Original Research Article

A study on clinical features and ECG changes in patients with hypokalemia in Konaseema Institute of Medical Sciences

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Abstract

Background : Hypokalemia is one of the commonly encountered electrolyte disturbances, and has the potential to increase the risk of arrhythmia. **Aim of the study :**To study clinical features and ECG(Electrocardiogram) changes in 50 pts of hypokalemia in ICU at Konaseema institute of medical sciences, Amalapuram.**Materials and Methods:** Prospective study was conducted on 50 patients with hypokalemia for duration of 2 years. 12 lead Electrocardiogram was done in all 50 patients with hypokalemia and observations were recorded.**Results** :In the present study in hypokalemia cases , most common ECG finding was,decrease in T wave height. ST depression & T wave inversion and prominent u wave. **Conclusion:** Hypokalemia could be studied easily by medical history and treated accordingly. Difficult cases of hypokalemia should be studied systematically to identify cause so that long term treatment can be applied.

Keywords: Hypokalemia, Electrocardiogram, Arrhythmia.

Introduction

One of the most common electrolyte complications are Potassium abnormalities in hospitalized patients. Potassium is one of the critical electrolytes involved in various cellular activities. Potassium loss in the body affects the quality of life of the patient and increases morbidity and mortality. Hypokalemia is one of the commonly encountered electrolyte disturbances, and has the potential to increase the risk of arrhythmia. Hypokalemia is defined as a potassium level <3.5 mmol/L, moderate hypokalemia as a potassium level of <3.0 mmol/L, and severe hypokalemia as a potassium level <2.5 mmol/L. Diarrhea and diuretic therapy are responsible for most cases of hypokalemia in the clinic[1,2]. U-wave development is a classic change in the ECG in patients with hypokalemia. A U wave is described as positive deflection after the T wave and it is often best observed in the mid-precordial leads. Under the condition of extreme hypokalemia, giant U waves may often merge and then smaller preceding T waves are covered[3].The level of serum potassium is vital for regulating depolarization and repolarization of the myocardium, and hypokalemia can alter the cardiac action potential and result in abnormities of cardiac conduction. Additionally, the P pulmonale pattern is occasionally observed in patients with hypo-kalemia, and it is transient and concomitant [7,8].These changes are responsible for findings on a surface electrocardiogram (ECG) associated with hypokalemia.When potassium levels are <2.7 mmol/L, changes in the ECG include dynamic changes in T-wave morphology (T-wave flattening and inversion), ST-segment depression, and U waves, which are often best seen in the mid-precordial leads (V1–V4)[4-8]. Furthermore, high potassium levels precipitate potentially life-threatening dysrhyt-hmias [9,10]. Therefore, serum potassium levels in patients who experience arrhythmia need to be checked, especially when they have diuretic therapy or diarrhea. Hypokalemia is often associated with hypomagnesemia,which increases the risk of malignant ventricular arrhythmias.Our study is about clinical features and ECG changes in 50 patients admitted in ICU with hypokalemia .

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Material and Methods

Prospective study was conducted on 50 patients with hypokalemia for duration of 2 years ie, from January 2017 to January 2019 in konaseema institute of medical sciences , Amalapuram ,Andhra Pradesh. Informed consent was taken from all the patients included in the study.Ethical permission was taken from the ethical committee.

Inclusion criteria

Patients with hypokalemia admitted in ICU and Patients who had concentrations of serum potassium of less than 3.5mEq. per liter.

Exclusion criteria

Patients of age < 20yrs and >80yrs and Patients who did not given consent

Methodology

All the patients were selected randomly.A questionnaire was prepared and clinical details were noted including age, gender, occupation, history of present illness, past history ,personal and drug history.Routine investigations were done including CBP. CUE. Serum potassium levels were done in the Biochemistry department and done in semiautomated analyser**.** Concentrations of serum potassium of less than 3.5 mEq.per liter was considered as hypo-kalemia.12 lead Electrocardiogram was done in all 50 patients with hypokalemia and observations were recorded. Various observations

were made on the electrocardiogram including rate, P-R interval, Q-T interval, amplitude of QRS complexes, deviations of S-T segment, amplitudes of T and U waves, and the incidence of ventricular and atrial premature contractions. Other abnormalities, such as left ventricular hypertrophy low voltage complexes, and ischemic changes.

Statistical analysis

Data will be entered in Microsoft Excel sheet and will be analysed using SPSS version 20.0 statistical software. Data will be depicted in the form of tables and charts.

Results

Table 1: Age distribution in present study

Age distribution in years	No. of cases	%
20-30	10	20%
31-40	20	40%
41-50	10	20%
51-60	05	10%
61-70	03	6%
71-80	02	4%
Total	50	100%

In the present study majority of the cases were among 31-40 years.ie, 40 % next common were among 20-30 years and 41-50 years.

Table 2: Gender distribution

Gender	Mild	Moderate	Severe	Total
Males	11(22%)	11(22%)	08(16%)	30(60%)
Females	14(28%)	05 (10%)	01(2%)	20 (40%)
Total	25(50%)	16(22%)	09(18%)	50(100%)

In the present study males (60%)were predominant when compared to females (40%).

Table 3: Clinical features in cases with hypokalaemia in Medical ICU

Severity of hypokalaemia	Serum potassium levels	Clinical symptoms	Total
Mild	3-3.5	Asymptomatic	15(30%)
		weakness & palpitations	10(20%)
Moderate	2.5-3.0	Muscle weakness & cramping	10(20%)
		Fasciculation	05(10%)
		Paralytic ileus	01 (2%)
Severe	< 2.5	Respiratory muscle paralysis	07(14%)
		Hypotension	02(4%)

Table 4:ECG changes in cases with hypokalemia in medical ICU

Severity ofhypokalaemia	Serum Potassiumlevels	ECG changes	Total
Mild	3.0 -3.5	Decrease in T wave height	20(40%)
Moderate	2.5-3.0	ST depression & T wave inversion	08(16%)
		Prominent U wave	08(16%)
Severe	< 2.5	Supra ventricular tachyaarrthmias	03 (5%)
		Torsedesdepointes	02(4%)
		VT,VF	04(8%)

In the present study In mild hypokalemia cases , most common ECG finding was, decrease in T wave height.In moderate hypokalemia cases,most common ECG finding was,ST depression & T wave inversion and prominent u waves.In Severe hypokalemia cases, most common ECG finding was ventricular tachycardia.

Table 5: Causes of hyokalemia

Causes	Mild	Moderate	Severe	Total
Diarrhea and vomiting	10 (20%)	10 (20%)	03 (6%)	23(46%)
Periodic paralysis	05 (30%)	02(4%)	05 (10%)	12(24%)
drug induced	05 (10%)	03 (6%)	01 (2%)	09(18%)
Thyroid disorder	05 (10%)	01 (2%)	-	06 (12%)
Total	25 (60%)	16 (32%)	09 (18%)	50 (100%)

In the present study most common cause of hypokalemia was due to Diarrhea and vomiting. Next common was periodic paralysis

Table 6: Lab parameters in patients with Hypokalemia in ICU

Investigation parameters	Males	Females	Total
Serum potassium(hypokalemia)	30 (60%)	20 (40%)	50(100%)
ECG changes	29(58%)	16 (32%)	50(100%)
Se.Magnesium (Hypomagnesimia)	08 (16%)	02 (4%)	10(20%)
serum thyroxine(thyrotoxicosis)	01 (2%)	05 (10%)	06(12%)
BP(increased)	09 (18%)	05(10%)	14(28%)

ABG	04 (8%)	02 (4%)	06(12%)

In the present study among 50 cases with hypokalemia,26 cases developed CVS manifestations and among 26 cases 30% showed increased BP,10% had Congestive heart failure and 12% had Arrhythmias. In the present study 03 males with hypokalemia died due to Arrhythmias and 01 female with hypokalemia died due to Hypoventilation.

Discussion

The earliest electrocardiographic (ECG) findings associated with hypokalemia is a decrease in T waves height. Then, ST depression and T inversion happens as serum potassium reduces further. Due to prolonged repolarization of ventricular Purkinje fibers, prominent U wave occurs(usually seen at V2 and V3 leads),frequently superi-mposed upon the T wave and therefore produces the appearance of a prolonged QT interval when serum potassium reduces to below 3 mEq/L. In our study 35 patients were symptomatic and 45 were showing ecg changes.All the observations were statistically significant Marti et al[9] During the study period, measurements of serum potassium for a total of 43 805 patients were performed.Of these patients, 4826 (11%) had hypokalemia(potassium<3.5 mmol /l) at presentation. Fifty-three (1%) patients with severe hypokalemia could be identified. Twenty-six (49%) patients were symptomatic, with weakness and muscle pain being the most common symptoms. Twenty (69%) patients had ECG changes with the presence of a U wave, followed by ST segment depression and ventricular extra systoles. Malnutrition and use of diuretics were the main causes of severe hypokalemia.In our study, we reported 7 cases of periodic paralysis over one year. In Agrawal et al study[10] , reported 40 cases of hypokalemic periodic paralysis in a period of 23 years. In an earlier series reported by Arya et al[11],a total of 22 cases of hypokalemic paralysis were reported ov In retrospective study from South India by Rao et al.[12] 31patients were detected over a period of 6 years. A recent prospective study from North India by Maurya and colleagues[13] reported 30 patients of HPP over years. Reports exist of rhabdomyolysis occurring with profound hypokalemia with serum potassium levels less Respiratory depression from severe impairment of skeletal muscle function is found in many patients. Many patients with potassium depletion may also have magnesium deficiency. In particular, loop diuretics (eg, furosemide) produce substantial serum and intracellular potassium and magnesium loss.Digoxin accelerates the excretion of magnesium by reducing its reabsorption at the renal tubules.The role of magnesium in maintaining intracellular potassium is particularly important in cardiac myocytes because it desensitizes them to the calcium-induced arrhythmogenic actions of cardiac glycosides. Routine determination of serum magnesium levels should be considered whenever the measurements of serum electrolytes are necessary in a patient. In our study,10 patients also had magnesium deficiency along with potassium. Patients Improved after dietary supplementation of magnesium.

Whang et al[14] recommend considering the repletion of both magnesium and potassium for patients with hypokalemia. Dietary sources of magnesium include whole-grain cereals, peas, beans, nuts, cocoa, seafood, and dark green vegetables.In our study,15 cases out of 50 had hypertension . After the use of potassium sparing diuretics, blood pressure came back to normal. Whelton et al [15] recently conducted a meta-analysis of randomized controlled trials evaluating the effects of oral potassium supplementation on blood pressure. This analysis included 33 clinical trials involving 2609 participants.

In these trials, the use of potassium supplementation was the only difference between the intervention and control arms. Dosages of potassium (mostly in the form of potassium chloride) ranged from 60 mmol/dL to greater than 100 mmol/dL. The results demonstrated that potassium supplementation was associated with a significant reduction in mean systolic and diastolic blood pressure (–4.4 mm Hg and –2.4 mm Hg, respectively; P<.001). The greatest effects were observed in participants who had a high concurrent sodium intake. This analysis suggests that low potassium intake may play an important role in the genesis of high blood pressure. Thus, the authors recommended increased potassium intake for the prevention and treatment of hypertension. Based on the strength of the available data, the Joint National Committee for Prevention, Detection, Evaluation, and Treatment. They estimated that the protective effect of antihypertensive treatment on mortality might be halved by the induction of sudden death following potassium loss.In our study 5 cases of hypokalemia showed CHF. Patients CHF subsided after giving spirnolactone. Leier et al [16] suggested that virtually all patients with CHF should receive potassium supplementation, a potassium-sparing diuretic, or an ACE inhibitor. This is a prudent management strategy in light of the potentially dire consequences of hypokalemia in these patients.In the heart, hypokalemia cause arrhythmias because of more complete recovery from sodium-channel inactivation, making the triggering of an action potential less likely. In addition, the reduced extracellular potassium (paradoxi-cally) inhibits the activity of the IKr potassium current and delays ventricular repolarization. This delayed repolarization may promote reentrant arrhythmias.Nolan et al[17]found that low serum potassium levels were related to sudden cardiac death in the United Kingdom Heart Failure Evaluation and Assessment of Risk Trial (N = 433). Grobbee and Hoes[18] reported similar results in an examination of published randomized trials and recent case-control studies; patients with hypertension who were prescribed non–potassium-sparing diuretic had approximately twice the risk of sudden cardiac death compared with users of potassium-sparing therapy. The authors recommended using thiazide diuretics at a low dose only, and adding a potassium-sparing diuretic drug when higher diuretic doses are needed. They estimated that the protective effect of antihypertensive treatment on mortality might be halved because of the induction of sudden death following potassium loss. In our study 05cases out of 50 patients of hypokalemia showed ventricular arrhythmia out of which two had sudden cardiac death and others revived and serum potassium corrected. The observations were statistically significant. In our study, we reported 13 cases of drug induced hypokalemia over six months.Ruberman and colleagues found a twofold increase in sudden coronary death in patients treated with diuretics during the 5-year period following an acute myocardial infarction [19]. Furthermore, in the multiple Risk Factor Intervention Study (MRFIT), the incidence of sudden death was increased twofold in the hypertensive group with EKG abnormalities receiving special intervention as compared with those receiving the usual care. Speculation has been raised concerning a possible role for diuretic therapy, because diuretics were the prescribed step-l antihypertensive drug in the special intervention group.

Conclusion

Hypokalemia could be studied easily by medical history and treated accordingly. Difficult cases of hypokalemia should be studied systematically to identify cause so that long term treatment can be applied.Recognizing plasma potassium dynamics and that hypokalemia is common and is often inadequately managed, it may be beneficial to pay more attention to hypokalemia and to maintain plasma potassium levels in the upper normal range to prevent the complications associated with hypokalemia. Hypokalemia may be of special importance in patients with cardiovascular diseases such as hypertension, coronary artery disease, heart failure and arrhythmia, especially if treated with nonpotassium-sparing diuretics, beta-adrenoceptor agonists and/or insulin. The more at risk of fatal arrhythmia and sudden cardiac death a patient is, the more attention should be given to the potassium homeostasis .

References

1. El-Sherif N,Turitto G. Electrolyte disorders and arrhythmogenesis. Cardiol J. 2011; 18:233-245.
2. Schulman M,Narins RG. Hypokalemia and cardiovascular disease. Am J Cardiol. 1990; 65:4E-9E.

3. Yan GX,Lankipalli RS,Burke JF et al.Ventricular repolarization components on the electrocardiogram: cellular basis and clinical significance.J Am Coll Cardiol.2003; 42:401-409.
4. Wang A, Li X, Dookhun MN, Zhang T, Xie P, Cao Y. A female patient with hypokalaemia-induced J wave syndrome: An unusual case report. Medicine (Baltimore).2017;96(38): e8098.
5. Kishimoto C, Tamaru K,Kuwahara H. Tall P waves associated with severe hypokalemia and combined electrolyte depletion. J Electrocardiol. 2014; 47:93-94.
6. Khan MU, Patel AG, Wilbur SL et al. Electrocardiographic changes in combined electrolyte depletion. Int J Cardiol. 2007; 116:276-278.
7. Chang KY, Lee SH, Park HS, Ko SH, Ahn YB, Kim HW. Severe hypokalemia and thyrotoxic paralysis from painless thyroiditis complicated by life-threatening polymorphic ventricular tachycardia and rhabdomyolysis. Intern Med. 2014; 53(16):1805-8.
8. Kumar V, Khosla S,Stancu M. Torsade de pointes induced by hypokalemia from imipenem and piperacillin. Case Rep Cardiol.2017; 2017:4565182.
9. Grischa Marti, Christoph Schwarz, Alexander B Leichtle, Georg-Martin Fiedler, Spyridon Arampatzis, Aristomenis K Exadaktylos, Gregor Lindner.Etiology and symptoms of severe hypokalemia in emergency department patients.Eur J Emerg Med. 2014;21(1):46-51.
10. Agrawal AK, Singla S, Kumar A. Hypokalemic paralysis; Medicine Updates Vol:19 (part 1) Noida, Association of physicians of India, 2009, 42-9.
11. Arya SN. Periodic paralysis. JIACM. 2002; 3:374-82.
12. Rao N, John M, Thomas N, Rajaratnam S, Seshadri MS. Aetiological, clinical and metabolic profile of hypokalaemic periodic paralysis in adults: A single-centre experience. Natl Med J India. 2006; 19:246-9.
13. Maurya PK, Kalita J, Misra UK. Spectrum of hypokalaemic periodic paralysis in a tertiary care centre in India. Postgrad Med J. 2010; 86:692-5.
14. Robert Whang, Edmund B. Flink, Thomas Dyckner; et al: Magnesium Depletion as a Cause of Refractory Potassium Repletion, Arch Intern Med. 1985;145(9):1686-1689.
15. PK Whelton, J He, JA Cutler, FL Brancati, LJ Appel, D Follmann, MJ Klag. Meta-Analysis- Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials: JAMA. 1997;277(20):1624-32.
16. Leier CV, Dei Cas L, Metra M. Clinical relevance and management of the major electrolyte abnormalities in congestive heart failure:hyponatremia,hypokalemia, and hypomag-nesemia. Am. Heart J. 1994; 128:564-574.
17. Nolan J, Batin PD, Andrews R, Lindsay SJ, Brooksby P, Mullen M et al. Prospective study of heart rate variability and mortality in chronic heart failure: results of the United Kingdom heart failure evaluation and assessment of risk trial (UK-heart). Circulation. 1998; 98:1510-1516.
18. DE Grobbee, AW Hoes. Review Non-potassium-sparing diuretics and risk of sudden cardiac death J Hypertens. 1995; 13(12 Pt 2):1539-45.
19. Ruberman W, Weinblatt E, Goldberg JD, Frank CW, Chaudary BS, Shapiro S. Ventricular premature complexes and sudden death after myocardial infarction. Circulation. 1981; 64:297-305.

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