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Case Report

Polypharmacy in the elderly: BRASH syndrome mimicking complete heart block with atrial fibrillation

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Introduction

Polypharmacy is defined as the concurrent use of an increased number of medications or the use of unnecessary medications. One well described but rare phenomenon associated with polypharmacy is BRASH syndrome which comprises bradycardia, renal impairment, AV blockade, shock and hyperkalemia in a vicious cycle. Symptomatic bradycardia is the main clinical presentation in BRASH syndrome and is secondary to AV blockade and hyperkalemia. We report a case of an elderly female on polypharmacy presenting with not only BRASH syndrome, but also lactic acidosis and myocardial ischemia which were linked to one another.

Case Presentation

A 74-year-old female with a past history of diabetes mellitus, hypertension and ischemic stroke presented to a local hospital with dizziness and central chest pain for 2 hours. Her drug history included metformin 1g bd, losartan 50 mg bd, atenolol 50 mg bd, hydrochlorothiazide 25 mg mane, prazosin 0.5 mg tds, nifedipine SR 20 mg mane and aspirin 75 mg nocte for which she had good compliance.

When admitted to the local hospital, the patient was afebrile and confused with pulse rate of 40/ min and a blood pressure of 100/ 40 mmHg. The ECG was interpreted as nodal bradycardia. She was transferred to the National Hospital of Sri Lanka for insertion of a pacemaker after administration of IV atropine 1.8 mg and oral salbutamol 4mg by the consultant cardiologist at the local hospital.

On arrival at the National Hospital of Sri Lanka, the patient was confused and pale with a pulse rate of 68/ min and blood pressure of 100/60 mmHg. There was evidence of acute heart failure with bi-basal fine crepitations and an on-air saturation of 88%. Capillary blood

sugar was 72 mg/ dl. The first ECG was interpreted as complete heart block with atrial fibrillation with possible acute coronary syndrome as the aetiology (Figure 1).

The acute heart failure was managed with supplementary oxygen and intravenous furosemide. Following the exclusion of intracranial bleeding by non-contrast computerized tomography, she was loaded with antiplatelets. Metformin and all antihypertensive medications were withheld. With the liaison of the cardiology team the patient was monitored without urgent pacing due to the improvement in heart rate.

As shown in Figure 1(b) the subsequent ECG showed definite tall tented T waves with absent P waves, widening of QRS complexes and bradycardia suggestive of hyperkalaemia. ST depressions in V4- V6 were also noted.

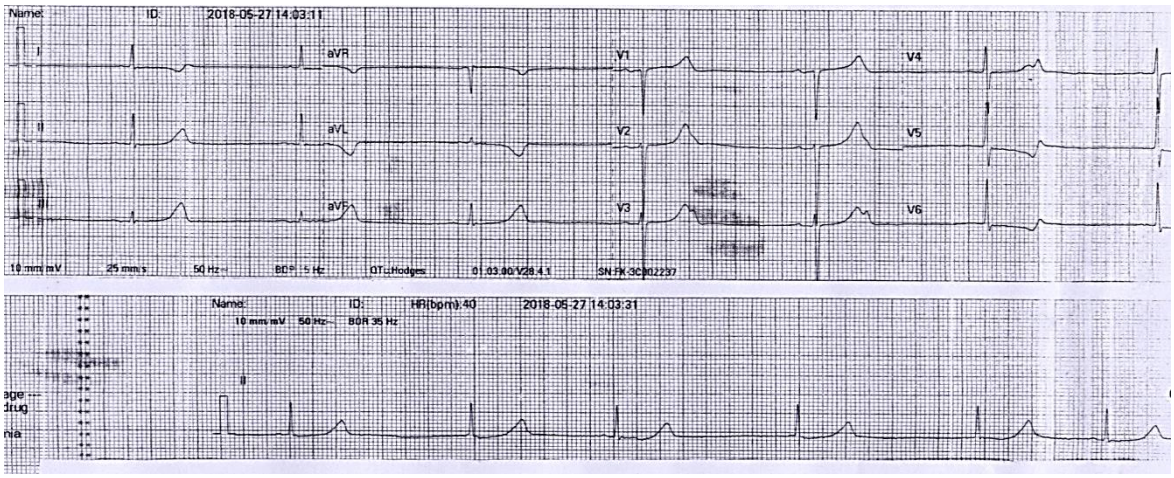

Figure 1(a): The first ECG showing ventricular rate of 42/min, absent p waves with regular R-R intervals, T inversion in aVL and ST depressions in V_5- V_6.

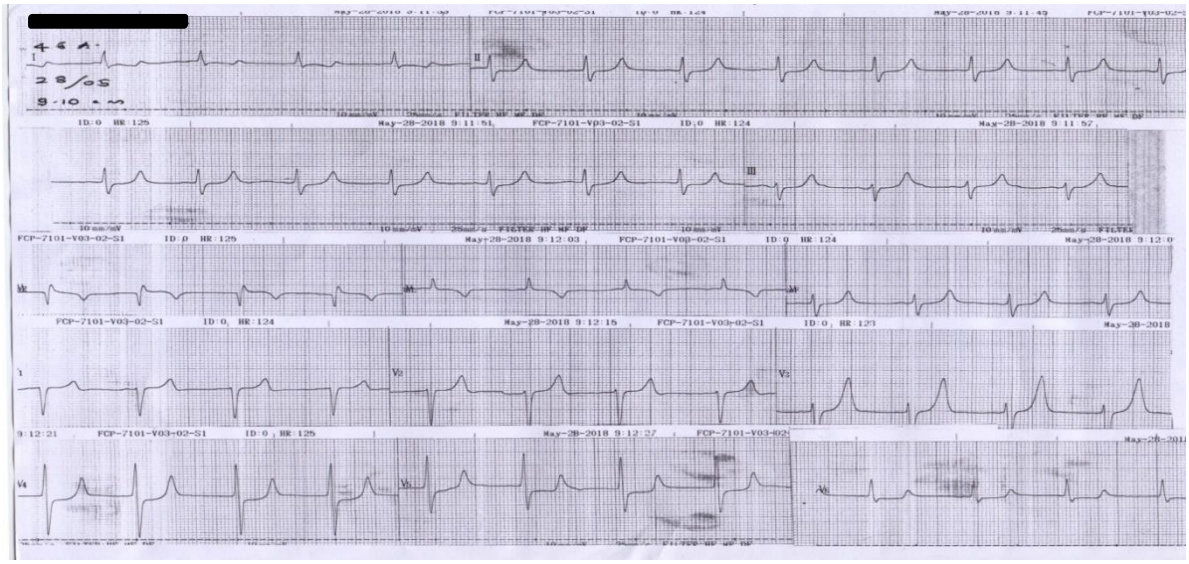

Figure 1(b): The subsequent ECG showing tall tented T waves with absent P waves, widening of QRS complexes and bradycardia suggestive of hyperkalaemia.

The initial blood gas analysis confirmed the hyperkalaemia of 7.6 mmol/l (3.5- 5.3 mmol/l). There was also evidence of a high anion gap metabolic acidosis (pH- 7.25) with a lactate of 8.4 mmol/l (<2 mmol/l) and serum creatinine of 188 µmol/l (60- 120 µmol/l).
The patient later became more confused with evidence of cardiogenic shock, for which she was started on an intravenous noradrenaline infusion. Both medically resistant hyperkalaemia and lactic acidosis warranted urgent hemodialysis after which a marked improvement was observed.

The subsequent ECG showed evidence of ischaemia, T inversions and ST depressions in the anterolateral leads with an elevation of cardiac troponin I of 16.6 ng/ml (<0.5 ng/ml). The possible precipitant was hypotension in a background of anemia which was found to be of mixed deficiency. Therefore, the noradrenaline dose was escalated, and one unit of packed red cell was transfused during the dialysis.

Her echocardiogram revealed mild left ventricular hypertrophy, diastolic dysfunction and an ejection fraction of 55%. The ultrasound scan revealed early chronic renal parenchymal changes. At the time of discharge on day 7, the patient had a heart rate of 72/min with a blood pressure of 170/80 mmHg and clear lung fields. Serum potassium reverted to 3.0 mmol/l with a serum creatinine of 125 µmol/l. Enalapril 2.5 mg was introduced with dual antiplatelet therapy, statins and a haematinic. Throughout the hospital stay, capillary blood sugar values were within normal limits. Therefore, dietary control was advised with the plan for follow up blood sugar measurements.

Discussion

Polypharmacy is common in the elderly due to the presence of multiple diseases as the patient ages. It increases negative health consequences in the elderly. As the number and percentage of the elderly population is increasing in Sri Lanka, this is a growing issue which is frequently overlooked by physicians.

As shown in Figure 2, BRASH syndrome is an interesting phenomenon encountered in polypharmacy which comprises bradycardia, renal impairment, AV blockers, shock, and hyperkalemia in a vicious cycle. The BRASH syndrome represents an overlap between hyperkalemia and AV nodal blocker intoxication [1, 2].

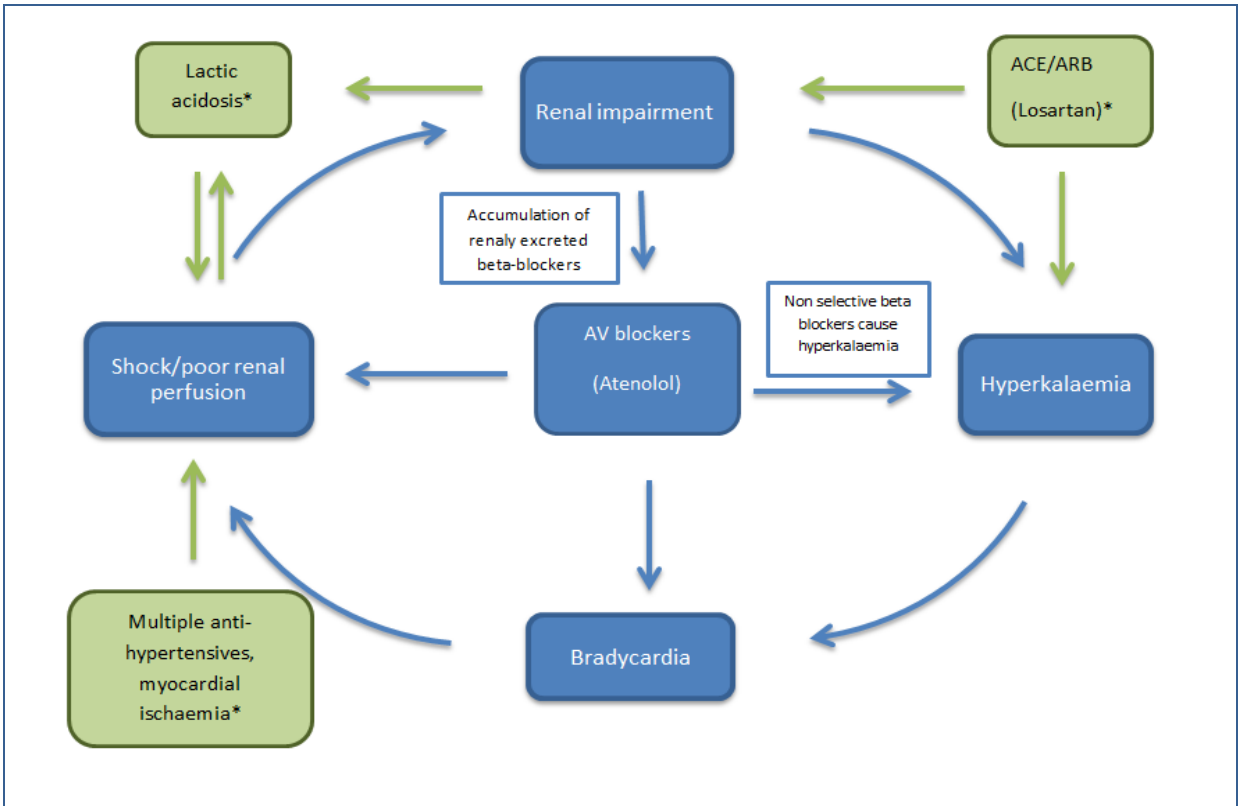

Figure 2: How factors interplay in BRASH syndrome
Adapted from Farkas J. BRASH syndrome: Bradycardia, Renal failure, Av blocker, Shock, Hyperkalemia. PulmCrit. 2016. Available from: https://emcrit.org/pulmcrit/brash-syndrome-bradycardia-renal-failure-av-blocker-shock-hyperkalemia/.)

*****Denotes the factors which are not the essential components for the BRASH syndrome, but contributed to a more complicated clinical picture in our patient.

The multiple antihypertensive medications in our patient, together with myocardial ischaemia and resultant period of poor cardiac output, may have contributed to the above vicious cycle. She had developed asymptomatic chronic renal impairment due to ~~chronic~~ diabetes and hypertension, as evident ultrasonically, predisposing to acute renal impairment on the slightest injury according to RIFLE classification [3].

The effect of a non-selective beta-blocker (atenolol), which is excreted via the kidneys, may have worsened the hyperkalaemia. The poor perfusion state together with a high dose of metformin can explain the lactic acidosis. The lactic acidosis might also cause accumulation of potassium in the body [4], reduction of cardiac contractility and vascular hypo responsiveness to vasopressors.

Interestingly, not only the general physicians but also the cardiology experts were misguided by the ECG findings of this patient. At first, the ECG was interpreted as complete heart block with atrial fibrillation. But the absent P waves were actually due to hyperkalemia. The bradycardia is explained by the interplay of beta blockers and hyperkalemia. Even though true second and third-degree atrioventricular (AV) block have been described in hyperkalemia [5], they are uncommon because the P wave usually disappears before such advanced AV block occurs [6]. On the other hand, the manifestations of hyperkalaemia in the elderly may not accurately correlate with the degree of hyperkalaemia [7]. This may further worsen with polypharmacy, as in our patient.

Conclusion

When managing elderly patients with multiple comorbidities, the complications of polypharmacy should be monitored and every possible measure should be taken to prevent them. The aetiology of bradycardia should always be evaluated and promptly managed to avoid life-threatening sequelae.

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