

electrocardiogram pattern

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ELECTROCARDIOGRAM PATTERN PRE AND POST POTASSIUM CORRECTION IN HYPOKALEMIA

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ABSTRACT

Background: Hypokalemia with ECG changes is very hazardous; therefore, it is essential to correct hypokalemia to avoid life-threatening cardiovascular complications.

Objective: To know the ECG pattern pre and post potassium correction in patients with hypokalemia.

Methods and results: A prospective study with longitudinal observational design in hospitalized hypokalemic patients who received intravenous potassium correction at Dr Wahidin Sudirohusodo Hospital, Makassar during February - May 2018. ECG changes are determined based on ECG examination. Potassium correction is given based on the potassium deficit formula. Data analysis using SPSS version 22 with McNemar and Chi-Square statistical test. The study included 80 subjects consisting of 26 males and 54 females, the mean age of $48,8 \pm 16$ years, 53,8% of moderate and 38,8% of severe hypokalemia, 85% having ECG changes. The ECG patterns were long QT (30%), prominent U (30%), T-inverted (21,2%), ST depression (15%), T-flat (10%), VES (6,3%), SVT (3,8%), first degree AVB (3,8%), and AF (2,5%). Significant improvement found in long QT and prominent U post-correction ($p < 0.05$). Decrease of post-correction potassium levels found 100% of prominent U and 75% of long QT became normal.

Conclusion: The ECG patterns in hypokalemia are mostly long QT and prominent U. Potassium correction may provide an improvement in ECG patterns even if potassium levels still below the normal range.

KEYWORDS ECG, hypokalemia, arrhythmia, long QT, prominent U

Introduction

Hypokalemia is an electrolyte abnormality commonly found in clinical practice.[1] In the general population, data on hypokalemia are difficult to estimate, but most likely, less than 1% of healthy subjects have potassium levels $< 3,5$ mEq/L. It is estimated that up to 21% of patients in the inpatient's department have potassium levels $< 3,5$ mEq/L, with 5% having potassium

² levels < 3 mEq/L.[2,3] Inadequate management of hypokalemia is found in 24% of patients.[4,5]

Hypokalemia is generally associated with the 10-fold high of cardiovascular morbidity and mortality, mainly due to cardiac arrhythmias or sudden cardiac death.[5] Worldwide, around three million people suffer sudden cardiac death (death due to heart disease in 1 hour) each year. Of these, around 0,5 million people aged < 50 years old.[4] These deaths often arise due to the interaction between substrate and complex triggers. Disruption of potassium homeostasis in heart cells is one of the triggers.[4,6]

Cardiac electrical activity is mediated by ionic shifts across the cellular membrane. An electrocardiogram records electrical activity that arises when the myocardium contracts. Changes in electrolyte levels can affect depolarization and/or repolariza-

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Table 1 Characteristic distribution (n = 80)

Variables		n	%
Gender	Male	26	32,5
	Female	54	67,5
Age	≤ 60 years old	55	68,8
	>60 years old	25	31,3
BMI	Normal	30	37,5
	Underweight	44	55
	Overweight	6	7,5
History of smoking	Yes	21	26,3
	No	59	73,8

tion and can cause electrocardiogram (ECG) changes. Although in some patient, ECG changes are not accompanied by serum potassium abnormalities, ECG is a screening tool that is useful for measuring the severity of serum potassium abnormalities and the urgency of therapeutic interventions.[7,8] Hypokalemia with ECG changes is very hazardous, so it is essential to correct hypokalaemia to avoid life-threatening complications rapidly. This study was conducted to obtain ECG pattern pre and post potassium correction in hypokalemic patients at Wahidin Sudirohusodo Hospital in Makassar.

Methods

This research was conducted on February - May 2018 using a longitudinal observational design. Location at Dr Wahidin Sudirohusodo Makassar, South Sulawesi. The population in this study were all inpatient of Dr Wahidin Sudirohusodo Hospital in Makassar with a diagnosis of hypokalemia aged ≥18 years and receiving intravenous potassium replacement therapy, whereas exclusion criteria were antiarrhythmic and digitalis users, acute myocardial infarction, end-stage renal disease, hypertension and hyperglycemic crisis. The number of samples meeting the inclusion criteria was 80 subjects by using consecutive sampling.

This research has been approved by the Ethics Committee of Medical Faculty of Hasanuddin University. All patients have granted informed consent before inclusion.

Technical Information

Potassium levels <3,5 mEq/l are classified as hypokalemia. Intravenous potassium correction using potassium chloride is given based on the potassium deficit formula in mEq = (3,5-potassium serum) body weight 0.4.[9] The ECG changes are determined based on ECG examination. The ECG interpreted was ECG when there was a decrease in potassium and 6 hours post intravenous potassium correction.[10]

Statistics

Data analysis was performed using the Statistical Package for Social Science (SPSS) program version 22 with McNemar and Chi-Square statistical tests. Statistical test results are significant if $p < 0,05$.

Results

Total of 80 patients in this study with hypokalemia ranging in age from 18 to 70 years old with a mean age $48,8 \pm 16$ years. Table 1 shows that most of the subjects were women (67,5%), aged ≤60 years (68,8%) with body mass index (BMI) in underweight category (55%), and history of smoking were 21 subjects (26,3%).

Out of the 80 subjects, 85% experienced ECG changes pre and post potassium correction. Table 2 shows significant changes in long QT and prominent U ($p < 0,05$). However, there were no significant changes in rhythm, first-degree atrioventricular block (AVB), ST depression, T-flat or inversion, ventricular extrasystole (VES), atrial extrasystole (AES), and right bundle branch block (RBBB) after potassium correction compared to before correction ($p > 0,05$). The ECG patterns in hypokalemia are mostly sinus rhythm (92,5%), normal QT intervals (66,2%), long QT (30%), prominent U (30%), normal PR intervals and ST segments (96,2% and 85%), ST depression 15%, normal T wave (68,8%), T-inverted (21,2%).

Table 3 shows that there was no significant relationship between post-correction potassium levels and ECG changes ($p > 0,05$). Table 4 shows that at decreased potassium levels after correction, 2 subjects with T-inverted became normal, 1 subject with ST depression converted to isoelectric, 1 subject with short QT became normal, 3 of 4 subjects with long QT became normal and 1 subject became short QT, and 3 subjects with prominent U being normal.

Discussion

In this study, there was more female than men, mostly the age of 60 years, BMI in the underweight category and not smoking.

Table 2 Comparison of ECG patterns pre and post potassium correction.

Pre correction			Post correction				<i>p</i>
	n	%	Sinus rhythm	SVT	AF	Sinus arrhythmia	
Sinus rhythm	74	92,5	74	0	0	0	*
SVT	3	3,8	3	0	0	0	
AF	2	2,5	2	0	0	0	
Sinus arrhythmia	1	1,3	1	0	0	0	
			VES		Non VES		
VES	5	6,3	1		4		0,125
Non VES	75	93,7	0		75		
			AES		Non AES		
AES	1	1,3	0		1		*
Non AES	79	98,7	0		79		
			AVB1		Non AVB1		
AVB1	3	3,8	0		3		>0,999
Non AVB1	77	96,2	4		73		
			RBBB		Non RBBB		
RBBB	2	2,5	1		1		>0,999
Non RBBB	78	97,5	0		78		
			ST depression		Non ST depression		
ST depression	12	15	2		10		0,454
Non ST depression	68	85	6		62		
			T-flat/inverted		Non T-flat/inverted		
T-flat/inverted	25	31,2	9		16		0,052
Non T-flat/inverted	55	68,8	6		49		
			Long QT		Non long QT		Short QT
Long QT	24	30	6		17	1	0,036
Non long QT	53	66,2	5		47	1	
Short QT	3	3,8	0		3	0	
			Prominent U		Non prominent U		
Prominent U	24	30	3		21		<0,001
Non prominent U	56	70	2		54		

Mc Nemar SVT: supraventricular tachycardia * Can not be tested AF: atrial fibrillation AVB1: first degree atrioventricular block

Table 3 Correlation of post correction potassium levels and ECG changes.

Potassium levels post correction			ECG changes post correction		P
			Changed	No change	
Increased	n	63	53	10	0.888
	%	78.8	84.1	15.9	
Constant	n	7	6	1	
	%	8.8	85.7	14.3	
Decreased	n	10	9	1	
	%	12.5	90	10	
Chi-square					

Study of 872 hypokalemic patients by Kleinfeld M, et al. (1993) reported that hypokalemia is more common in old age and women. Differences in mass body composition based on age and gender made these results, which is physiologically total body potassium in the group is lower.[11] More common in young age might be related to the aetiology of hypokalemia, but this study did not determine the aetiology. Subjects who do not have a history of smoking were more numerous because of the influence of an eastern culture that considers smoking in women is not a good thing. As with Osman EA, et al. (2011) in his study of 60 subjects in Sudan reported no association between smoking and serum potassium levels.[12]

In this study, ECG patterns were mostly long QT, and prominent U, followed by T-inverted and ST depression. While SVT, AF and first-degree AVB only occurred in a small number of subjects. This study showed a significant improvement in long QT and prominent U after correction ($p < 0,05$). Characteristics of ECG changes in hypokalemia occur due to a slowing of repolarization of myocardial cells. Hypokalemia causes reduced membrane permeability to potassium, causing a decrease in potassium efflux. In a literature study by Glover P in 1999, reported that the prominent U wave in leads V2-4 is the earliest ECG pattern and most commonly found in hypokalemia. Intra-ventricular conduction defects due to hypokalemia are rarely obtained.[13]

A study of 53 hypokalemic patients with acute gastroenteritis by Jalani NT in 2005, reported 94,3% had hypokalemic ECG signs: 19 with prominent U (75%) and T-flat or T-inverted (66%), then followed by a prolongation of the PR interval (24%) and ST depression (9%).[14] Marti G et al. in 2014 conducted a study of 53 severe hypokalemia patients, reported that 69% of subjects had an ECG changes, where prominent U was the most common hypokalemic ECG sign (24%), followed by multiple ST depression and multiple ventricular extrasystoles (21%).[15]

The myocardial resting membrane potential is determined by the ratio of intra and extracellular potassium. Hypokalemia causes the resting membrane potential to become more negative, and the membrane to be hyperpolarized, causing an increase in phase 0 velocity.[6] Heart cells become less responsive to the stimulus, and the rate of cardiac contractility will decrease. In-

creased depolarization in myocardial cells potentially causes ectopies.[16] The effect of hypokalemia on cell membranes is an increase in resting membrane potential, which results in an increase in membrane automaticity, prolongation of potential action, especially phase 3 repolarization and refractory period.[6,7] That all makes prolongation of QT interval, flattening of T wave and prominence of U wave.[6]

The ECG changes are not related to the degree of hypokalemia in each patient. But typical changes occur in 90% of cases when plasma potassium levels are $< 2,5$ mmol/L. The prominent U wave in lead V2-4 is the earliest encountered. Slowing ventricular repolarization causes an increase in the relative refractory period, increasing the risk of re-entrant tachycardia. The most common cardiac arrhythmias are AES, atrial tachycardia with or without AVB, and SVT. Atrioventricular blocks other than prolonged PR intervals are rarely found.[13]

There was no significant relationship between potassium post-correction level with ECG changes ($p > 0,05$). At decreased potassium levels after correction, there were 3 subjects with prominent U where 100% became normal, while 4 subjects with long QT, 75% became normal and 25% became shortened QT intervals. The ECG changes are frequently reflected serum electrolyte alteration, but also may be due to local electrolyte changes within the heart itself, which may not manifest significant changes in serum electrolytes. A study conducted by Choy AM et al. (1997) on 8 congestive heart failure (CHF) patients and 12 healthy controls found improvement in the QT interval, U wave and T wave after administration of potassium infusion in 13 CHF patients, but this effect was not found in control group.[17] Small changes in potassium levels can have severe effects on the heart. Potassium as a primary ion that plays a role in cardiac repolarization.[18] The prolonged QT interval reflects abnormal cardiac repolarization, a hazardous predictor of arrhythmias because it is early after depolarization risk factor that can trigger torsades de pointes.[19] This indicates the importance of potassium therapy in hypokalemic patients.

Table 4 Distribution of ECG changes based on post correction potassium levels

ECG pre correction		Potassium levels post correction								
		Increased			Constant			Decreased		
		Normal	Flat	Inverted	Normal	Flat	Inverted	Normal	Flat	Inverted
T wave	Normal	34	0	6	6	0	0	8	0	0
	Flat	4	0	3	0	0	1	0	0	0
	Inverted	11	2	3	0	0	0	2	0	0
QT interval		Normal	Long QT	Short QT	Normal	Long QT	Short QT	Normal	Long QT	Short QT
	Normal	40	4	1	3	0	0	4	1	0
	Long QT	12	5	0	2	1	0	3	0	1
	Short QT	1	0	0	1	0	0	1	0	0
P-R interval		Normal	AVB1		Normal	AVB1		Normal	AVB1	
	Normal	56	4		7	0		10	0	
	AVB1	0	0		0	0		0	0	
ST segment		Isoelectric	ST depression		Isoelectric	ST depression		Isoelectric	ST depression	
	Isoelectric	49	4		5	1		8	1	
	ST depression	8	2		1	0		1	0	
U wave		Normal	Prominent		Normal	Prominent		Normal	Prominent	
	Normal	41	2		5	0		7	0	
	Prominent	16	3		2	0		3	0	
Rhythm		Sinus rhythm			Sinus rhythm			Sinus rhythm		
	Sinus rhythm	57			7			10		
	SVT	3			0			0		
	AF	2			0			0		
	Sinus arrhythmia	1			0			0		
RBBB		Normal	CRBBB	ICRBBB	Normal	CRBBB	ICRBBB			
	CRBBB	0	0	1	0	0	0			
	ICRBBB	0	0	0	1	0	0			
Extrasistole		Normal	VES	AES						
	VES	3	0	1						
	Quadrigeminal VES	0	1	0						
	AES	1	0	0						

CRBBB: complete RBBB ICRBBB: incomplete RBBB

Conclusion

The ECG patterns in hypokalemia are mostly long QT and prominent U followed by T-inverted and ST depression. Potassium correction can provide an improvement in the ECG pattern even the potassium levels still below the normal range, especially the improvement of the QT and U wave intervals. We need an ECG examination in detecting and monitoring hypokalemia therapy. It is essential to provide immediate potassium therapy in hypokalemic patients to avoid lethal arrhythmias to reduce the risk of cardiovascular morbidity and mortality.

Competing Interests

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No conflict of Interest.

Funding

None

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