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Original Research Article.

Measurement of Serum Levels of Sodium and Potassium among Patients with Sickle Cell Anemia in Wed Madani Gezira State, Sudan January (2017)

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ABSTRACT

Objective: The study aimed to estimate serum levels of sodium and potassium in patients with sickle cell anemia. This analytical study was conducted in Wad Medani pediatric teaching hospital, Gezira State, during July to October 2016.

Material and Methods: The study involved 60 patients with sickle cell anemia, both sex in age range of 6-17 years. 2.5 ml of blood samples was collected into lithium heparin containers and separated by centrifugation, and then sodium and potassium levels were estimated by ion selective electrode. Data were analyzed statistically by SPSS program.

Results: The results showed that the serum level of potassium was significantly high in crisis state patients when compared with steady state patients (6.347±0.2809, 4.285 ±0.7649) respectively. And Serum level of sodium was significantly low in crisis state patients when compared with steady state patients (M± SD= 130.65. ±4.898, 137.84 ±4.530) respectively. There was no significantly difference in serum levels of sodium and potassium between gender groups (*P* 0.174, *P* 0.355) respectively. There was no significantly difference in serum levels of sodium and potassium between age groups (*P* 0.378, P 0.560) respectively.

Conclusion: The study recommended that the potassium level should be evaluate in sickle patients to reduce complication, more studies to evaluate the role of blood transfusion and correlation of potassium serum level.

Key words: Sickle Cell, Sodium, Potassium.

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INTRODUCTION

Sickle cell anemia is inherited hemolytic anemia due to defective hemoglobin molecule in red blood cells which delivers oxygen to cells throughout the body .It is inherited in an autosomal recessive pattern, due to point mutation (substitution) that alter thymine to adenine in beta globin chain result in replacement of glutamate to valine. This defect result in new abnormal hemoglobin known as sickle cell hemoglobin (HbS), which can distort red blood cells a sickle or crescent shape in state of flexible round shape.[1]

Red cells acquire the sickle or elongated shape upon deoxygenation as a result of intracellular polymerization of HbS, a phenomenon that is reversible on reoxygenation. Even in the normally shaped red cells, however, the presence of HbS polymer reduces deformability, with consequent increase in blood viscosity. Repeated or prolonged sickling progressively damages the red cell membrane, which is a phenomenon of primary importance in the pathophysiology of SCA. Membrane damage causes movement of potassium ions and water out of the cell by the Gardos pathway and potassium chloride cotransport, leading to dehydration of red cells. It is worldwide distribution but more common in Africa. It responsible for high rate of mortality and morbidity.[2] Sodium the major cation in extracellular fluid, such as plasma water, sodium plays a major role in maintaining osmotic pressure. Sodium levels in body fluids are maintained by renal reabsorption in the proximal convoluted tubules, based on a sodium threshold. Sodium balance is also maintained by the influence of the hormone aldosterone on the distal convoluted tubules, as a consequence of changes in blood volume and blood pressure. Extremely high or low sodium concentrations in plasma

will cause severe osmotic pressure changes that can induce serious consequences to several organs. The most immediate effect swelling on the brain and potential coma.[3] Potassium is the major intracellular cation is also controlled by the Na-K-ATPase pump. Potassium maintains cardiac rhythm and contributes to neuromuscular conduction. Imbalances in potassium level, as indicated by hyperkalemia or hypokalemia, will cause cardiac arrhythmias and neuromuscular weakness.[3]

LITERATURE REVIEW

Sickle Cell Anemia (SCA): Definition

Inherited hemolytic anemia due to defective in hemoglobin molecule in red blood cells that delivers oxygen to cells throughout the body .It is inherited in an autosomal recessive pattern, due to point mutation (substitution) that alter thymine to adenine in beta globin chain result in replacement of glutamate to valine. This defect result in new abnormal hemoglobin known as sickle cell hemoglobin (HbS), which can distort red blood cells a sickle or crescent shape in state of flexible round shape.[1]

Epidemiology of SCA

It is seen worldwide but occurs most frequently in Africans and less commonly in those of Mediterranean, Latino, East Indian, and Arab descent. It is estimated that 16% of the population in Africa has a sickle hemoglobinopathy which is the highest proportion worldwide. The Americas and the East Mediterranean region represent the next highest proportion of sickle cell hemoglobinopathy as delineated by the World Health Organization.[4]

Pathophysiology

The Pathophysiologic processes that result in the clinical phenotype extend beyond the red blood cell. There is marked clinical heterogeneity from one patient to another and in the same patient overtime. The heterogeneity for the same genotypic abnormality therefore implies that a multitude of other factors must contribute to the pathology of sickle cell anemia.[2]

THE PATHOLOGIC PROCESSES OF SICKLE CELL ANEMIA ARE

1. Molecular Basis of Sickling

Deoxygenation of HbS leads to a conformational change that exposes a hydrophobic patch on the surface of the β s – globin chain at the site of β 6 valine. Binding of this site to a complementary hydrophobic site on a β - subunit of another haemoglobin tetramer triggers the formation of large polymers.

The polymerization of HbS in the circulating red cells is influenced by the oxygenation status, the intracellular haemoglobin concentration and the presence of non - sickle haemoglobins. Acidosis and elevated level of 2,3 – diphosphoglycerate (2,3 - DPG) promote polymer formation by reducing the oxygen affinity of haemoglobin. The presence of HbA within the red cells, as in sickle trait, inhibits polymerization by diluting HbS.2

2. Effect on Erythrocytes

Red cells acquire the sickle or elongated shape upon deoxygenation as a result of intracellular polymerization of HbS, a phenomenon that is reversible on reoxygenation. Even in the normally shaped red cells, however, the presence of HbS polymer reduces deformability, with consequent increase in blood viscosity. Repeated or prolonged sickling progressively damages the red cell membrane, which is a phenomenon of primary importance in the pathophysiology of SCA. Membrane damage causes movement of potassium ions and water out of the cell by the Gardos pathway and potassium – chloride cotransport, leading to dehydration of red cells. A second key consequence of membrane damage is alteration of the chemistry of the red cell membrane. Perturbation of lipid organization causes negatively charged phosphatidylserine to appear on the red cell surface instead of its normal location in the inner monolayer. In addition, the red cells become abnormally adherent to the vascular endothelium through vascular cell adhesion molecule (VCAM) - 1, thrombospondin and fibronectin.[2]

3. Vaso - occlusion

Several processes contribute to development of vaso – occlusion in SCA. Slowing of blood flow arises from abnormal regulation of vascular tone as a result of diminished nitric oxide (NO) induced vasodilatation. This is aggravated by increase in blood viscosity, resulting from less deformable red cells. Vaso - occlusion is initiated by adhesion of young deformable red cells to the vascular endothelium, and is promoted by leucocytosis, platelet activation and inflamatory cytokines.[2]

4. Haemolysis

SCA is characterized by chronic intravascular and extravascular haemolysis. Sickling- induced membrane fragmentation and complement - mediated lysis cause intravascular destruction of red cells. Membrane damage also leads to extravascular haemolysis through entrapment of poorly deformable cells or uptake by macrophages.

Patients have greatly expanded bone marrow space, but the serum erthropoietin level is lower than expected for the extent of anaemia because of the decreased oxygen affinity of HbS.[2]

CLINICAL COMPLICATIONS

1. Hemolytic Crisis

This due to an increased rate of hemolysis with a fall in hemoglobin but rise in reticulocytes, patients present as a case of as of acute hemolytic anemia usually accompany a painful crisis.

2. A plastic Crisis

This due to infection with parvovirus or form of folate deficiency characterized by sudden deceased in Hb and deceased in reticulocytes, mostly self-limiting and blood transfusion may be needed.

3. Vaso-occlusive Crises

Most frequent type of crisis, caused by sickled of red cell in blood capillaries and restrict blood flow to an organ resulting in ischemia, pain, necrosis and often organ damage, precipitated by infections, cold, exercise, dehydration, acidosis, deoxygenation.[5]

4. Splenic Sequestration Crisis

Acute pain full enlargement of the spleen, caused by intrasplenic trapping of red cells and resulting in precipitous fall in hemoglobin levels with the potential for hypovolemic shock. Spleen damage increases risk of infection from encapsulated organisms sequestration crisis are considered an emergency. If not treated, patients may die within 1-2 hours due to circulatory failure.[6]

5. Infection

Children with sickle cell disease are at increased risk for bacteremia that can result in sepsis and death; due in large part to functional a splenia that develops over time in these children. The most common organisms involved include *Streptococcus pneumoniae*, Salmonella species, and *Haemophilus influenza*.[2]

6. Leg Ulcers

Chronic leg ulcers are frequent in adult patients with SCA, particularly affecting males with the HbSS genotype. Ulcers arise near the medial or lateral malleolus and may be single or multiple. Occlusion of skin microvasculature from sickle red cells predisposes to ulcers, which are made worse by trauma, infection or warm climate. Ulcers are always colonized with pathogenic bacteria (Pseudomonas aeruginosa, S. aureus and Streptococcus spp) and acute infection can occur. The ulcers are painful and resistant to healing, and although bed rest and elevation of the leg are efficacious.[2]

7. Renal Defect

The hypoxic, acidotic and hypertonic renal medulla favours vaso - occlusion, leading to destruction of the vasa recta and hyposthenuria in the first year of life. It presents clinically as enuresis or nocturia, and patients are susceptible to dehydration in hot weather. Haematuria as a result of papillary necrosis usually originates from the left kidney. Management is generally by bed rest and hydration, although sometimes blood transfusion and ε - aminocaproic acid are required. Proteinuria due to glomerular injury precedes development of nephrotic syndrome and eventual chronic renal insufficiency in one - quarter of adults. The progression to renal failure can be delayed by angiotensin - converting enzyme inhibitors. Careful control of blood pressure, avoidance of non - steroidal anti- inflammatory drugs (NSAIDs) and aggressive treatment of urinary tract infection and anaemia are important for patients with chronic renal insufficient.[2]

8. Bone pain

In infants mostly affecting fingers and toes causing painful swelling (hand and foot syndrome).

9. CNS

Stroke may occur due to sinus thrombosis.

10. Chest pain

May occur due to bone involvement or pulmonary infection .precipitating factors include dehydration, chilling, and infection. Pain is excruciatingly severe fever, increasing jaundice and malaise are frequent.

11. Penile involvement

Vaso-occlusion may lead to priapism.

DIAGNOSIS OF SICKLE CELL ANEMIA

I. Sickling of the red cells, on a blood film, can be induced by the addition of sodium metabisulfite.
II. Sickle solubility test, a mixture of HbS in reducing solution (sodium and dithionate) gives a turbid appearance, whereas normal Hb gives a clear solution.
III. Electrophoresis, to detect abnormal forms of Hb.[7]

THE ELECTROLYTE

1. SODIUM

Sodium is the most abundant cation in extracellular fluid about 90.1% of all ECF and largely determines the osmolality of the plasma. Normal plasma osmolality is approximately 295 mmol/L, with 270 mmol/L as the result of sodium and associated anions. The two sides (ICF and ECF) would eventually reach equilibrium. To prevent equilibrium from occurring, active transport system, such as ATPase ion pumps, are present in all cells. Potassium the major ICF, pottasium would eventually diffuse across the cell membrane until equilibrium is reached. The sodium and potassium ATPase ion pump moves three sodium ions out of the cell in exchange for two K^+ ions moving into the cell as ATP is converted to ADP .Because water follows electrolytes across cell membrane, the continual removal of sodium$^+$ from the cell prevent osmotic rupture of the cell by drawing water from the cell.[8]

Regulation

The plasma sodium concentration depends greatly on the intake and excretion of water and lesser degree, the renal regulation of sodium.

The primary important processes of regulation are

1. The intake of water in response to thirst.
2. The excretion of water.
3. The blood volume status.

The kidneys have the ability to excrete large amount of Na^+, depending on the Na^+ content of the ECF and blood volume .Normally 60 % to 75% of filtrated Na^+ is reabsorbed in proximal tubule; electroneutrality is maintained by either Cl^- reabsorption or hydrogen ion (H^+) secretion. Some $Na^+$is also reabsorbed in the loop and distal tubule and exchange for K^+ in connecting segment and cortical collecting tubule.[9]

Metabolic Abnormalities

Hyponatremia

Hyponatremia occur when serum or plasma less than 135mmol/L, is one of the most common electrolyte disorders in patients levels below 130 mmol/L are clinically significant.

Hyponatremia is caused by renal and nonrenal causes also. Salt-losing renal nephritis, renal tubular acidosis, or syndrome of inappropriate antidiuretic hormone secretion (SIADH) are common causes of renal loss of sodium and may be evaluated by testing for the presence of excess urinary sodium and hyperosmolar urine. Certain diuretics, such as thiazine, can cause renal loss of sodium. Increased urine sodium levels usually indicate sodium loss. Chronic renal failure can cause water overload due to inability to regulate water and results in hyponatremia, while nephritic syndrome can cause fluid imbalances and edema with resulting hyponatremia. Urine sodium levels are usually normal or decreased in hyponatremia due to edema. Non renal causes of hyponatremia include psychogenic water overload, cellular shift changes from acidosis, and edema secondary to cirrhosis or congestive heart failure.[3]

Symptoms of Hyponatremia

Symptoms depend on the serum level, between 125and 130 mmol/L symptoms include primarily gastrointestinal, more severe symptoms are seen below 125 mmol/L including nausea, vomiting, muscular weakness, headache. More severe symptoms also include seizures, coma, and respiratory depression .a level below 120 mmol/L for 48 hours or less considered a medical emergency.[3]

Hypernatremia

Hypernatremia is caused by renal and non-renal disorder. A common non renal cause is hypotonic dehydration from severe diarrhea, extensive burns, or excessive sweating without proper fluid replacement. Infants, the elderly, and other patients not able to ingest sufficient amounts of water, and who are not properly hydrated, will also experience hypernatremia. Renal loss of water, such as in nephrogenic diabetes insipidus, Serum osmolality and urinary sodium levels can help to differentiate renal loss of water versus non renal causes.[3]

Symptoms of Hypernatremia

Symptoms most commonly involve the CNS as the result of hyperosmolar state .These symptoms include altered mental status, irritability, restlessness, fever, nausea, and increased thirst.[3]

2. POTASSIUM

Potassium is the major ICF cation in the body, with a concentration 20 times greater inside the cells than outside. Many cellular functions require that the body maintain a low ECF concentration of K^+ ions. Only 2% of the body total K^+ circulates in the plasma. Functions of potassium in the body include regulation of neuromuscular excitability, contraction of the heart, ICF volume and H^+ concentration.

Regulation

The kidney have important role in regulation of potassium .initially ,the proximal tubules reabsorb nearly all the potassium .Then additional k^+ is secreted in urine in exchange of Na^+ under the influence of aldosterone in both the distal tubules and collecting ducts.

I. In an acute rise in plasma K^+, excess plasma K^+ rapidly enters the cells to normalize plasma K^+, and a cellular K^+ gradually returns to the plasma, it is removed by urinary excretion. Three factors that influence the distribution of K^+ between cells and ECF are: K^+ loss frequently occurs whenever the Na^+ and K^+ ATPase pump is inhibited by conditions such as hypoxia, hypomagnesemia, or digoxin overdose.
II. Insulin promotes acute entry of K^+ into skeletal muscle and liver by increasing Na^+ K^+ ATPase activity.
III. Cathecholamines, such as epinephrine which promote cellular entry whereas propranol impairs cellular entry.[9]

Metabolic Abnormalities

Hyperkalemia

Hyperkalemia may be caused by decreased renal excretion in acute or chronic renal failure, certain diuretics, or hypoaldosteronism or hypocortisolism. Hyperkalemia may also be caused by ion shift, such as the ion shift that is seen in cases of diabetic ketoacidosis or other mestabolic acidoses, leukemia, excessive muscle activity, or hemolysis. Finally, hyperkalemia is associated with iatrogenic causes of excessive intravenous or oral therapy.[3]

Symptoms of Hyperkalemia

Muscle weakness, tingling, numbness, disturbs cardiac conduction which can lead to cardiac arrhymias and cardiac arrest.[10]

Hypokalemia

Is the plasma K^+ concentration below the lower limit of the reference range. Hypokalemia is caused by renal loss such as renal tubular acidosis, hyperaldosteronism, hypercortisolism, and certain diuretics. Potassium can also be decreased due to gastrointestinal dietary deficit or loss from severe vomiting, diarrhea, nasogastric suctioning, laxatives, and malabsorption. A cellular shift in cases of insulin overdose and alkalosis can also cause hypokalemia.[3]

Symptoms of Hypokalemia

Weakness, Fatigue, Constipation. In cardiovascular disorder patients in which increased risk of arrhythmia which lead to sudden death in certain patients.[10]

Previous study

A study done by Agoreyo and Nwanze (2009) in Nigeria. They were measured the body electrolytes (in plasma) such as sodium (Na+) and potassium (k+) in adult sickle cell patients compared with control group. The study involved a total of 38 individuals, both males and females in the age range of 5 - 20 years.

There were 3 study groups; steady state group, crisis state group and control group. Flame photometry was used to analyze sodium and potassium. In the males there was statistically significant reduction in the concentration of sodium in both the steady state (129.40 ± 1.462) and crisis state (121.60 ± 0.678) when compared with control group (134.40 ± 2.040). Also, there was a statistically significant increase ($P < 0.05$) in the concentration of potassium in both the steady state(4.58 ± 0.171 mmol/l) and crisis state (4.66 ± 0.154 mmol/l) when compared with normal group (3.50 ±0.172 mmol/l).

JUSTIFICATION

Sickle cell anemia responsible for high rate of morbidity and mortality and due to series complication such as heart failure and renal defect. Associated with multiple risk factors as abnormal sodium and potassium required additional precaution and investigation for follow up of patients.

OBJECTIVES

General Objective

To detect the effect of sickle cell anemia on electrolytes (sodium and potassium).

Specific Objectives

1. To measure serum level of sodium in sickle cell patients in steady state and crisis state.
2. To measure serum level of potassium in sickle cell patients in steady state and crisis state.
3. To find out possible correlation between patients age, gender and serum level of sodium.
4. To find out possible correlation between patients age, gender and serum level of potassium.

MATERIALS AND METHODS

Study Design & Area

A Cross sectional laboratory based study was conducted in Gezira state, in Wad Medani pediatric teaching hospital.

Study Population

Patients with sickle cell anemia in age range of 6-17 years.

Study Period

July to October 2016

linclusion Criteria

Patient with Sickle cell anemia.

Exclusion Criteria

Renal disease patients
Diabetes mellitus patients
Blood transfusion patients
Heart disease patients

Sample Size

Sixty sample from patients with sickle cell anemia.

Ethical Consideration

- The objectives of this study were explained to all individual participating in this study.
- An informed consent was obtained from all participants.

Data Collection and Analysis

Data was collected by using a questionnaire. Questionnaires were then filled by the investigator during each time when blood samples collected. Data was then analyzed and tabulated using statistical package for social sciences (IBM SPSS) program version 20, T test, a crosstabs and correlation were performed.

Materials and Equipment

- Roche diagnostic (ISE indirect Na, K, Cl)
- Centrifuge and Automatic pipette.
- Syringes, gloves, alcohol (70% ethanol) and lithium heparin containers.

Blood sampling and Collection

Two ml of venous blood were collected from each participants by use a sterile syringe into a labeled lithium heparin container. Blood samples were then centrifuged to obtain the serum. All serum was kept at −20°C until using for measurement of sodium and potassium levels.

METHOD

Test Principle

An Ion-Selective Electrode (ISE) makes use of the unique properties of certain membrane materials to develop an electrical potential (electromotive force, EMF) for the measurements of ions in solution. The electrode has a selective membrane in contact with both the test solution and an internal filling solution. The internal filling solution contains the test ion at a fixed concentration. Because of the particular nature of the membrane, the test ions will closely associate with the membrane on each side. The membrane EMF is determined by the difference in concentration of the test solution and the internal filling solution. The EMF develops according to the Nernst equation for a specific ion in solution:

(1) $$E = Eo + \frac{RT}{nF} \times \ln \frac{(f \times Ct)}{(f \times Ci)}$$

Where:

E = Electrode EMF

Eo = Standard EMF

R = Constant

T = Temperature

n = Charge of the Ion

F = Faraday's Constant

In = Natural Logarithm (base e)

F = Activity Coefficient

Ct = Ion Concentration in Test Solution

Ci = Ion Concentration in Internal Filling Solution

Reference Range

Na=135-145mmol/l

K=3.5-5mmol/l

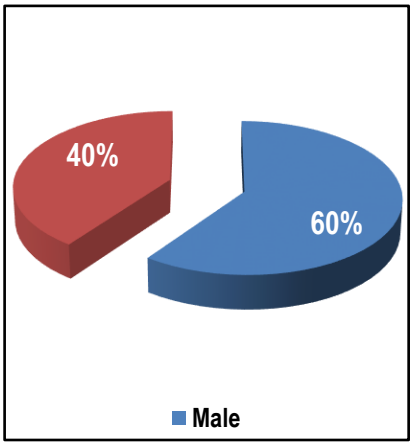

Figure 1: Distribution of study population according to gender

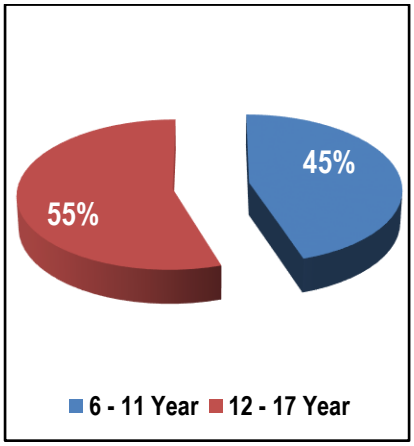

Figure 2: Distribution of study population according to Age

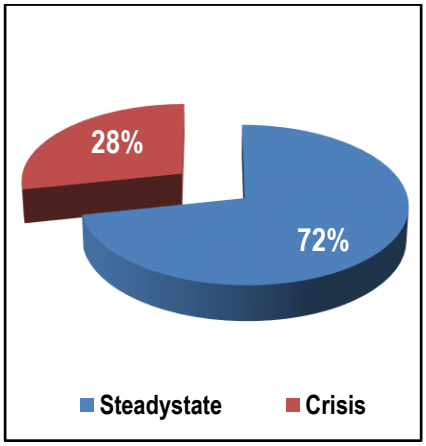

Figure 3: Distribution of study population according to clinical states

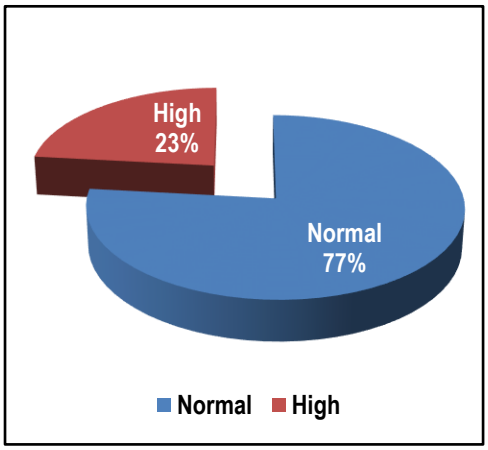

Figure 4: Distribution of serum level of potassium according to reference range.

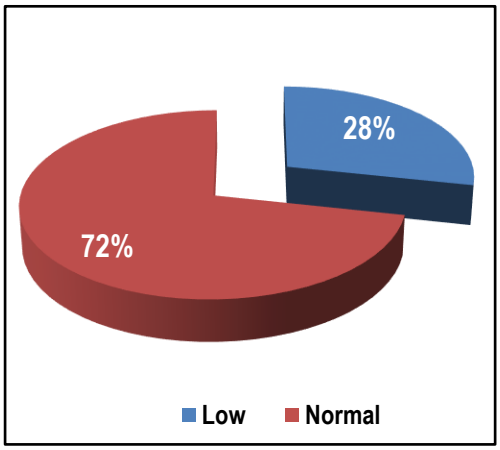

Figure 5: Distribution of serum level of sodium according to reference range

Table 1: The mean of serum level of potassium in crisis patients and in steady state patients.

	State	No	Mean	Std. Deviation	P. Value
K^+	Steady state	43	4.286	.7649	0.000
	Crisis	17	6.347	.2809	

Table 2: The mean of serum level of sodium in crisis patients and steady state patients.

	States	No	Mean	Std. Deviation	P. Value
Na^+	Steady state	43	137.84	4.530	0.000
	Crisis	17	130.65	4.898	

Table 3: The mean of serum level of potassium in patients according to gender

	Gender	No	Mean	Std. Deviation	P. Value
K^+	Male	36	4.981	1.1923	0.355
	Female	24	4.704	1.0780	

Table 4: The mean of serum level of sodium in patients according to gender

	Gender	No	Mean	Std. Deviation	P. Value
Na^+	Male	36	135.03	6.115	0.174
	Female	24	136.96	4.723	

Table 5: The mean of serum level of potassium in patients according to age

	Age group	No	Mean	Std. Deviation	P. Value
K^+	6 - 11 Year	27	4.774	1.1220	0.560
	12 - 17 Year	33	4.948	1.1780	

Table 6: The mean of serum level of sodium in patients according to age

	Age group	No	Mean	Std. Deviation	P. Value
Na^+	6 - 11 Year	27	136.52	5.713	0.378
	12 - 17 Year	33	135.21	5.594	

RESULTS

Serum level of potassium was significantly high in crisis patients when compared with steady state patients (P = 0.000). Serum level of sodium was significantly low in crisis patients when compared with steady state patients (P = 0.000). There was no statistically significant differences in serum level of potassium regarding patients gender (*P* 0.355). There was no statistically significant differences in serum level of sodium regarding patients gender (*P* 0.174).

There was no statistically significant differences in serum level of potassium according patients age (P 0.560). There was no statistically significant differences in serum level of sodium regarding patients age (*P* 0.378).

DISCUSSION

Sickle cell anemia (SCA) is inherited hemolytic anemia result in destruction of red cell membrane, this resulting in disturbance in sodium and potassium metabolism. The study aimed to estimate serum levels of sodium and potassium in patients with sickle cell anemia. This analytical study was conducted in Wed Medani pediatric teaching hospital, Gezira State, during July to October 2016. The study involved 60 patients with sickle cell anemia, both sex in age range of 6-17 years**.** Serum level of potassium was significantly high in crisis patients when compared with steady state patients (M± SD= 6.347±0.2809, 4.285 ±0.7649, *P* = 0.000) respectively as presented in table (1). This due to membrane damage causes movement of potassium ions and water out of the cell by the Gardos pathway and potassium – chloride cotransport, leading to dehydration of red cells.[8] This findings agree in case of crisis state patients and disagree in case of steady state patients with study done by Agoreyo and Nwanze, (2012) whom found that there was a statistically significant increase (*P* < 0.05) in the concentration of potassium in both the steady state (4.58 ± 0.171 mmol/l) and crisis state (4.66 ± 0.154 mmol/l).[11] Serum level of sodium was significantly low in crisis patients when compared with steady state patienst (M± SD= 130.65. ±4.898, 137.84 ±4.530, *P* = 0.000) respectively as presented in table (2). This due to sodium loss. This findings agree in case of crisis state and disagree in case of steady state patients with study done by Agoreyo and Nwanze, 2012 whom found that there was a statistically significant reduction in the concentration of sodium in both the steady state (129.40 ± 1.462) and crisis state (121.60 ± 0.678).[11] There was no significantly difference in serum levels of potassium and sodium between gender groups (*P* 0.355, *P* 0.174) respectively as presented in table (3, 4) respectively. There was no significantly difference in serum levels of potassium and sodium between age groups (*P* 0.560, P 0.378) respectively as presented

in table (5, 6) respectively. This disagree with Agoreyo and Nwanze, 2012 whom found that there was a correlation between patients serum potassium and sodium level with gender "increased potassium and decreased sodium in male only".

CONCLUSION

This study conducted that Serum level of potassium was significantly increased in crisis state patients (M± SD= 6.347±0.2809) and Serum level of sodium was significantly low in crisis state patients ((M± SD= 130.65. ±4.898). Patients age and gender do not have any significant affect on serum levels of potassium and sodium.

RECOMMENDATIONS

1. The study recommended to evaluate the potassium level in patients with sickle cell anemia to reduce complication.
2. More studies can be done to evaluate the role of blood transfusion and correlation of serum potassium level**.**

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