A CORRELATIONAL STUDY BETWEEN CHANGES IN SERUM ELECTROLYTES (NA, K, CL, MG) AND ACUTE MYOCARDIAL INFARCTION COMPLICATION

Ketan Bharatbhai Parmar[1], Raj N. Sharma[2], Jyotin Shah[3]

[1]Registrar, Department of Critical Care Medicine, Sterling Hospital, Rajkot, Gujarat.
[2]Consultant Physician, Department of General Medicine, Shalby Hospital, Ahmedabad, Gujarat.
[3]Professor, Department of Medicine, C.U. Shah Medical College, Surendranagar, Gujarat.

ABSTRACT

BACKGROUND

The term "arrhythmia" refers to any change from the normal sequence of electrical impulses. The electrical impulses may happen too fast, too slowly or erratically causing the heart to beat too fast, too slowly or erratically. When the heart doesn't beat properly, it can't pump blood effectively. When the heart doesn't pump blood effectively, the lungs, brain and all other organs can't work properly and may shutdown or be damaged. Normally, the heart's most rapidly firing cells are in the sinus (or sino-atrial or SA) node making that area a natural pacemaker. Under some conditions, almost all heart tissue can start an impulse of the type that can generate a heartbeat. Cells in the heart's conduction system can fire automatically and start electrical activity. This activity can interrupt the normal order of the heart's pumping activity. Secondary pacemakers elsewhere in the heart provide a "backup" rhythm when the sinus node doesn't work properly or when impulses are blocked somewhere in the conduction system. An arrhythmia occurs when the heart's natural pacemaker develops an abnormal rate or rhythm. The normal conduction pathway is interrupted. Another part of the heart takes over as pacemaker.

The aim of the study is to observe the prevalence of various electrolyte (Na, K, Cl and Mg) imbalances in complications of arrhythmias.

MATERIALS AND METHODS

This is a prospective study in which the patient admitted with signs and symptoms of cardiac arrhythmias diagnosed clinically, 100 cases were selected over 1 year.

RESULTS

The serum magnesium, sodium and potassium levels were significantly lower in the AMI patients at baseline and gradually becomes near normal on 4th day. K and Mg are showing significant difference between pre and post values in males and Mg show significant difference between pre and post values day 1 and day 5 in females with arrhythmia.

CONCLUSION

Persistent hyponatraemia is indication of worsening cardiac failure and cardiogenic shock. There is also relationship between serum potassium and QTc interval, so estimation of sodium, potassium, chlorine and magnesium levels in arrhythmia patients can help to assess prognosis.

KEYWORDS

Arrhythmias, Electrolyte Imbalances, Hyponatraemia.

HOW TO CITE THIS ARTICLE: Parmar KB, Sharma RN, Shah J. A correlational study between changes in serum electrolytes (Na, K, Cl, Mg) and acute myocardial infarction complication. J. Evid. Based Med. Healthc. 2017; 4(64), 3860-3865. DOI: 10.18410/jebmh/2017/771

BACKGROUND

The term "arrhythmia"[1] refers to any change from the normal sequence of electrical impulses. The electrical impulses may happen too fast, too slowly or erratically causing the heart to beat too fast, too slowly or erratically. When the heart doesn't beat properly, it can't pump blood effectively. When the heart doesn't pump blood effectively, the lungs, brain and all other organs can't work properly and may shutdown or be damaged. Normally, the heart's most rapidly firing cells are in the sinus (or sino-atrial or SA) node making that area a natural pacemaker. Under some conditions, almost all heart tissue can start an impulse of the type that can generate a heartbeat. Cells in the heart's conduction system can fire automatically and start electrical activity. This activity can interrupt the normal order of the heart's pumping activity. Secondary pacemakers elsewhere in the heart provide a "backup" rhythm when the sinus node doesn't work properly or when impulses are blocked somewhere in the conduction system. An arrhythmia occurs when the heart's natural pacemaker develops an abnormal rate or rhythm. The normal conduction pathway is

Financial or Other, Competing Interest: None.
Submission 20-07-2017, Peer Review 27-07-2017,
Acceptance 08-08-2017, Published 10-08-2017.
Corresponding Author:
Dr. Raj N. Sharma,
Consultant Physician, Department of General Medicine,
Shalby Hospital, Ahmedabad, Gujarat.
E-mail: dr.rajssharma@gmail.com
DOI: 10.18410/jebmh/2017/771

interrupted. Another part of the heart takes over as pacemaker.

When arrhythmias are severe or last long enough to affect how well the heart works, the heart may not be able to pump enough blood to the body. This can cause you to feel tired, lightheaded or may make you pass out. It can also cause death. Tachycardia can reduce the heart's ability to pump causing shortness of breath, chest pain, lightheadedness or loss of consciousness. If severe, it can also cause heart attack or death. Arrhythmias can produce a broad range of symptoms from barely perceptible to cardiovascular collapse and death.[2] A single premature beat maybe felt as a "palpitation" or "skipped beat." Premature beats that occur often or in rapid succession may cause a greater awareness of heart palpitations or a "fluttering" sensation in the chest or neck. When arrhythmias last long enough to affect how well the heart works, more serious symptoms may develop fatigue, dizziness, lightheadedness, fainting (syncope) or near-fainting spells, rapid heartbeat or pounding, shortness of breath, chest pain and in extreme cases, collapse and sudden cardiac arrest. The four main types of arrhythmia are premature (extra) beats, supraventricular arrhythmias, ventricular arrhythmias and bradyarrhythmias. Premature (extra) Beats- Premature beats are the most common type of arrhythmia. They are harmless most of the time and often don't cause any symptoms. When symptoms do occur, they usually feel like fluttering in the chest or a feeling of a skipped heartbeat. Most of the time, premature beats need no treatment, especially in healthy people. Premature beats that occur in the atria (the heart's upper chambers) are called premature atrial contractions or PACs. Premature beats that occur in the ventricles (the heart's lower chambers) are called premature ventricular contractions or PVCs. In most cases, premature beats happen naturally. However, some heart diseases can cause premature beats. They also can happen because of stress, too much exercise or too much caffeine or nicotine. Supraventricular arrhythmias- Supraventricular arrhythmias are tachycardias (fast heart rates) that start in the atria or Atrioventricular (AV) node. The AV node is a group of cells located between the atria and the ventricles. Types of supraventricular arrhythmias include Atrial Fibrillation (AF), atrial flutter, Paroxysmal Supraventricular Tachycardia (PSVT) and Wolff-Parkinson-White (WPW) syndrome. In the production of experimental arrhythmias, potassium, calcium, sodium and magnesium plays an important role. The cardiac ionic currents kinetics can be altered by electrolyte disorders and depending on the changes can promote proarrhythmic or arrhythmic effects. The normal electrical behaviour of the heart depends on the transmembrane ionic gradients and the time and voltage dependent alterations of their conductance.

In the normal cardiac tissue, abnormal electrolytes may generate or facilitate cardiac arrhythmias. By modulating the conduction of ions across specific cardiac membrane channels, electrolyte disorders are exerted and this results in cardiac arrhythmias. The most important determinant of resting membrane potential and the most abundant intracellular cation is potassium. The rate of change, the direction and extracellular concentration affects the EP effects of potassium. The effect of potassium on the RMP is modulated by the simultaneous calcium concentration. The elevated calcium level causes a decrease in the depolarising effect of elevated potassium level and lower calcium levels diminish the depolarisation caused by hypokalaemia.[3] The potassium currents are effected by electrolyte concentration and the instantaneous inward rectification on depolarisation is because of the magnesium block. The low potassium decreases the delayed rectifier current (I_{kr}). The transient outward potassium one type is affected by neurotransmitters and is activated by voltage and the other type is activated by intracellular calcium. Potassium opens in the presence of high intracellular calcium and sodium.[4] The factors that affect the transcellular shift of potassium from inside to outside are acidosis, alpha adrenergic receptor stimulation, digitalis and solvent drag and from outside to inside is affected by alkalosis, β_2-adrenergic receptor stimulation and insulin. The most common electrolyte abnormality is hypokalaemia. It results from decreased potassium levels intake, transcellular shift, increased renal or extrarenal losses. Causes of potassium hypokalaemia is decreased intake, potassium shift into the cell.[5]

Renal potassium loss is due to increased mineralocorticoid effects, primary or secondary aldosteronism, ectopic ACTH producing tumour or Cushing syndrome, Bartter's syndrome, licorice and renin producing tumour. Hyperkalaemia result from either decreased excretion or a shift of potassium from within the cell. Hyperkalaemia is compromised renal function in association of a variety of nephrotoxic medications. Hyperkalaemia is associated with increased membrane permeability to potassium, a consequence of an increase in the inward going rectifier and the delayed rectifier current. The rate of repolarisation is accelerated and the AP duration is shortened. It shortens the plateau of Purkinje fibers, the dispersion of repolarisation in the ventricle is decreased. The atrial myocardium is sensitive and the effects of hyperkalaemia depend on the tissue involved. The ventricular myocardium being less sensitive and the sino-atrial and His bundle being least sensitive. The objective of the study is to observe the prevalence of various electrolyte (Na, K, CL and Mg) imbalances in complications of arrhythmias.

MATERIALS AND METHODS

This is a prospective study in which the patient admitted with signs and symptoms of cardiac arrhythmias diagnosed clinically, both male and female in Osmania Medical College, Hyderabad, over a period of June, 2015 to July 2016. 100 cases were selected over 1 year.

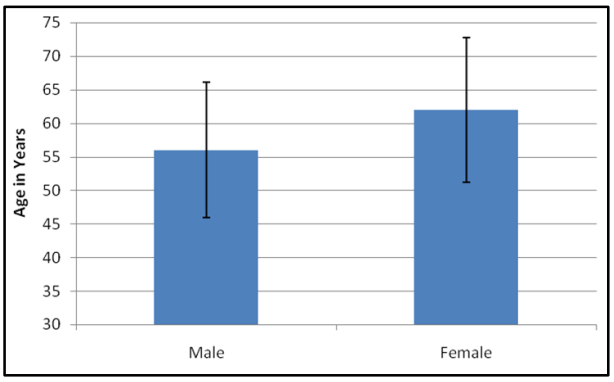
Inclusion Criteria- Patients presented with symptoms of cardiac arrhythmias within 48 hours of onset. History of chest discomfort. ECG changes of arrhythmias and rise of cardiac enzymes.

Exclusion Criteria- History of chronic ischaemic heart disease and patients with chronic kidney disease.

Blood sample cases selected were subjected to a detailed history and thorough physical examination, routine investigation like haemoglobin, blood count, urine examination, blood sugar, serum creatinine, serum electrolytes and cardiac enzymes was performed in cases. The baseline data was taken of the patients who fulfilled the selection criteria. The purpose and procedure of the study was explained to the patients and consent was taken from all patients. Prior to the study, blood sample of the subjects was taken on the day one of admission and on day 5th. The descriptive analysis was done to find mean and standard deviation.

RESULTS

Age distribution among the 100 subjects. The mean age of male patients was 56.28 ± 10.82 and female patients was 62.11 ± 10.99.



Age Distribution

According to parametric paired t-test, p-value show significant difference between pre and post values on day 1 and day 5 in both groups except in p-value for CL showing no significant difference between pre and post values of Cl of day 1 and day 5 in male hypertensive, p-value for Na, K there is no significant difference between pre and post values of Na, K of day 1 and day 5 in females with hypertension and also p-value for CL is 0.228 showing that there is no significant difference between pre and post values of CL of day 1 and day 5 in females without hypertension.

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (hypertensive)	Pre (Day 1)	138.98	3.25	0.147	4.08	0.80	0.213	96.7	4.82	0.923	2.10	0.20	0.000
	Post (Day 5)	140.8	0.98		4.32	0.36		96.8	2.97		2.55	0.23	
Male (nonhypertensive)	Pre (Day 1)	138.56	3.51	0.383	3.63	0.39	0.003	98.1	1.81	0.001	2.01	0.17	0.000
	Post (Day 5)	140.7	0.78		4.04	0.45		99.3	1.47		2.49	0.22	
Female (with hypertension)	Pre (Day 1)	138.67	2.62	0.051	4.24	0.96	0.585	98.20	2.68	0.018	1.86	0.28	0.001
	Post (Day 5)	140.34	0.98		4.11	0.49		99.04	2.65		2.29	0.39	
Female (nonhypertensive)	Pre (Day 1)	139.7	1.45	0.002	3.76	0.43	0.000	99.7	3.87	0.228	2.00	0.15	0.000
	Post (Day 5)	140.4	1.29		4.26	0.54		98.6	2.85		2.48	0.24	

Table 1. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Hypertension in Both Genders

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (diabetic)	Pre (Day 1)	136.9	2.88	0.025	4.43	0.75	0.826	98.3	5.71	0.550	1.93	0.36	0.000
	Post (Day 5)	139.8	0.28		4.37	0.16		99.03	3.69		2.35	0.45	
Male (nondiabetic)	Pre (Day 1)	139.09	2.95	0.210	3.58	0.65	0.001	99.7	4.03	0.755	1.98	0.16	0.000
	Post (Day 5)	140.9	0.95		4.05	0.45		99.9	2.37		2.45	0.24	
Female (diabetic)	Pre (Day 1)	139.56	3.51	0.239	4.07	0.69	0.220	97.8	4.12	0.755	1.83	0.20	0.000
	Post (Day 5)	140.45	1.18		4.33	0.37		98.05	2.90		2.33	0.31	
Female (nondiabetic)	Pre (Day 1)	139	1.39	0.000	3.66	0.39	0.001	98.25	2.42	0.002	1.98	0.17	0.000
	Post (Day 5)	140	1.14		4.06	0.52		99.74	2.88		2.40	0.28	

Table 2. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Diabetes in Both Genders

In males, K and Cl show no significant difference between pre and post.

Na and Cl has no significant difference between pre and post values of Na and Cl of day 1 and day 5 in male nondiabetics.

Na, K and Cl show no significant difference between pre and post values of Na, K and Cl of day 1 and day 5 in females with diabetes.

Na, K, Cl and Mg show significant difference between pre and post values of Na, K, Cl and Mg on day 1 and day 5 in females without diabetes.

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (cardiac failure)	Pre (Day 1)	135.5	4.48	0.004	4.08	0.89	0.335	98.4	5.04	0.061	1.95	0.25	0.000
	Post (Day 5)	140.4	1.10		4.27	0.43		99.9	2.76		2.35	0.33	
Male (without cardiac failure)	Pre (Day 1)	139.6	0.92	0.001	3.68	0.39	0.001	98.2	1.02	0.010	2.06	0.16	0.000
	Post (Day 5)	139.3	0.67		4.08	0.44		99.0	1.03		2.53	0.21	
Female (cardiac failure)	Pre (Day 1)	137.67	2.66	0.010	3.90	0.88	0.486	99.82	3.24	0.037	1.82	0.23	0.000
	Post (Day 5)	140.9	0.98		4.02	0.60		97.04	3.26		2.23	0.33	

Female (without cardiac failure)	Pre (Day 1)	139.5	1.31	0.011	3.75	0.18	0.000	98.09	3.42	0.227	2.04	0.13	0.000
	Post (Day 5)	141.67	1.05		4.29	0.29		99.08	2.64		2.55	0.23	

Table 3. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Cardiac Failure in Both Genders

Paired t-test, p-value for K, CL show no significant difference between pre and post values of K, CL of day 1 and day 5 in males with cardiac failure.

K has no significant difference between pre and post values of K on day 1 and day 5 in females with cardiac failure.

CL has no significant difference between pre and post values of CL on day 1 and day 5 in females without cardiac failure.

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (with cardiogenic shock)	Pre (Day 1)	134.56	2.67	0.000	4.07	0.83	0.471	99.6	3.19	0.001	1.94	0.35	0.000
	Post (Day 5)	140.7	1.17		4.22	0.42		96.9	2.17		2.31	0.46	
Male (without cardiogenic shock)	Pre (Day 1)	139.9	2.48	0.529	3.57	0.45	0.000	98.07	1.14	0.018	1.95	0.11	0.000
	Post (Day 5)	140.7	0.96		4.12	0.39		98.95	1.02		2.45	0.19	
Female (with cardiogenic shock)	Pre (Day 1)	135.57	3.16	0.038	3.85	0.84	0.400	96.09	2.74	0.291	1.02	2.74	0.291
	Post (Day 5)	139.09	0.40		4.11	0.48		97.09	1.90		1.03	1.90	
Female (without cardiogenic shock)	Pre (Day 1)	140.5	0.28	0.035	3.68	0.18	0.027	99.7	4.28	0.614	1.99	0.21	0.000
	Post (Day 5)	141.45	0.80		4.12	0.43		98.09	2.67		2.47	0.27	

Table 4. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Cardiogenic Shock in Both Genders

P value for K show significant difference between pre and post values of K of day 1 and day 5 in males with cardiogenic shock.

NA show no significant difference between pre and post values of Na of day 1 and day 5 in males without cardiogenic shock.

K, Cl, Mg are not significant between pre and post values of K, Cl, Mg on day 1 and day 5 in females with cardiogenic shock.

Cl show no significant difference between pre and post values of Cl on day 1 and day 5 in females without cardiogenic shock.

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (with arrhythmia)	Pre (Day 1)	136.09	3.80	0.005	3.79	0.97	0.236	97.09	4.51	0.337	1.83	0.24	0.000
	Post (Day 5)	140.4	1.22		4.04	0.37		98.03	2.28		2.15	0.27	
Male (without arrhythmia)	Pre (Day 1)	136.09	3.80	0.005	3.79	0.97	0.236	97.09	4.51	0.337	1.83	0.24	0.000
	Post (Day 5)	140.4	1.22		4.04	0.37		98.03	2.28		2.15	0.27	
Female (with arrhythmia)	Pre (Day 1)	137.6	2.91	0.095	3.79	0.79	0.800	98.74	4.07	0.274	1.75	0.11	0.002
	Post (Day 5)	139.6	0.91		3.85	0.59		99.42	3.35		2.04	0.05	
Female (without arrhythmia)	Pre (Day 1)	139.56	1.44	0.034	3.85	0.31	0.004	98.09	4.68	0.539	2.06	0.16	0.000
	Post (Day 5)	140.22	1.26		4.35	0.26		99.09	2.96		2.55	0.18	

Table 5. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Arrhythmia in Both Genders

Na and Mg are significantly difference between pre and post values on day 1 and day 5 in males without arrhythmia.

K, Mg are showing significant difference between pre and post values on day 1 and day 5 in males with arrhythmia.

Mg show significant difference between pre and post values day 1 and day 5 in females with arrhythmia.

Na, K and Mg are showing significant difference between pre and post values on day 1 and day 5 in females without arrhythmia.

DISCUSSION

In the present study, the serum magnesium, sodium and potassium levels were significantly lower in the arrhythmia patients, which was similar to those, which were seen in other studies.[4] Fall in serum electrolyte levels on day of admission is similar to finding was observed by Shah et al[5] who reported hyponatraemia on day first. Flear and Hilton reported a progressive fall in the mean daily serum sodium concentration until day 4 and rise thereafter in all cases.[6] Significant decrease in serum sodium concentration in both sexes was also reported by Flear and Singh,[7] which is similar to our observation.

In acute myocardial infarction, nonosmotic release of vasopressin may occur due to the acute development of left ventricular dysfunction in response to pain, nausea and major stress. The most common mechanisms of hyponatraemia in adults or in response to the administration of analgesics and diuretics.[8]

In this setting, vasopressin level increases concomitantly with the activation of other neurohormones such as renin and norepinephrine. Moreover, the renal effect of vasopressin is enhanced in heart failure as the vasopressin-regulated water in the collecting duct is up regulated. Mean serum sodium level at baseline in our study of males (n=70) was 137.88 ± 2.73 and of females (n=30) was 138.81 ± 2.59. The mean serum sodium level in cardiac failure of males (n=15) was 135.50 ± 4.48 and of females (n=10) 137.67 ± 2.66 at baselines. While mean serum sodium level in cardiogenic shock of males (n=10) was 134.56 ± 2.67 and of females (n=5) 135.57 ± 3.16 at baselines. Mean serum sodium level in study by Esha Mati[9] et al (n=50) was 135 ± 5.17, while in Vinod Wali et al[10] (n=36), it was 129.47 ± 4.87. There is not much significant differences observed in serum chloride levels in various groups. Serum potassium concentration was decreased significantly in patients with arrhythmias in our study. In

present study, the mean of baseline of K was 3.77 ± 0.55 in males and 3.85 ± 4.18 in females. The mean of K in males at baseline was 3.79 ± 0.97 with arrhythmia and without arrhythmia K 3.94 ± 0.37. The mean of K in female at baseline was with arrhythmia 3.79 ± 0.79 and without arrhythmia was 3.85 ± 0.31. Solmon et al and Hulting J et al also observed hypokalaemia[11] in patients of acute myocardial infarction in their studies. In present study, ventricular premature beats were present more commonly in hypokalaemic group similar to T. Dyckner et al and Erik J. observed higher incidence of ventricular premature beats in hypokalaemic group as compared to normokalaemic and hyperkalaemic group.[12] Highest incidence of ventricular tachycardia was observed in hypokalaemic group by Dyckner and Erik J. and Solmon et al. In our study also, ventricular tachycardia was present in significant number of patients with hypokalaemia. In present study, ventricular fibrillation was present only in hypokalaemic group. Similarly, Fredensohn A., Duke M. and others observed higher incidence of ventricular fibrillation in hypokalaemic group.[13] Serum magnesium concentration was decreased significantly in patients with arrhythmias in our study.

Many studies have been reported regarding the relation of electrolytes and cardiac arrhythmias. Charles Fisch et al,[14] while a number of electrolytes play a role in the genesis of the transmembrane Action Potential (AP), the changes in the action potential most clearly related to arrhythmias are dependent to a large extent on K+. Potassium gradient is a major determinant of the magnitude of Transmembrane Resting Potential (TRP) and secondarily the rate of rise (dV/dt) of phase 0 and consequently the speed of conduction. The cell membrane conductance for K+ or a decrease therein is most likely the major determinant of spontaneous low depolarisation during phase 4. Thus, K+ has a pronounced effect on both conduction and automaticity. Furthermore, these electrophysiological properties are altered within levels of K+ encountered in clinical medicine, a situation which, with rare exceptions is not seen with Ca+, Mg+ or Na. These latter ions affect the action potential and induce experimental arrhythmias at concentrations, which are unphysiologic and frequently incompatible with life. Consequently, of all the electrolytes, disturbed K+ metabolism accounts for the vast majority of clinical arrhythmias. For the same reasons with the exception of the ability of Na+ and Ca++ to reverse the K+-induced depression of conduction, K+ is the only electrolyte with clinically significant antiarrhythmic properties.

Nabil El Sherif et al[15] electrolyte disorders can alter cardiac ionic currents kinetics and depending on the changes can promote proarrhythmic or antiarrhythmic effects. The present report reviews the mechanisms, Electrophysiological (EP), Electrocardiographic (ECG) and clinical consequences of electrolyte disorders. Potassium (K^+) is the most abundant intracellular cation and hypokalaemia is the most common electrolyte abnormality encountered in clinical practice. The most significant ECG manifestation of hypokalaemia is a prominent U wave. Several cardiac and noncardiac drugs are known to suppress the hERG K^+ channel and hence the I and especially in the presence of hypokalaemia can result in prolonged action potential duration and QT interval, QTU alternans, early after depolarisations and Torsade de pointes Ventricular Tachyarrhythmia (TdPVT). Hyperkalaemia affects up to 8% of hospitalised patients mainly in the setting of compromised renal function. The ECG manifestation of hyperkalaemia depends on serum K^+ level. At 5.5-7.0 mmol/L K^+, tall peaked, narrow-based T waves are seen. At >10.0 mmol/L K^+, sinus arrest, marked intraventricular conduction delay, ventricular tachycardia and ventricular fibrillation can develop. Isolated abnormalities of extracellular calcium (Ca^{++}) produce clinically significant EP effects only when they are extreme in either direction. Hypocalcaemia frequently seen in the setting of chronic renal insufficiency results in prolonged ST segment and QT interval, while hypercalcaemia usually seen with hyperparathyroidism results in shortening of both intervals. Although, magnesium is the second most abundant intracellular cation, the significance of magnesium disorders are controversial partly because of the frequent association of other electrolyte abnormalities. However, IV magnesium by blocking the L-type Ca^{++} current can successfully terminate TdPVT without affecting the prolonged QT interval. Finally, despite the frequency of sodium abnormalities, particularly hyponatraemia, its EP effects are rarely clinically significant.

Brown MJ et al[16] conducted a study to determine whether epinephrine-induced hypokalaemia is due to beta 2 adrenoceptor stimulation and whether hypokalaemia can occur at physiologic concentrations of the agonist, epinephrine was infused into six normal volunteers at a rate of 0.1 micrograms/kg of body weight per minute. The circulating epinephrine concentration was increased to 1.74 ± 0.65 ng/mL, plasma potassium was reduced by 0.82 ± 0.19 mEq per litre, plasma insulin fell by 12 ± 4 mU per litre, plasma renin activity was elevated and tachycardia occurred. Isoproterenol infused at 0.02 micrograms per kilogram per minute caused similar tachycardia (25 beats/minute) and elevation in plasma renin activity (6.0 to 6.5 ng per millilitre per hour), but no hypokalaemia. The difference in responses to the two catecholamines was ascribed to the relative beta 2 selectivity of epinephrine. This hypothesis was tested in six subjects given infusions of epinephrine (0.05 micrograms per kg per minute) after administration of either 2.5 to 5 mg of ICI 118551 - a selective beta 2 receptor antagonist or placebo. After placebo, epinephrine infusion elevated the circulating epinephrine concentration and reduced plasma potassium, hypokalaemia was prevented by the beta 2 antagonist. This drug only partially inhibited the rises in the plasma renin and glucose and the shortening of systolic time intervals; there was no tachycardia. 15 fold to 30 fold increases in circulating epinephrine concentration appear to cause hypokalaemia by a specific beta 2 receptor effect distinct from others actions of epinephrine. This phenomenon maybe physiologic importance after severe myocardial infarction when similar increases in plasma epinephrine have occurred.

Eva Z Hesselkilde et al[17] suggest that arrhythmias occurring secondary to systemic disease are seen more commonly in the clinic than arrhythmias caused by cardiac disease. No significant difference between the two groups was found for AV blocks, SVPCs and VPCs (P = 0.08-0.76). The mean levels of potassium, sodium, ionized calcium and chloride were significantly lower in the colic group compared to the control group at admission. Although, we only observed VPCs in the colic horses, no significant differences between colic horses and controls were found. Despite the colic horses having electrolyte changes at admission, no correlation was found between the electrolyte disturbances and cardiac arrhythmias. Although, no clear conclusions can be drawn from the present study, the results indicate that relatively mild colic per se is not proarrhythmogenic, whereas severe colic probably are more likely to result in ventricular arrhythmia.

Sumedh S Hoskote et al[18] disorders of potassium homeostasis are common electrolyte abnormalities encountered in hospitalised patients. Hypokalaemia and hyperkalaemia have been estimated to occur in about 21% and 3% of hospitalised patients, respectively; though the morbidity and mortality associated with the latter is significantly higher. Potassium is a predominantly intracellular ion and the understanding of its dynamics between intra- and extracellular fluid milieus along with its handling by the kidneys is important in the diagnosis and treatment of potassium disorders. This article aims to provide a clinically relevant update on management of potassium disorders for internists.

CONCLUSION

In the present study, serum magnesium, sodium and potassium levels were significantly lower in the arrhythmia patients, which was similar to those, which were seen in other studies. In acute myocardial infarction, no osmotic release of vasopressin may occur due to the acute development of left ventricular dysfunction in response to pain, nausea and major stress, the most common mechanisms of hyponatraemia in adults or in response to the administration of analgesics and diuretics. In this setting, vasopressin level increases concomitantly with the activation of other neurohormones such as renin and norepinephrine.

REFERENCES

[1] Sakmann B, Trube G. Conductance properties of single inwardly rectifying potassium channels in ventricular cells from guinea pig heart. J Physiol 1984;347(1):641-657.

[2] Pelzer D, Trautwein W. Currents through ionic channels in multicellular cardiac tissue and single heart cells. Experientia 1987;43(11-12):1153-1162.

[3] Paice BJ, Paterson KR, Onyanga-Omara F, et al. Record linkage study of hypokalaemia in hospitalized patients. Postgrad Med J 1986;62(725):187-191.

[4] Solini A, Zamboni P, Passaro A, et al. Acute vascular events and electrolytes variations in elderly patients. Horm Metab Res 2006;38(3):197-202.

[5] Shah PKD, Saxena M, Khangarot D, et al. Study of serum sodium in myocardial infarction and its relation to severity and complications. J Assoc Physicians India 1986;34(3):195-196.

[6] Flear CTG, Hilton P. Hyponatremia and severity and outcome of myocardial infarction. Br Med J 1979;1(6173):1242-1246.

[7] Flear CT, Singh CM. Hyponatraemia and sick cells. Br J Anaesth 1973;45(9):976-994.

[8] Rowe JW, Shelton RL, Helderman JH. Influence of the emetic reflex on vasopressin release in man. Kidney Int 1979;16(6):729-735.

[9] Mati E, Krisnamurthy N, Ashakiran S, et al. Dyselectrolytemia in acute myocardial infarction-a retrospective study. J Clin Biomed Sci 2012;2(4):167-174.

[10] Wali V, Singi Y. Study of serum sodium and potassium in acute myocardial infarction. Journal of Clinical and Diagnostic Research 2014;8(11):7-9.

[11] Solomon RJ, Cole AG. Importance of potassium in patients with acute myocardial infarction. Acta Med Scand 1981;647:87-93.

[12] Nordrehaug EJ. Malignant arrhythmias in relation to serum potassium values in patients with an acute myocardial infarction. Act Med Scand 1981;647:101-107.

[13] Fredensohn A, Faibel HE, Bairey O, et al. Malignant arrhythmia in relation to values of serum potassium in patients acute myocardial infarction. Int J Cardiol 1991;32(3):331-338.

[14] Fisch C. Relation of electrolyte disturbances to cardiac arrhythmias. Circulation 1973;47:408-419.

[15] El Sherif N, Turitto G. Electrolytes disorders and arrhythmogenesis. Cardiol J 2011;18(3):233-245.

[16] Brown MJ, Brown DC, Murphy MB. Hypokalemia from beta 2-receptor stimulation by circulating epinephrine. N Engl J Med 1983;309:1414-1419.

[17] Hesselkilde EZ, Almind ME, Petersen J, et al. Cardiac arrhythmias and electrolyte disturbances in colic horses. Acta Vet Scand 2014:56(1):58.

[18] Hoskote SS, Joshi SR, Ghosh AK. Disorders of potassium homeostasis: pathophysiology and management. JAPI 2008;56:685-693.