node blocking agents. The proposed pathophysiology of this condition is augmentation of hyperkalemia by AV nodal blocking agents resulting in reduced chronotropy as well as hypotension. This causes hypoperfusion and renal failure forming a vicious cycle of BRASH syndrome. This hyperkalemia reflects by causing arrhythmia which can be documented on an ECG. AV Nodal Blocking Agents may induce Bradycardia with even mild hyperkalemia (Prabhu et al., 2020). It is crucial to identify this clinical entity as a standard ACLS algorithm involving atropine and cardiac pacing may not be able to treat bradycardia associated with BRASH syndrome. Thus, the treatment involves treating the hyperkalemia along with toxicity of calcium channel blocker and beta blocker.

Treatment of hyperkalemia includes calcium for stabilization of cardiac membrane with calcium and intra venous insulin and glucose which lead to shifting of potassium intracellularly causing a decrease in potassium levels in the body. As it is often associated with hypovolemia IV fluids also form a crucial part of BRASH syndrome management. It is important to maintain the renal perfusion while waiting for toxic drugs to be excreted. Catecholamine may be initiated if patient is hemodynamically unstable. Potassium loosing diuretics like furesemide may be given to reduce potassium levels. However in some patient's hemodialysis may be required in emergency if the measures listed above fail.

4. CONCLUSION

CKD patients often experience metabolic complications and are associated with severe results. Hyperkalemia The rise in serum potassium is mainly due to the reduction in GFR and in our patient its affect was aggravated by AV nodal blocking agents leading to BRASH syndrome which was treated by emergency hemodialysis. Thus we highlight the importance of diagnosing and treating BRASH syndrome which is a commonly missed entity and can be tackled by vigilance and prompt management by the physicians preventing mortality.

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Conflict of interest
The Authors have no conflicts of interest that are directly relevant to the content of this clinic-pathological case

Financial Resources
There are no financial resources to fund this study

Informed Consent
Informed Consent was obtained from the patient.

Author's Contribution
All the authors contributed equally to the case report.

Data and materials availability
All data associated with this study are present in the paper.

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