Acute Renal Failure with Hyperkalemia Presenting as Recurrent Syncope

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Introduction

Common causes of syncope in elderly include acute myocardial infarction, cardiomyopathies, postural hypotension and degenerative complete heart block. We report a case that presented with syncope due to sinus node disease and was found to have acute renal failure with hyperkalemia, which recovered after the renal failure was controlled.

Case Report

A 78 years male presented on 17th Oct 2007, with the history of syncope for two days. The syncopal episodes occurred at rest, lasted for 4-5 minutes and patient had incontinence of urine in one episode. The episodes were relieved on their own, without any residual neurological deficit. The frequency of the episodes was 2-3 times a day, but for the last six hours, these occurred every 30-60 minutes. In addition the patient also gave history of decreased urinary output for the last 3-4 days and had not passed urine for the last 6-8 hours. There was increased swelling of feet for two days. There was no history of chest pain or palpitation preceding the syncope. Patient also had history of increased dyspnoea for four days with paroxysmal nocturnal dyspnoea.

He was a known case of hypertension with coronary artery disease with extensive anterior wall myocardial infarction (MI) and had undergone coronary artery bypass grafting (CABG) for three vessel disease, about 15 years back. He had left bundle branch block (LBBB) in old electrocardiogram (ECG). His echocardiography showed severe left ventricular (LV) dysfunction with ejection fraction (EF) of 22%. He was on platelet inhibitors, ramipril (5 mg twice a day), digoxin (0.125 mg/day), torsimide (20 mg/day) and statins. He used to be in NYHA class II-III and had been admitted three times with left ventricular failure during the last one year, last time around two months back. During that admission, his renal function, electrolytes and routine blood parameters were almost normal.

On examination, the patient was dyspnoeic, his heart rate was 40/min and blood pressure 110/80mm Hg. His jugular

venous pulse (JVP) was raised. Cardiovascular examination showed apex beat in 6th intercostal space, outside the mid clavicular line. First and second heart sounds were normal and third heart sound was present at apex. He had mild mitral regurgitation clinically.

Keeping in view the history of old MI, severe LV dysfunction and baseline LBBB, we kept complete heart block as the first possibility leading to syncope. Second possibility was intermittent ventricular tachycardia.

ECG was done which showed bradycardia with absent P waves and ventricular rate of 42/minute. The QRS complexes were wide with RBBB morphology, with poor progression of R wave in precordial leads and there was left axis deviation (Fig. 1). So we kept the possibility of sinus node disease and planned temporary pacemaker insertion (TPI) followed by permanent pacemaker insertion (PPI).

After around 10 minutes of admission, as the patient was being prepared for TPI, he suddenly developed cardiac arrest with monitor showing no P waves or QRS complexes. Immediate cardiac massage was started and TPI was done through internal jugular vein and the patient was revived. The attendants were explained the need for PPI. In the mean while his blood investigations were ordered. The blood reports showed that urea was 150 mg/dl, creatinine 2.8mg/dl and he had serum potassium levels of 6.9 meq/L. Table 1 shows the blood parameters on different days as well as the report of arterial blood gases (ABG).

Based on the blood reports, the plan of PPI was deferred. His ramipril and digoxin were stopped. He was given injection calcium gluconate, dextrose insulin infusion, injection furosemide and oral sodium polystyrene sulfonate.

Gradually, the patient's urine output improved. By 6th day his potassium levels were normal. After two days, his ECG showed normal sinus rhythm and sinus tachycardia. He was started on low dose ramipril and digoxin with a watch on blood parameters, which remained normal thereafter. TPI was removed. Abdominal ultrasound showed normal sized kidneys with well marked cortico medullary differentiation. The patient was discharged on 29th October.

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Fig. 1 : ECG showing bradycardia, absent P waves and wide QRS complexes.

Table 1

Routine blood reports and changes in renal functions and electrolytes with treatment

Hb	8.9 gm/dl						
TLC	8400/cmm						
DLC	Neutrophils 56, Lymphocytes 40, Monocytes 2, Eosinophils 4						
Serum proteins	6.1 gm/dl						
Calcium	1.17 mg/dl						
Serum bilirubin	0.6mg/dl						
ABG: PaO ²	108 mmHg						
PaCO ²	24.1 mmHg						
pH	7.26						
HCO ³	10.8 meq/L						
<i>Date wise renal functions:</i>							
	17.10	18.10	19.10	20.10	21.10	22.10	28.10
Urea (mg/dl)	150	181	124	124	125	116	50
Creatinine (mg/dl)	2.8	4.0	2.5	2.4	2.3	1.8	1.3
Sodium (mmol/L)	142	136	138.3	134	132.7	130	130
Potassium (mmol/L)	6.9	6.6	5.7	5.26	4.66	4.5	4.3
Chloride (mmol/L)	107	108	103.5	102.3	102.3	104	104

Discussion

Acute renal failure (ARF) complicates about 5% of hospital admissions and up to 30% of admissions into the intensive care units. It may complicate a wide variety of diseases, which can be divided into three categories i.e diseases that cause renal hypoperfusion without compromising the integrity of renal parenchyma-prerenal azotemia (~55%); diseases that directly involve renal parenchyma –renal azotemia (~40%) and diseases associated with urinary tract obstruction-post renal azotemia (~5%). Pre renal azotemia is rapidly reversible upon restoration of renal blood flow, as happened in our case.

Hyperkalemia is a common acute life-threatening

Fig. 2 : Normalization of ECG changes after treatment of hyperkalemia.

emergency seen in the emergency department. It results from electrolyte imbalance and commonly occurs in patients with known chronic renal insufficiency or end-stage renal disease [1]. The usual causes include renal insufficiency or failure, drug induced -such as angiotensin-converting enzyme inhibitors or use of potassium-sparing diuretics, insulin deficiency or resistance and haemolysis. Although laboratory tests are the gold standard in diagnosing changes in the serum electrolyte concentration, delays may be experienced in obtaining the results. The electrocardiographic changes are the result of the physiological effect of the electrolytes on the myocardial cells. Mild levels of elevated potassium result in the acceleration of terminal repolarisation, causing the tall T-wave changes that are the earliest manifestation on the ECG. Mild to moderate hyperkalemia depresses conduction and results in prolongation of PR and QRS intervals. With increasing severity of hyperkalemia, the sino-atrial and atrio-ventricular conduction is further suppressed and results in the appearance of escape beats and escape rhythms [2, 3]. The QRS complex will continue to widen and may blend with the T wave, creating a “sine-wave” appearance in the ECG. Further rise in the potassium level will result in ventricular fibrillation [2].

Despite this, the classic ECG changes do not always manifest and the relationship between serum potassium concentration and ECG changes varies among people [4, 5]. Hence, ECG alone is not reliable for mild to moderate hyperkalaemia [6]. Minimal changes on ECG may be seen in severe hyperkalaemia [5].

Urgent treatment of hyperkalemia includes stabilizing the myocardium to protect against arrhythmias and shifting potassium from the vascular space into the cells.

Intravenous calcium is administered to stabilize the myocardium, which lowers the threshold potential, thus counteracting the toxic effect of high potassium. Improvement in the ECG changes should be visible within two to three minutes of administration of calcium. Repeated doses can be given while other measures are initiated [7].

Shifting potassium intracellularly is done using insulin or a beta 2 agonist. Nebulized beta agonist such as salbutamol in a dose of 10-20mg every 3-4 hourly is the fastest means of translocating potassium from plasma to intracellular compartment. Insulin typically is given as 10 units intravenously with 50 ml of 50 percent glucose to counteract hypoglycemia. Repeated doses can be given if the potassium level remains elevated.

Sodium bicarbonate is no longer recommended to lower potassium, although it may be appropriate in patients with severe metabolic acidosis [8]. Gastrointestinal excretion is accomplished using sodium polystyrene sulfonate (Kayexalate), which binds potassium in the colon in exchange for sodium. It can be given orally or as a retention enema.

Excretion of renal potassium can be increased with the use of diuretics, particularly loop diuretics (e.g., furosemide). Patients with decreased kidney function may be relatively resistant to the effects of diuretics. If the patient is volume depleted, saline can be given with the diuretic. Haemodialysis or continuous renal replacement therapy is the treatment of last resort, with the exception of patients already receiving these therapies.

Our patient had severe LV dysfunction and was in heart failure at the time of admission. The renal failure was of pre-renal type. The commonest cause of syncope in such patients is ventricular tachycardia/fibrillation (VT/VF). Since he had baseline LBBB with prolonged PR interval, the other cause of syncope in such patients is complete heart block. Hyperkalemia in our case was precipitated by ACE inhibitor and digitalis. The case was unusual in that he presented with recurrent syncope and developed asystole as he was waiting for TPI. His ECG showed wide QRS complexes, no P waves, and T waves

were not tall. His old ECG showed LBBB, therefore wide QRS complexes did not attract much attention. However, retrospectively we can say that the ECG showed sine waves in V4-V6, seen in hyperkalemia, which could have been detected (Fig. 1), though they were ignored due to our previous knowledge of wide complexes in his old ECG. Moreover asystole is not a very common presentation of hyperkalemia and is mostly a terminal event. More common cause of syncope in hyperkalemia is VT/VF or complete heart block. Even decreased urine output was attributed to LVF. We thought that the patient would require PPI but blood reports and ABG led to the correct diagnosis. We even repeated serum potassium to rule out pseudohyperkalemia. The disproportionately high potassium could have been contributed by drugs. Patient responded well to potassium lowering measures after the offending drugs were omitted. He is on our follow up for more than a year now and is maintaining normal renal parameters.

Conflicts of Interest

None identified

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Answers to MCQs

1) b	2) c	3) d	4) d	5) b
6) a	7) b	8) b	9) a	10) c
11) a	12) a	13) b	14) b	15) c