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EC CARDIOLOGY
Case Report

Severe Hypokalemia with Premature Ventricular Contraction

Han Naung Tun[1,2]*

[1]*Department of Cardiology, Pun Hlaing Siloam Hospital, Yangon, Myanmar*

[2]*Council of Hypertension and Stroke, European Society of Cardiology*

***Corresponding Author**: Han Naung Tun, Department of Cardiology, Pun Hlaing Siloam Hospital, Yangon, Myanmar.

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Abstract

Electrolytes imbalance caused by potassium disorders are commonly seen in many clinical conditions due to alterations in potassium intake, changes in excretion or transcellular shifts. Hypokalemia is a common electrolyte disorder in clinical practice and that can lead to serious arrhythmias. Most of the arrhythmias caused by electrolytes imbalance can be usually noticed by electrocardiogram changes. Despite the underlying causes of arrhythmia can be predicted by history taking from patients and ECG changes, sometime it can be missed to provide optimal care and effective medication in time. Thus, physicians and health care providers should be familiar with ECG changes and of electrocardiogram manifestations about electrolyte imbalance in clinical care. Typically, the first ECG manifestation of hypokalemia is decreased T-wave amplitude but arrhythmias associated with hypokalemia include sinus bradycardia, premature ventricular contractions, ventricular tachycardia, or fibrillation, and torsade de pointes can lead to life threatening condition. The present case report article shows the association between the premature ventricular contraction and severe hypokalemia caused by electrolyte imbalance due to chronic diarrhea.

Keywords: *Hypokalemia; ECG Changes; Ventricular Arrhythmia; Premature Ventricular Contraction*

Introduction

Potassium is a very significant body mineral, important to both cellular and electrical function. It is one of the main blood minerals called "electrolytes". Potassium is the primary positive ion (cation) found within the cells, where 98 percent of the 120 grams of potassium contained in the body is found [1]. The blood serum contains about 4 - 5 mg (per 100 ml) of the total potassium; the red blood cells contain 420 mg, which is why a red-blood-cell level is a better indication of an individual's potassium status than the commonly used serum level. The energy for the potassium current is provided by the Na^+ - K^+ ATPase (NKA) of the plasma membrane. It maintains the extracellular and intracellular K^+ concentrations by cytosolic ATP [2]. Magnesium helps maintain the potassium in the cells, but the sodium and potassium balance are as finely tuned as those of calcium and phosphorus or calcium and magnesium. Hypokalemia is a common biochemical finding in electrolytes imbalance patients and may represent a side effect of diuretic therapy or result from endogenous activation of renin-angiotensin system and high adrenergic tone.

Case Report

70 years old lady came to emergency department with fit, up-rolling of eyes on 3-2-2018 at 7pm, loose motion for 2 days, lower limb weakness for 15 days to 1 month. She couldn't walk well for 2 weeks. She has no history of chest pain, no breathlessness, no shortness of breath, no paroxysmal nocturnal dyspnea and no orthopnea. She has no history of stroke. Known Diabetes is present for 3 years. At ED, ventricular bigeminy and lightheadedness frequent attack were occurred. Patient was regularly taking oral hypoglycemic agents, beta-blocker and calcium channel blocker but not on diuretic therapy.

On physical examination:

GCS 15/15, weakness, confusion, BP- 230/125 mmHg.

Oxygen saturation is 99% on air. RR 28 times/min

Body temperature was normal.

Heart sounds were audible with holosystolic murmur and Lung examinations were normal. Extremities were healthy without cyanosis or edema.

Abdominal and Neurological examinations were also normal.

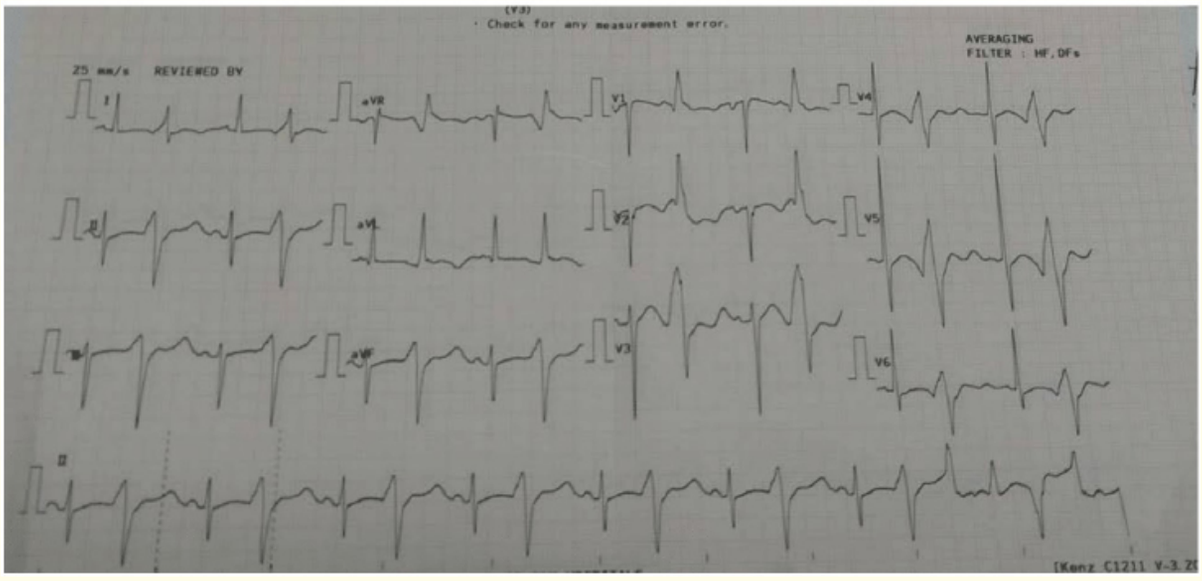

Figure 1: *ECG at the time of admission to ED.*

ECG Interpretation: Underlying Rhythm is Ventricular Bigeminy. That is, there is sinus rhythm shows sinus p wave in lead V1 and in the long lead II at the bottom of the tracing. Every other beat is PVCs. Otherwise there are non-specific ST-T wave changes (including some ST coving in the chest leads).

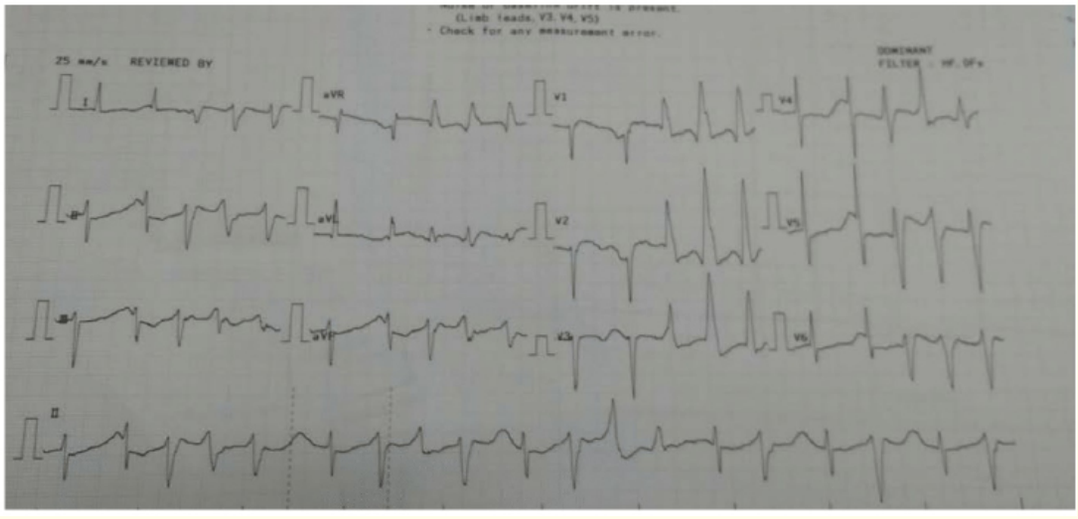

Figure 2: *ECG after 1 hr of admission.*

ECG Interpretation: Fast PVCs with couplets and triplets. ECG shows 3 PVCs in a row (Triplet) or 2PVCs in a row, multifocal. This ECG concern is three PVC or more. It can lead that patient end up having VT, unless underlying cause is corrected promptly. It also shows not specific ST changes in inferior leads and u waves in precordial leads with poor R wave progression due to LAFB.

On Admission, gave Labetalol 20 mg by slow IV injection over a 2-minute period. Additional injections of 40 mg were given at 10-minute intervals until a desired supine blood pressure. After 30 minutes, BP was fallen down to 170/100 mmHg and continued with oral calcium channel blocker and betablocker until blood pressure reached back to normal.

Investigations

Brain CT Scan showed normal imaging findings.
Chest X-ray showed cardiomegaly with mild bronchitis.
Ultrasound abdomen showed no obvious abnormality seen.
Transthoracic echocardiography findings showed moderate MR with diastolic dysfunction grade II and no regional wall motion abnormality seen.

Lab Results

Trop-T 259 pg/ml
Urea 15.8 mg/dl, Cr- 1.5 mg/dl (eGFR- 30), K- 1.6 mmol/l, Na - 139 mmol/l Cl -81 mmol/l, HCO3 - 28.10 mmol/l
RBS- 175 mg/dl and HBA1c- 6.9%
Mg- 1.36 mg/dl, Ca- 9.0 mg/dl, urine potassium level is 25 mEq/L
CK MB 71 U/l, ESR- 30, CRP - 4.42, Hb- 10.8 g/dl, WBC - 11.85, Plt count - 449
pH- 7.553
PCO2- 36.7 mmHg, PO2- 176.5 mmHg, Bicap - 32.3 mmol/l
TFTs - Normal
LFTs - Normal
Lipid Profile - Total Cholesterol 240 mg/dl, LDL- 160 mg/dl, TG and HDL are normal.

Final Diagnosis

Diagnosis is Hypokalemic periodic paralysis due to lose motion with underlying DM and Hypertension.

Potassium Level Monitoring

3-2-18	4-2-18	4-2-18 (6pm)	5-2-18	5-2-18, 12 noon	6-2-18	7-2-18
1.6	1.9	2.2	2.5	3.5	3.2	4.7

Treatment

After measured to identify and stop ongoing losses of potassium, replaced potassium 10 meq/hr, 10 - 40 mEq infused over 2 - 3h with continuous cardiac monitoring until serum potassium level of (2.5 - 3.5 mEq/L). After third day, patient serum K level reached to 2.5 mEq/L, switched to oral potassium replacement therapy and magnesium supplement along with tight measured of potassium. Oral supplementation is given; 50 - 100 mEq/d divided two-four times per day.

ECG was rechecked when serum potassium reached to 3.5 that showed no more premature ventricular contraction and converted to sinus rhythm. The patient was given potassium supplement, Amlodipine 10 mg, Aspirin 80 mg OD, Pantocid 40 mg prn, Carvedilol 6.25 BD, Atorvastatin 20 mg hs, oral hypoglycemic agents and gave health education about electrolyte imbalance. She was discharged from the hospital in stable condition with no further arrhythmias and gave follow up appointment to come back after 5 days to do aldosterone, rennin and other endocrine related disorder tests.

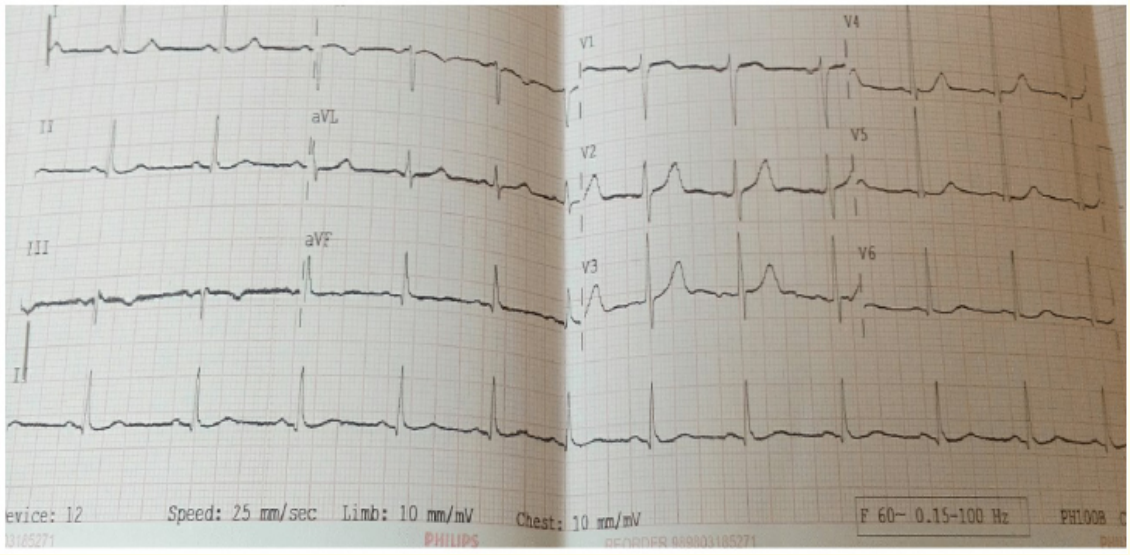

Figure 3: *Rechecked ECG after potassium reached back to 3.5 mmol/L.*

Discussion

Extracellular potassium concentration is normally maintained between 4.0 and 4.5 mEq/L. Ninety-five percent of total body potassium is intracellular; only 2% is extracellular. A 70-kg man, for instance, has about 3,920 mEq of potassium in the intracellular space but only 59 mEq in the extracellular space. Hypokalemia with hypertension can be seen in renovascular disease, Cushing's syndrome, primary aldosteronism, renin secreting tumors, congenital adrenal hyperplasia and other endocrine related disorders. Most hypokalemia is also seen in secondary to diuretic therapy. In this case, cause of hypokalemia is due to chronic diarrhea that in turn made the potassium ion loss led to acid-base imbalance. Symptoms of hypokalemia include weakness (especially if $K^+ < 2.5$), myalgia, constipation, and rhabdomyolysis. Patients can develop Premature Atrial Complexes, Premature Ventricular Contractions, Sinus bradycardia, Premature Arterial Tachycardia, Junctional tachycardia, AV Block, Ventricular tachycardia, and Ventricular Fibrillation. ECG changes include flattened T-waves, U-waves (due to prolonged Purkinje fiber repolarization), and prolonged QT.

Most cases of chronic hypokalemia are well tolerated, and if the patient has no symptoms, potassium would not be replaced intravenously unless the K^+ were below 2.5 mEq/L. If treatment were required, the underlying cause should be addressed. Replacement can be provided with an intravenous infusion of no greater than 20 mEq/hr or, even safer, PO administration. Patients who are hypokalemic by 1 mEq/L in the plasma are at least 2 - 300 mEq deficient in the body, thus replacement of extremely low levels. In hypertensive patients, it seems beneficial to aim for serum potassium levels 3.5 to 5.0 mEq/L.

Hypokalemia is independent risk factor contributing to reduced survival of cardiac patients and increased incidence of arrhythmic death. In severe hypokalemia, fatal ventricular tachycardia/fibrillation, prolongation of ventricular repolarization and rarely atrioventricular block is caused by inhibition of outward potassium currents and often associated with increased propensity for early after depolarizations [3,4]. Potassium ion concentrations are a major determinant in the magnitude of the electrochemical potential of cells, and hypokalemia makes it more likely that cells will depolarize spontaneously. Bellet., *et al*. reported extrasystoles with hypokalemic alkalosis that decreased with potassium administration. The findings of Gettes., *et al*. in 1962 showed that perfusion of low potassium solutions resulted in ventricular ectopic beats, ventricular tachycardia, and ventricular fibrillation [4].

Recently several studies have evaluated the relationship between potassium levels and arrhythmias in patients with and without hypertension who were receiving diuretic therapy. Although the most obvious ECG manifestation of hypokalemia is a prominent U wave, several cardiac conduction abnormalities such as prolonged action potential duration and QT interval, QTU alternans, early afterdepolarizations, and torsade de pointes ventricular tachyarrhythmia can also be seen [5,6]. Even when there is evidence of hypokalemia on a patient's electrocardiogram, physicians often miss the diagnosis. Thus, the present case report shows correlation between premature ventricular contraction appearance in ECG with severe hypokalemia and it can also help to notice and aware the occurrence of ventricular arrhythmias in patient with severe hypokalemia caused by loose motion.

Conclusion

Hypokalemia can cause cardiac arrhythmias and neurologic manifestations, which range from generalized weakness to ascending paralysis. Early detection of hypokalemia by clinical presentations and ECG changes can help to know the underlying disorders and can also predict diagnosis. Thus, clinicians should be knowledgeable about the electrocardiogram changes and manifestations of electrolyte imbalance to provide effective initial management for arrhythmia in emergency care setting to prevent serious arrhythmias and can save life.

Conflict of Interest

I declare there is no conflict of intertest to disclose.

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