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Assessment of the Associations between Serum Electrolytes (Sodium, Potassium, Calcium and Magnesium) and Sudanese Hypertensive patients in Different Hospitals at Khartoum State -Sudan

A Thesis Submitted in Partial Fulfillment for the Requirements of the Master Degree in Medical Laboratory Sciences (Clinical Chemistry)

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بسم الله الرحمن الرحيم

قال تعالي:

(إِنَّ اللَّهَ عِنْدَهُ عِلْمُ السَّاعَةِ وَيُنَزِّلُ الْغَيْثَ وَيَعْلَمُ مَا فِي الأَرْحَامِ وَمَا تَدْرِي نَفْسٌ مَاذَا تَكْسِبُ غَداً وَمَا تَدْرِي نَفْسٌ بِأَيِّ أَرْضٍ تَمُوتُ إِنَّ اللَّهَ عَلِيمٌ خَبِيرٌ)

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Declaration and Statement

I Nedal Babiker, under signed, declare and affirm that this Thesis is my own original work. I have followed all ethical and technical principles in the preparation, data collection, data analysis and compilation of this Thesis. Any scholarly matter that is included in the Thesis has been given recognition through citation. I, solemnly declare that this Thesis has not been submitted to any other institution anywhere for the award of any academic degree, diploma or certificate.

Signature .....

Date ……..

Dedication

I would like to dedicate my research to all those who helped me and guided me to the best.

To my Mother and Father

To my brothers and sisters

To my friends and colleagues.

Acknowledgments

First of all, I render my thanks and praise to almighty Allah, who offered me the health and strength to accomplish this work.

With these few words I would like to express my extremely gratefulness to my supervisor

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Special thanks to my mother and father

My deepest appreciation and gratitude to all person who helped me in this work

Finally, I would like to acknowledge the staff members of the Department of clinical chemistry- faculty of graduate studies and scientific research.

Abstract:

This study was performed in different hospitals in Khartoum State regarding hypertensive patients in a period from March to August 2018.

The aim of this study was to assess the association between hypertension and serum (sodium, potassium, magnesium and calcium) in hypertensive Sudanese males and females.

This study presented that there were non-significant correlations between levels of (sodium, potassium, magnesium and calcium) of hypertensive patients compared to normal range. The illustrated that there were non-significant correlations between age, duration, social status, gander, job, education status of hypertensive tested group.

The study revealed that there was a finding referring to significant correlation between level of (magnesium) and economic status of test group, but non-significant in (sodium, potassium, magnesium).

Based on the finding of this study –can be concluded that there is no relations between hypertension and the level of blood (Na^{+}, K^{+}, Ca^{2+} and Mg^{2+})

الخلاصة:

أجريت هذه الدراسة في مختلف المستشفيات في ولايه الخرطوم في الفترة من مارس الي أغسطس٢٠١٨. وكان الهدف من هذه الدراسة تقيم تأثير الضغط على مستوى نسبه الصوديوم والبوتاسيوم والماغنيسيوم والكالسيوم في الدم على مرضى الضغط السودانيين من الذكور والاناث.

لاحظت الدراسة عدم وجود علاقة بين نسبه الصوديوم والبوتاسيوم والماغنيسيوم والكالسيوم في الدم لمرضى ارتفاع ضغط الدم مقارنه بالوضع الطبيعي. كما أثبتت عدم وجود علاقة بين العمر، الفترة الزمنية للمرض، الحالة الاجتماعية، الجنس، الوظيفة والمستوي التعليمي للمرضي.

كما اشارت الدراسة لوجود علاقة بين مستوي الماغنيسيوم وبين الحالة الاقتصادية للمرضي. ولكن لم توجد علاقة بين مستوى الصوديوم و البوتاسيوم والكالسيوم.

اعتمادا علي ما وجد في هذه الدراسه نستخلص عدم وجود علاقه بين ارتفاع ضغط الدم ومستوي نسبه الصوديوم واالبوتاسيوم والكالسيوم والماغنيسيوم في الدم

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List of abbreviations

ADH	Antidiuretic hormone
ATP	Adenosine triphosphate
BP	Blood pressure
Ca^{2+}	Calcium
CKD	Chronic kidney diseases
DNA	Deoxyribonucleic acid
ECG	Electrocardiogram
GWAS	Genome-wide association studies
HTN	Hypertension
HBP	High blood pressure
K^{+}	Potassium
MEN	Multiple endocrine neoplasia
Mg^{2+}	Magnesium
MmHg	Millimeters mercury
MEq/L	MilliEquivalent per liter
Mmol/L	Millimol per liter
Mg/dl	Milligram per liter
Na^{+}	Sodium
SNP	Single nucleotide polymorphism
SLE	selective electrode
SPSS	Social sciences software package
RAS	Renal artery stenosis
PPI	Proton pump inhibitors

Chapter One

Introduction

Rationale

Objectives

1.1 Introduction:

Hypertension (HTN) refer to high blood pressure (HBP), defined by American Heart Association (AHA), Blood pressure (BP) is determined by cardiac output and peripheral vascular resistance.[1] Systolic BP, which is the peak pressure in the arteries, represents the ejection of blood into the aorta during ventricular systole. It is largely determined by stroke volume, the elastic properties of aorta and the velocity of ejection.[5] Diastolic BP refers to the lowest pressure in the arterial system during diastole. the diastolic blood lowest pressure in the arterial system during diastole pressure rises when peripheral vascular resistance increases.[5] change with age. Although the precise mechanism is not understood, the contributing factors of aging to increased BP may include arterial stiffness, atherosclerosis, decreased functional decreased functional efficiency of the heart, age-related changes in hormone profiles, and salt-sensitivity among older people etc. In some non-industrialized countries, BP is less likely to increase as people age.[2] This difference may be explained by differences in diet and stress, among other things.[3]

Systolic BP increases with age for men and women, while diastolic BP rises up to the age of (50-59) years Thus, the difference between systolic and diastolic BP (defined as pulse pressure), increases steeply with age in the elderly population. Increasing evidence suggests that high pulse pressure in the elderly marker of increased artery stiffness and widespread atherosclerosis. Elevated pulse pressure is also recognized as an enhanced risk for cardiovascular events. BP reading of (>=140 mm Hg) systolic and/or (>=90 mm Hg) diastolic or any treatment for (HTN). For people who are categorized as normal, a BP reading of (<120mm Hg) systolic and (< 80mm Hg) diastolic, whereas a BP reading of (120-139 mm Hg) systolic and/or (80-89Hg) diastolic is categorized as “pre hypertension”. [2][5] HTN for those who are not on antihypertensive treatment consists of two stages: stage 1: BP reading of (140-159mm Hg) systolic and/or (90-99 mm Hg) diastolic: and stage 2 BP reading of (>=160mm Hg) systolic and/or (>=100 mmHg) diastolic.[2][4] Prevalence of systolic HTN is directly

proportional to advancing age. It is estimated that more than half of Americans over age (65) years had isolated systolic or combined systolic-diastolic HTN while fewer than (10%) of individuals in this age group had diastolic HTN in 2005. HTN is a well-known independent risk factor for cardiovascular disease (CVD), stroke, and renal failure (RF).[4] Globally, the overall prevalence of HTN in adults aged (18) and over was (22%) in 2014. HTN is one of the leading risk factors for global mortality.[5] An electrolyte is any substance that contains free ions that behaves as an electrically conductive medium (conducts electricity).

Sodium (Na^+) has a pulling effect on water it affects extracellular fluids equally (plasma and intestinal). However, because there is considerably more Na^+ outside cells than inside, the water is pulled out of cells into extracellular fluid. Na^+ determines osmotic pressure of extracellular fluid. Diet sodium is easily absorbed from the diet Na^+-K^+ ATP-ase enzyme regulate Na^+ and K^+ pump, pumps Na^+ out and K^+ into cells. Without this active transport pump, the cells would fill with Na^+ and subsequent osmotic pressure would rupture the cells. The increase in "effective" body Na^+ usually causes a rise in BP that characterized by an increase in peripheral vascular resistance.

High Na^+ diet (>2800 mg/day) risk in heart failure was associated with of acute decompensated heart failure, all-cause hospitalization, and all-cause mortality over a median three years' follow-up period. people with high Na^+ intake and low potassium (K^+) diet alters the electrolyte balance inpatients of HTN developed higher grades of coronary collaterals which lead to coronary disease.[6] Magnesium (Mg^{+2}) has additive antihypertensive effects. Furthermore, Mg^{+2} is more involved in the functional vascular changes, and also on local metabolic stability with no influence on the vascular structure. Calcium (Ca^{+2}) ion affects BP acting as regulator of hormones have all been found to have vasoactive properties and therefore may influence BP.

1.2. Rationale:

Hypertension increases the risk of heart failure and kidney failure.[4] Worldwide, hypertension is estimated to cause (7.5) million deaths, about (12.8%) of the total of all deaths. In Africa, however, more than (40%) (and up to 50%) of adults in many countries are estimated to have hypertension.[5]

1.3. Objectives:

1.3.1 General Objective:

-To identify the significant associations between serum electrolytes in Sudanese hypertensive peoples.

1.3.2 Specific objectives:

-To assess the association between hypertension and electrolytes by measuring the following:

- Sodium.
- Potassium.
- Magnesium.
- Calcium.

-To assess the effect of hypertension on serum levels of electrolytes.

-To evaluate the effect the duration of hypertension on serum electrolytes.

-To correlate between the age, gender, education status, marital status of hypertensive Sudanese peoples on serum electrolytes.

-To correlate between job, economic status effect of hypertensive Sudanese peoples on serum electrolytes (Na^{+}, K^{+}, Mg^{+2} and Ca^{+2})

Chapter Two

Literature Review

2. Literature Review:

2.1 Hypertension:

Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the BP in the arteries is persistently elevated.[13] HBP usually does not cause symptoms.[7] Long-term HBP, however, is a major risk factor for coronary artery disease (CAD), stroke, heart failure, atrial fibrillation, peripheral vascular disease, vision loss, chronic kidney disease (CKD), and dementia.[8]

2.1.1 Classification of Hypertension

2.1.1.1Primary (Essential) Hypertension:

About (90–95%) of cases are primary, defined as HBP due to nonspecific lifestyle and genetic factors.[9] Lifestyle factors that increase the risk include excess salt in the diet, excess body weight, smoking, and alcohol use.[7][9]

2.1.1.2 Secondary Hypertension:

The remaining (5–10%) of cases are categorized as secondary HBP, defined as HBP due to an identifiable cause, such as CKD, narrowing of the kidney arteries, an endocrine disorder, or the use of birth control pills.[9]

BP is expressed by two measurements, the systolic and diastolic pressures, which are the maximum and minimum pressures, respectively.[7] For most adults, normal blood pressure at rest is within the range of (100–130 mmHg) systolic and (60–80 mmHg) diastolic. For most adults, high blood pressure is present if the resting BP is persistently at or above (130/90 or 140/90 mmHg).[9] Different numbers apply to children. Ambulatory over a (24-hours) period appears more accurate than office-based BP measurement.[9][13] Lifestyle changes and medications can lower blood pressure and decrease the risk of health complications.[11] Lifestyle changes include weight loss, decreased salt intake, physical exercise, and a healthy diet.[9] If lifestyle changes are not sufficient then BP medications are used.[11] Up to three medications can control BP in (90%) of people.[9] The treatment of moderately high arterial BP (defined as >160/100

mmHg) with medications is associated with an improved life expectancy. The effect of treatment of blood pressure between (130/80 mmHg) and (160/100 mmHg) is less clear, with some reviews finding benefit[10] and others finding unclear benefit. HBP affects between 16 and 37% of the population globally.[9] In 2010 HTN was believed to have been a factor in 18% of all deaths (9.4 million globally).[12]

2.1.1.3 Hypertensive Crisis:

Severely elevated BP (equal to or greater than a systolic 180 or diastolic of 110) is referred to as a hypertensive crisis. Hypertensive crisis is categorized as either hypertensive urgency or hypertensive emergency, according to the absence or presence of end organ damage, respectively.[17]

In hypertensive urgency, there is no evidence of end organ damage resulting from the elevated BP. In these cases, oral medications are used to lower the BP gradually over (24 to 48 hours). [18]

In hypertensive emergency, there is evidence of direct damage to one or more organs. The most affected organs include the brain, kidney, heart and lungs, producing symptoms which may include confusion, drowsiness, chest pain and breathlessness.[18] In hypertensive emergency, the BP must be reduced more rapidly to stop ongoing organ damage,[18] however, there is a lack of randomized controlled trial evidence for this approach.

2.1.1.4 Gestational Hypertension and Pre-Eclampsia:

Hypertension occurs in approximately (8–10%) of pregnancies. [16] Two BP measurements six hours apart of greater than (140/90 mm Hg) is diagnostic of HTN in pregnancy. High blood pressure in pregnancy can be classified as pre-existing hypertension, gestational hypertension, or pre-eclampsia.[19]

Pre-eclampsia is a serious condition of the second half of pregnancy and delivery characterized by increased blood pressure and the presence of protein in the urine.[16] It occurs in about (5%) of pregnancies and is responsible for approximately (16%) of all maternal deaths globally.[16] Pre-eclampsia also

doubles the risk of death of the baby around the time of birth.[16] Usually there are no symptoms in pre-eclampsia and it is detected by routine screening. When symptoms of pre-eclampsia occur the most common are headache, visual disturbance (often "flashing lights"), vomiting, pain over the stomach, and swelling. Pre-eclampsia can occasionally progress to a life-threatening condition called eclampsia, which is a hypertensive emergency and has several serious complications including vision loss, brain swelling, seizures, kidney failure, pulmonary edema, and disseminated intravascular coagulation (a blood clotting disorder).[16]

In contrast, gestational hypertension is defined as new-onset hypertension during pregnancy without protein in the urine.[19]

2.1.1.5 Hypertension in Children:

Failure to thrive, seizures, irritability, lack of energy, and difficulty in breathing[20] can be associated with HTN in newborns and young infants. In older infants and children, HTN can cause headache, unexplained irritability, fatigue, failure to thrive, blurred vision, nosebleeds, and facial paralysis.[20]

2.1.2 Signs and Symptoms of Hypertension:

HTN is rarely accompanied by symptoms, and its identification is usually through screening, or when seeking healthcare for an unrelated problem. Some with HBP report headaches (particularly at the back of the head and in the morning), as well as lightheadedness, vertigo, tinnitus (buzzing or hissing in the ears), altered vision or fainting episodes.[14] These symptoms, however, might be related to associated anxiety rather than the high blood pressure itself. On physical examination, NTN may be associated with the presence of changes in the optic fundus seen by ophthalmoscopy.[15] The severity of the changes typical of hypertensive retinopathy is graded from I–IV; grades I and II may be difficult to differentiate.[15] The severity of the retinopathy correlates roughly with the duration or the severity of the HTN.[14]

2.1.2.1 Secondary Hypertension Signs:

Hypertension with certain specific additional signs and symptoms may suggest secondary hypertension, i.e. hypertension due to an identifiable cause. For example, Cushing's syndrome frequently causes truncal obesity, glucose intolerance, moon face, a hump of fat behind the neck/shoulder (referred to as a buffalo hump), and purple abdominal stretch marks.[16]Hyperthyroidism frequently causes weight loss with increased appetite, fast heart rate, bulging eyes, and tremor. Renal artery stenosis (RAS) may be associated with a localized abdominal bruit to the left or right of the midline (unilateral RAS), or in both locations (bilateral RAS). Coarctation of the aorta frequently causes a decreased blood pressure in the lower extremities relative to the arms, or delayed or absent femoral arterial pulses. Pheochromocytoma may cause abrupt ("paroxysmal") episodes of hypertension accompanied by headache, palpitations, pale appearance, and excessive sweating.[16]

2.1.3 Causes of Hypertension:

2.1.3.1 Primary hypertension Causes:

HTN results from a complex interaction of genes and environmental factors. Numerous common genetic variants with small effects on blood pressure have been identified[2] as well as some rare genetic variants with large effects on blood pressure. Also, genome-wide association studies (GWAS) have identified (35) genetic loci related to blood pressure; (12) of these genetic loci influencing blood pressure were newly found.[22] Sentinel SNPs for each new genetic loci identified has shown an association with DNA methylation at multiple nearby CpG sites. These sentinel SNPs are located within genes related to vascular smooth muscle and renal function. DNA methylation might affect in some way linking common genetic variation to multiple phenotypes even though mechanisms underlying these associations are not understood. Single variant test performed in this study for the (35) sentinel SNPs (known and new) showed that genetic variants singly or in aggregate contribute to risk of clinical phenotypes

related to high blood pressure.[22] BP rises with aging and the risk of becoming hypertensive in later life is considerable. Several environmental factors influence BP. High salt intake raises the blood pressure in salt sensitive individuals; lack of exercise, obesity, and depression can play a role in individual cases. The possible role of other factors such as caffeine consumption, and vitamin D deficiency are less clear. Insulin resistance, which is common in obesity and is a component of syndrome X (or the metabolic syndrome), is also thought to contribute to hypertension.[23] One review suggests that sugar may play an important role in hypertension and salt is just an innocent by standard .Events in early life, such as low birth weight, maternal smoking, and lack of breastfeeding may be risk factors for adult essential hypertension, although the mechanisms linking these exposures to adult hypertension remain unclear.[24] An increased rate of high blood urea has been found in untreated people with hypertensive in comparison with people with normal blood pressure, although it is uncertain whether the former plays a causal role or is subsidiary to poor kidney function. Average blood pressure may be higher in the winter than in the summer.[26]

2.1.3.2 Secondary Hypertension causes:

Secondary hypertension results from an identifiable cause. Kidney disease is the most common secondary cause of HTN.[16]HTN can also be caused by endocrine conditions, such as Cushing's syndrome, hyperthyroidism, hypothyroidism, acromegaly, Conn's syndrome or hyperaldosteronism, renal artery stenosis (from atherosclerosis or fibromuscular dysplasia) ,hyperparathyroidism, and pheochromocytoma.[16][26] Other causes of secondary hypertension include obesity, sleep apnea, pregnancy, coarctation of the aorta, excessive eating of liquor ice, excessive drinking of alcohol, and certain prescription medicines, herbal remedies, and illegal drugs such as cocaine and methamphetamine.[16]Arsenic exposure through drinking water has been shown to correlate with elevated BP.

2.2 Electrolyte:

An electrolyte is a compound that ionizes when dissolved in suitable ionizing solvents such as water. This includes most soluble salts, acids, and bases. Some gases, such as hydrogen chloride, under conditions of high temperature or low pressure can also function as electrolytes. Electrolyte solutions can also result from the dissolution of some biological (e.g., DNA, polypeptides) and synthetic polymers (e.g., polystyrene sulfonate), termed polyelectrolyte, which contain charged functional groups.[27] Electrolytes are present in the human body, and the balance of the electrolytes in bodies is essential for normal function of the cells and organs. [27]

2.2.1 Sodium:

Na^+ plays a key role in the body. It helps maintain normal BP, supports the work of the nerves and muscles, and regulates the body's fluid balance. [28] When the Na^+ level in the blood becomes too low, extra water enters the cells and causes them to swell. Swelling in the brain is especially dangerous because the brain is confined by your skull and unable to expand without causing symptoms. [28]

2.2.1.1 Sources of Sodium:

Na^+ occurs naturally in most foods. The most common form of Na^+ is sodium chloride, which is table salt. Milk, beets, and celery also naturally contain, Na^+ as does drinking water, although the amount varies depending on the source.

Na^+ is also added to various food products. Some of these added forms are monosodium glutamate, sodium nitrite, sodium saccharin, baking soda (sodium bicarbonate), and sodium benzoate. [29]

2.2.1.2 Function of Sodium:

Na^+ using for generation of nervous impulses and finer regulation of fluid balance. In animals, sodium ions (often referred to as just " Na^+") are necessary for regulation of blood and body fluids, transmission of nerve impulses, heart activity, and certain metabolic functions much or too little) can be fatal [30].

2.2.1.3 Disorder of Sodium Metabolism:

2.2.1.3.1 Hyponatremia:

Hyponatremia, also called water intoxication, is generally the result of drinking excessive amounts of plain water which causes a low concentration of Na^+ in the blood. Once a rare occurrence at sporting events, it is becoming more prevalent as participation increases and more novice exercisers are entering endurance events. (28)

Prolonged and excessive sweating increases the risk that an athlete will alter the delicate balance of blood-sodium concentration. Because Na^+ is lost in sweat it is important for those exercising at high intensities for long periods of time to replace any loses. (28)

i. Types of Hyponatremia:

A normal sodium level is between (135 and 145) (mEq/L) of sodium.

Hyponatremia occurs when the Na^+ in your blood falls below 135 mEq/L.

The imbalance between Na^+ and water in the blood may occur in three primary ways:

A. Hypovolemic Hyponatremia: in which the body has too much water. Hypovolemic hyponatremia is commonly the result of kidney failure, heart failure or liver failure.

B. Euvolemic Hyponatremia: the water level is too high. This condition is commonly due to chronic health conditions, cancer or certain medications.

C. Hypovolemic Hyponatremia: there is too little water and sodium. This may occur, for example, when exercising in the heat without replenishing your fluid electrolytes or with marked blood loss. (28)

ii. Causes of Hyponatremia:

Many conditions including congestive heart failure, liver failure, kidney failure and pneumonia can have an associated hyponatremia. It can also be caused by over hydration from drinking too much water (polydipsia).(28)

In the vast majority of cases, hyponatremia occurs as a result of excess body water diluting the serum Na^+ (salt level in the blood). Lack of sodium (salt) is very rarely the cause of hyponatremia, although it can promote hyponatremia indirectly. In particular, Na^+ loss can lead to a state of volume depletion (loss of blood volume in the body), with volume depletion serving as a signal for the release of ADH (anti-diuretic hormone). As a result of ADH-stimulated water retention (too much water in the body), blood Na^+ becomes diluted and hyponatremia results. (28)

iii. Symptoms of Hyponatremia:

Include nausea and vomiting, headache, confusion, lethargy, fatigue, loss of appetite, restlessness and irritability, muscle weakness, spasms, or cramps, seizures, and decreased consciousness or coma. The presence and severity of symptoms are associated with the level of serum Na^+ (salt level in the blood), with the lowest levels of serum Na^+ associated with the more prominent and serious symptoms (the less the salt the more severe the symptoms). However, emerging data suggest that mild hyponatremia serum Na^+ levels at (131 mEq/L) or above is associated with numerous complications and undiagnosed symptoms. (31)

iv. Complications of Hyponatremia:

Chronic hyponatremia can lead to such complications as neurological impairments. These neurological impairments most often affect gait (walking) and attention, and can lead to falls, osteoporosis, and decreased reaction time.

Acute hyponatremia can lead to much more serious complications including brain disease, brain herniation, cardiopulmonary arrest (heart attack), cerebral edema (brain swelling), seizures, coma and death.(32)

2.2.1.3. 2 Hypernatremia:

Is an electrolyte disturbance that is defined by an elevated Na^+ level in the blood.(33) Hypernatremia is generally not caused by an excess of, but Na^+ rather

by a relative deficit of free water in the body. For this reason, hypernatremia is often synonymous with the less precise term, dehydration.

Water is lost from the body in a variety of ways, including perspiration, imperceptible losses from breathing, and in the feces and urine. If the amount of water ingested consistently falls below the amount of water lost, the serum Na^+ level will begin to rise, leading to hypernatremia. Rarely, hypernatremia can result from massive salt ingestion,[34] such as may occur from drinking seawater.

Ordinarily, even a small rise in the serum Na^+ concentration above the normal range results in a strong sensation of thirst, an increase in free water intake, and correction of the abnormality. Therefore, hypernatremia most often occurs in people such as infants, those with impaired mental status, or the elderly, who may have an intact thirst mechanism but are unable to ask for or obtain water.[34]

i. Signs and Symptoms of Hypernatremia:

Clinical manifestations of hypernatremia can be subtle, consisting of lethargy, weakness, irritability, neuromuscular excitability, and edema. With more severe elevations of the sodium level, seizures and coma may occur. Severe symptoms are usually due to acute elevation of the Na^+ in plasma concentration to above (157 mEq/L), values above (180 mEq/L) are associated with a high mortality rate, particularly in adults. [34]

2.2.2 Potassium:

Potassium (K^+) is an essential mineral macronutrient and is the main intracellular ion for all types of cells. It is important in maintaining fluid and electrolyte balance in the bodies of humans and animals . [35]

2.2.2.1 Sources of Potassium:

Eating a variety of foods that contain K^+ is the best way to get an adequate amount. Healthy individuals who eat a balanced diet rarely need supplements. Foods `with high sources of K^+ include kiwifruit, orange juice, potatoes, bananas, coconut, avocados, apricots, parsnips and turnips, although many other fruits, vegetables, legumes, and meats contain $K.^+$[35]

2.2.2.2Functions of Potassium:

K^+ is a very important mineral to the human body for Building proteins, Break down and use carbohydrates, Building muscle, maintain normal body growth, Control the electrical activity of the heart and control the acid-base balance. (36)

2.2.2.3 Disorder of Potassium Metabolism:

2.2.2.3.1Hyperkalemia:

Hyperkalemia is a potentially life-threatening metabolic problem caused by inability of the kidneys to excrete, K^+ impairment of the mechanisms that move potassium from the circulation into the cells, or a combination of these factors.(37)

Acquired hyporeninemic hyperaldosteronism Addison's disease, Congenital adrenal hyperplasia (recessive or autosomal dominant), Mineralocorticoid deficiency, Primary hyperaldosteronism or hyporeninemia, Pseudohypoaldosteronism, renal insufficiency or failure, systemic lupus erythematosus, type IV renal tubular acidosis, acidosis, damage to tissue from rhabdomyolysis, burns, or trauma, familial hyperkalemic periodic paralysis, hyperosmolar states, (e.g., uncontrolled diabetes, glucose infusions), insulin deficiency or resistance, tumor lysis syndrome. · (38)

i. Symptoms of Hyperkalemia:

Symptoms are fairly nonspecific and generally include malaise, palpitations and muscle weakness; mild hyperventilation may indicate a compensatory response to metabolic acidosis, which is one of the possible causes of hyperkalemia. Often, however, the problem is detected during screening blood tests for a medical disorder, or it only comes to medical attention after complications have developed, such as cardiac arrhythmia or sudden death.(37)

ii. Complication of Hyperkalemia:

Too much K^+ in the blood from the improper functioning of the kidneys results in impaired nerve function. People experiencing hyperkalemia often feel unusual sensations, such as a tingling or numbness throughout the body. (40)

Arrhythmias or abnormal heart rhythms are a potentially life-threatening complication occurring from hyperkalemia. Individuals experiencing arrhythmias display irregular heartbeats· High potassium causes a change in muscle control, resulting in cardiac arrest or an absent or weak heartbeat. (40)

2.2.2.3.2 Hypokalemia:

Hypokalemia is a metabolic disorder that occurs when the level of K^+ in the blood drops too low. (41)

i. Causes of Hypokalemia:

Antibiotics (penicillin, nafcillin, carbenicillin, gentamicin, amphotericin B, foscarnet), diarrhea (including the use of too many laxatives, which can cause diarrhea diseases that affect the kidneys' ability to retain potassium (Liddle syndrome, Cushing syndrome, hyperaldosteronism, Bartter syndrome, Fanconi syndrome) diuretic medications, which can cause excess urination, eating disorders (such as bulimia), eating large amounts of licorice or using products such as herbal teas and chewing tobaccos that contain licorice made with glycyrrhetinic acid (this substance is no longer used in licorice made in the United States),magnesium deficiency, Sweating, Vomiting.(41)

ii. Symptoms of Hypokalemia:

Abnormal heart rhythms (dysrhythmias), especially in people with heart disease, constipation, fatigue, muscle damage (rhabdomyolysis), muscle weakness or spasms and paralysis (which can include the lungs).(41)

ii. Complication of Hypokalemia:

Hypokalemia can cause irregular heartbeats, known medically as cardiac dysrhythmias. The heartbeat may be unusually fast, a condition called tachycardia. Severe dysrhythmias can lead to cardiac arrest and lung paralysis.

Severe vomiting and diarrhea in small children, which leads to fluid loss and hypokalemia, can cause cardiac arrest. Hypokalemia can also cause high blood pressure or hypertension.

Hypokalemia can cause fatigue. A person may experience muscle weakness, cramping and muscle pain (myalgia). Reduced blood flow to the skeletal muscles caused by hypokalemia can lead to rhabdomyolysis, a condition in which the muscle fibers break down. Hypokalemia can lead to paralysis, affecting the legs more frequently than the arms, although that complication is uncommon. Diabetic patients with hypokalemia frequently have hyperglycemia, or high blood sugar levels. Hypokalemia interferes with the release of insulin and with the body's sensitivity to insulin. Controlling and treating hypokalemia can help reduce hyperglycemia and the diabetic complications it brings. (42)

2.2.3 Calcium:

Is a chemical element with symbol $Ca^{2+}$and atomic number 20. Calcium is the fifth most abundant element in the human body and the most abundant metal.

2.2.3.1 Functions of Calcium:

Calcium (Ca^{2+}) ions play a vital role in the physiology and biochemistry of organisms and the cell as electrolytes. They play an important role in signal transduction pathways, where they act as a second messenger, in neurotransmitter release from neurons, in contraction of all muscle cell types, and in fertilization. Many enzymes require Ca^{2+} ions as a cofactor. Ca^{2+}ions outside cells are also important for maintaining the potential difference across excitable cell membranes, as well as proper bone formation.

2.2.3.2 Disorders of Calcium Metabolism:

Occur when the body has too little or too much calcium. The serum level of is Ca^{2+} closely regulated within a fairly limited range in the human body. In a healthy physiology, extracellular Ca^{2+} levels are maintained within a tight range through the actions of parathyroid hormone, vitamin D and the Ca^{2+}sensing

receptor.[43] Disorders Ca^{2+} in metabolism can lead to hypocalcemia, decreased plasma levels of Ca^{2+} or hypercalcemia, elevated plasma Ca^{2+} levels.

2.2.3.2.1Hypocalcemia:

Hypocalcaemia, is low calcium levels in the blood serum.[46] The normal range is (2.1–2.6 mmol/l) (8.8–10.7 mg/dl, 4.3–5.2 mEq/l) with levels less than (2.1 mmol/l) defined as hypocalcemia. [44] Mildly low levels that develop slowly often have no symptoms.[45] Otherwise symptoms may include numbness, muscle spasms, seizures, confusion, or cardiac arrest.[44][45]

i. Causes of hypocalcemia:

Common causes include hypoparathyroidism and vitamin D deficiency.[45] Others causes include kidney failure, pancreatitis, calcium channel blocker overdose, rhabdomyolysis, tumor lysis syndrome, and medications such as bisphosphonates.[44] Diagnosis should generally be confirmed with a corrected calcium or ionized Ca^{2+} level.[45] Specific changes may be seen on an electrocardiogram (ECG).[44]

ii. Signs and Symptoms of Hypocalcemia:

The neuromuscular symptoms of hypocalcemia are caused by a positive bathmotropic effect due to the decreased interaction of Ca^{2+} with sodium channels. Since Ca^{2+}blocks sodium channels and inhibits depolarization of nerve and muscle fibers, reducedCa^{2+}lowers the threshold for depolarization. [47]

iii. Complications of Hypocalcemia:

Complications from calcium deficiency disease include eye damage, an abnormal heartbeat, and osteoporosis.

Complications from osteoporosis include:

• Disability

• Spinal fractures or other bone fractures

• Difficulty walking

If left untreated, Ca^{2+} deficiency disease could eventually be fatal.

2.2.3.2.2 Hypercalcemia:

Hypercalcemia, is a high calcium (Ca^{2+}) level in the blood serum.[48] The normal range is(2.1–2.6 mmol/L) (8.8–10.7 mg/dL, 4.3–5.2 mEq/L) with levels greater than(2.6 mmol/L) defined as hypercalcemia.[48][49] Those with a mild increase that has developed slowly typically have no symptoms.[48]

i. Causes of Hypercalcemia:

Most cases are due to primary hyperparathyroidism or cancer.[48] Other causes include sarcoidosis, tuberculosis, Paget disease, multiple endocrine neoplasia (MEN), vitamin D toxicity, familial hypocalciuric hypercalcemia, and certain medications such as lithium and hydrochlorothiazide.[48][49]

ii. Signs and Symptoms of Hypercalcemia:

The neuromuscular symptoms of hypercalcemia are caused by a negative bathmotropic effect due to the increased interaction of Ca^{2+} with sodium channels. Since calcium blocks sodium channels and inhibits depolarization of nerve and muscle fibers, increased Ca^{2+}raises the threshold for depolarization. [50] This results in diminished deep tendon reflexes (hyporeflexia), and skeletal muscle weakness. There is a general mnemonic for remembering the effects of hypercalcemia: "Stones, Bones, Groans, Thrones and Psychiatric Overtones"

iii. Complications of Hypercalcemia:

Hypercalcemia complications can include:

Osteoporosis. If your bones continue to release calcium into your blood, you can develop the bone-thinning disease osteoporosis, which could lead to bone fractures, spinal column curvature and loss of height.

Kidney stones. If your urine contains too much calcium, crystals may form in your kidneys. Over time, the crystals may combine to form kidney stones. Passing a stone can be extremely painful.

Kidney failure. Severe hypercalcemia can damage your kidneys, limiting their ability to cleanse the blood and eliminate fluid.

Nervous system problems. Severe hypercalcemia can lead to confusion, dementia and coma, which can be fatal.

Abnormal heart rhythm (arrhythmia). Hypercalcemia can affect the electrical impulses that regulate your heartbeat, causing your heart to beat irregularly.

2.2.4 Magnesium:

Magnesium (Mg^{2+}) is the second most abundant intracellular cation and the fourth most abundant cation in the body.

2.2.4.1 Functions of Magnesium:

Mg^{2+} plays an essential physiological role in many functions of the body. This role is achieved through two important properties of magnesium; the ability to form chelates with important intracellular anionic-ligands, especially ATP, and its ability to compete with calcium for binding sites on proteins and membranes. [51] [52] Mg^{2+} is essential for the synthesis of nucleic acids and proteins, for intermediary metabolism and for specific actions in different organs such as the neuromuscular and cardiovascular systems. Over (300) enzymes are dependent on Mg^{2+}. Magnesium influences the activity of enzymes by (i) binding to ligands such as ATP in ATP-requiring enzymes, (ii) binding to the active site of the enzyme, such as: enolase, pyruvate kinase, pyrophosphatase, (iii) causing a conformational change during the catalytic process, such as: Na^{+}, K^{+}-ATPase, (iv) promoting the aggregation of multi-enzyme complexes such as: aldehyde dehydrogenase.

2.2.4.2 Disorders of Magnesium Metabolism:

2.2.4.2.1 Hypomagnesemia:

Hypomagnesemia, is an electrolyte disturbance in which there is a low level of magnesium in the blood.[53]Normal magnesium levels are between (1.46–2.68 mg/Dl) (0.6-1.1 mmol/L) with levels less than (1.46 mg/dL)(0.6 mmol/L) defining hypomagnesemia.

i. Causes of Hypomagnesemia: It may be caused by:

- Malabsorption syndromes, including:
  - Celiac disease.
  - Crohn's disease and ulcerative colitis.
  - Chronic diarrhoea.
  - Steatorrhoea.
  - Short bowel syndrome.
  - Prolonged nasogastric suction.
- Protein-calorie malnutrition. Dietary deficiency causing symptomatic hypomagnesaemia in otherwise healthy individuals is uncommon. Anorexia nervosa may be a cause.
- Disorders of the parathyroid gland.
- Chronic alcoholism - Mg^{2+} depletion occurs via a number of mechanisms in this condition. It has also been found in those with nonalcoholic fatty liver. (55)
- Patients on long-term proton pump inhibitors (PPIs). There is emerging recognition that long-term PPIs can cause hypomagnesaemia, but debate about how to monitor or prevent this. It is a rare side-effect, is associated with all PPIs, and is often accompanied by low potassium and calcium levels. (56) Levels recover quickly when medication is stopped, but drop again when restarted, even with a different PPI. H2 receptor antagonists do not have the same effect. (54)
- Other medications. These include diuretics, digoxin, calcineurin inhibitors, theophylline, cisplatin and some aminoglycosides. These mostly have the result of reducing reabsorption of Mg^{2+} within the kidney via a variety of mechanisms.
- Renal disorders causing reduced Mg^{2+}resorption - acute tubular necrosis, post-obstructive diuresis, renal tubular acidosis, post-kidney transplantation.

- Diabetes (due to glucose-induced diuresis secondary to poor glucose control).
- Acute pancreatitis.
- Re-feeding syndrome. [57]
- Genetic causes. Inherited forms exist
- Severe burns.

ii. Signs and Symptoms of Hypomagnesaemia:

Most cases are asymptomatic until levels of Mg^{2+}drop below (0.5 mmol/L). It is commonly associated with other metabolic abnormalities such as hypokalemia, hypocalcemia and metabolic acidosis, making it difficult to distinguish the symptoms of hypomagnesaemia itself. **[58]** Features may include:

- Neuromuscular symptoms:
    - Weakness and apathy.
    - Tremor.
    - Paraesthesia.
    - Tetany.
    - Muscle fasciculations.
    - Seizures, drowsiness, confusion and coma when very low levels of Mg are reached.
- Cardiovascular features:
    - Arrhythmias.
    - ECG signs may include wide QRS complexes, a prolonged QT interval, flattened T waves and the presence of U waves.

iii. Complications of Hypomagnesaemia:

The possible complications include:

- Cardiac arrest
- Respiratory arrest
- Death

- Hypocalcemia (reduced serumCa^{2+}levels), due to inhibition of parathyroid hormone bioactivity (hypoparathyroidism)
- Increased intracellular calcium level

2.2.4.2.2 Hypermagnesemia:

Hypermagnesemia is an electrolyte disturbance in which there is a high level of magnesium in the blood. It is defined as a level greater than (1.1 mmol/L). [59]

i. Causes of Hypermagnesemia:

It is most frequently encountered in patients with end-stage kidney disease, those taking medication containing Mg^{2+} (particularly laxatives, antacids and rectal enemas) and those on parenteral nutrition. In healthy individuals, excess intake is excreted by the kidneys. Other causes of mildly raised Mg^{2+}levels include lithium therapy, dialysis, hypercalcemia, hypothyroidism and Addison's disease. It may also occur in neonates born to mothers receiving IV Mg^{2+}therapy for pre-eclampsia. [59]

ii.Signs and Symptoms of Hypermagnesemia: Features include:

- Nausea and vomiting.
- Facial flushing.
- Hypotension.
- Paralytic ileus (due to smooth muscle paralysis).
- Weakness, followed by flaccid muscle paralysis.
- Disappearance of deep tendon reflexes.
- Respiratory depression.
- Bradycardia
- Complete heart block or cardiac arrest (at levels >6.0-7.5 mmol/L).

iii.Complications of Hypermagnesemia:

- Abnormal heart rhythms and a systole are possible complications of Hypermagnesemia related to the heart. [59]

2.3 Previous Study:

Jiang He and GretheS.Tell et al, From Peking Union Medical College and Beijing, People's Republic of China (1991). They done previous study to assessment the effect of hypertension in serum Na^{+}, K^{+}, Mg^{2+} and Ca^{2+},they reported non-significant change in serum level of Sodium , magnesium and calcium of test group with normal range ,But they founding negative significant change between potassium level of test group and normal range.

Chapter Three

Materials and Methods

3 Materials and methods

3.1. Study Approach:

A quantitative approach was used to measure Na^{+}, K^{+}, Mg^{2+} and Ca^{2+} in hypertensive Sudanese patients in period from March to Augustus 2018.

3.2. Study Type and design:

prospective, hospitals/laboratory based study.

3.3. Study Area:

Different hospitals at Khartoum State

3.4. Sample size:

The test group covered (30) patients with hypertension

3.5. Selection criteria:

3.5.1 Criteria of Inclusion:

The criteria of inclusion based on patients with hypertension.

3.5.2 Criteria of Exclusion:

The criteria of exclusion based on other disorder like renal disease, heart disease, liver disease.

3.6. Study variables:

Plasma sodium, potassium, magnesium and calcium measured by semi automation instruments.

3.7. Sampling:

Non probability sampling was carried out.

3.7.1 Sampling frame:

Khartoum State

3.7.2. Sample unit:

The study was restricted on hypertensive patients.

3.7.3 Sample Size:

(30) samples

The restriction of the sample size to (30) subjects is due to lack of financial support.

3.8. Data Collection Method and tools:

3.8.1. Data collection:

Data were collected using structural interviewing questionnaire. which is designed to collect and maintain all valuable information concerned each case examined.

3.8.2. Specimen Collection:

(2.5) ml from venous blood sample was collected in heparin container, by using sterile disposable plastic syringes and aseptic condition, vein puncture technique was applied. The sample was centrifuged at (3500 rpm) for (5 minute), and reading.

3.9 Ethical Consideration:

Permission to carry out the study was taken from health administration and Ethical Research Committee and hypertension patients were informed for the purpose of the study before collection of samples and verbal consent was taken.

3.10 Methods:

3.10.1 Estimaton of Plasma Mg^{2+} and Ca^{2+} levels:

i. Principle:

A quantitative method can be used to measuring Ca^{2+} and Mg^{2+} concentration by used semi automation method by used spectrophotometer instrument. It requires standards with known analyte content for both Mg^{2+} and Ca^{2+} to establish the relation between the measured absorbance and the analyte concentration and relies therefore on the Beer-Lambert Law. The measurement of calcium in the sample is based on formation of color complex between calcium and o-cresol phthalein in alkaline medium. The intensity of the color formed is proportional to the calcium concentration in the sample

Magnesium combines with calmagite in an alkaline medium to form a red colored complex. Interference of calcium and protein are eliminated by the addition of specific chelating agents and detergents. Intensity of color formed is directly proportional to the amount of magnesium present in the sample.

3.10.2 Estemation of Na^+ and K^+ Level:

i. Principle:

Its methodology is based on the selective electrode measurement (SLE) principle to precisely determine measurement values.

Each electrode has an ion selective membrane that undergoes a specific reaction with the corresponding ions contained in the sample being analyzed. The membrane is an ion exchanger, relating to the electrical charge of the ion causing a change in the membrane potential or measuring voltage, which is built up in the film, between the sample and the membrane.

3.11. Quality control:

Sample representing the normal and pathological level of serum Na^+, K^+, Mg^{2+} and Ca^{2+}, was used for assessment of the quality control.

Result (±2SD) of the target values of the control sera were accepted.

3.12. Statistical analysis:

Data was analyzed by computer software, by using SPSS program manual master sheet. The mean and standard deviation of Na^+, K^+, Mg^{2+} and Ca^{2+}, level was obtained, and the T- test was used for the comparison of Na^+, K^+, Mg^{2+} and Ca^{2+} between the test and normal range, and the mean difference is significant at ($p \leq 0.05$). Correlation(r) between mean of Na^+, K^+, Mg^{2+} and Ca^{2+} with duration of hypertension and age, gander, education status economic status, social status, jobs of hypertensive patients are considered to be statistically significant at ($P \leq 0.05$).

Chapter Four

Results

4.1 Results:

The associations between hypertension and electrolytes by measuring Sodium, Potassium, Magnesium and Calcium.

The result of Na^+ showed normal concentration with mean (138mmol/l) compared with normal range (135 – 145mmol/I). Result of K^+ showed mean (4.4 mg/dl) which is normal concentration compared with normal range (3.5 – 5.0 mmol/l). Result of Mg^{2+} showed mean (2.2 mg/dl) which is normal concentration compared with normal range (1.7 – 2.7mg/dl), also result of Ca^{2+} showed normal concentration with mean (8.8 mg/dl) compared with normal range (8.5-10.5) mg/dl.

Table (4.1): Mean and Std. Deviation of Na^+, K^+, Mg^{2+}, and Ca^{2+}.

	Na^+	K^+	Mg^{2+}	Ca^{2+}
Mean	138	4.4	2.1	8.8
Std. Deviation	3.37	0.70	0.29	0.36

Table (4.2): Mean and Std Deviation of Na^+ and K^+ with age, gender, duration and social status

		Na^+			K^+		
		Mean	Std. Deviation	P.value	Mean	Std. Deviation	P.value
Age	30-60	138	3.17	0.5	4.4	0.57	0.9
	61-80	137	4.03		4.4	1.04	
Gender	Male	137	3.45	0.5	4.6	0.90	0.2
	Female	138	3.37		4.3	0.53	
Duration	1-10	138	3.47	0.4	4.5	0.75	0.4
	11-20	139	3.16		4.3	0.61	
Married	Married	138	3.27	0.5	4.5	0.74	0.5
	Single	137	3.85		4.3	0.60	

- Non-significant correlations ($P > 0.05$)

Table (4.3): Mean and Std. Deviation of Mg^{2+}, and $Ca^{2+}$with age, gender, duration and social status

		Mg^{2+}			Ca^{2+}		
		Mean	Std. Deviation	P.value	Mean	Std. Deviation	P.value
Age	30-60	2.1	0.30	0.1	8.8	0.35	0.3
	61-80	2.3	0.22		8.7	0.37	
Gender	Male	2.2	0.30	0.5	8.7	0.44	0.7
	Female	2.1	0.28		8.8	0.30	
Duration	1-10	2.1	0.30	0.9	8.7	0.37	0.2
	11-20	2.1	0.29		8.9	0.31	
Married	Married	2.1	0.28	0.3	8.7	0.38	0.4
	Single	2.2	0.32		8.9	0.30	

-The above table denoted that there were no significant correlations (P> 0.05)

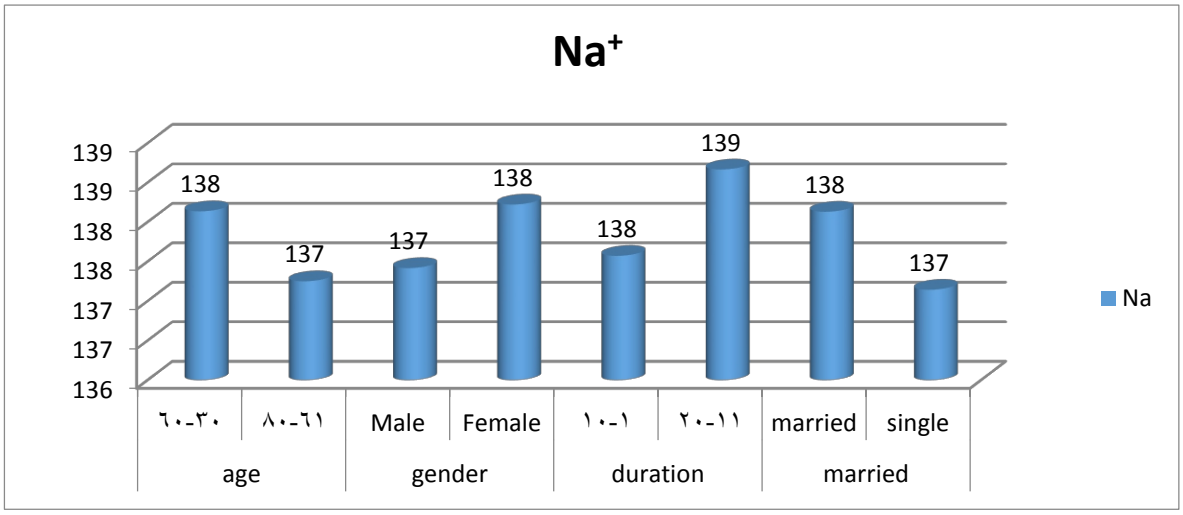

Figure (4.1): Mean and Std. Deviation of Na^+ with age, gender, duration and social status

- The above mentioned figure presented that there was non-significant difference ($p > 0.05$)

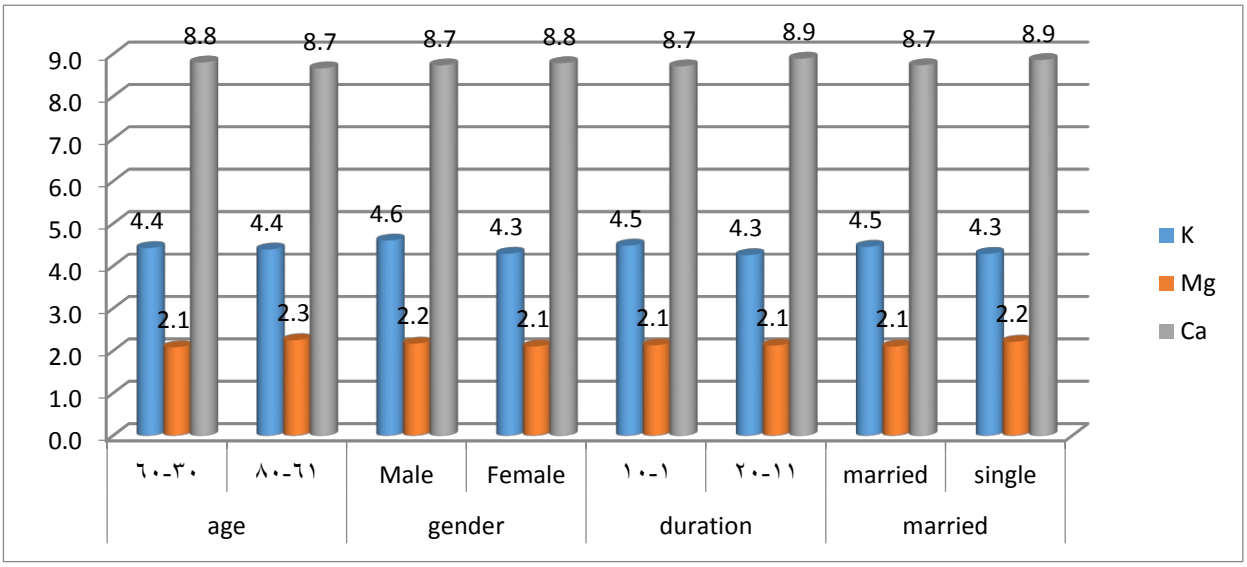

Figure (4.2): Mean and Std. Deviation of K^{+}, Mg^{2+} and $Ca^{2+}$with age, gender, duration and social status.

-non-significance difference ($p > 0.05$).

Table (4.4): Mean and Std. Deviation of Na^+ and K^+ with job

Job	Na^+			K^+		
	Mean	Std. Deviation	P.value	Mean	Std. Deviation	P.value
No job	140	2.22	0.3	3.9	0.26	0.4
Employer	137	3.59		4.5	0.72	
Paterfamilias	140	2.59		4.5	0.15	
Pensionary	137	5.66		4.8	0.92	
Other	137	3.31		4.5	1.05	

-The above table illustrated that there were non-significant correlation ($P > 0.05$)

Table (4.5) : Mean and Std. Deviation of Mg^{2+} andCa^{2+} with job

Job	Mg^{2+}			Ca^{2+}		
	Mean	Std. Deviation	P.value	Mean	Std. Deviation	P.value
No job	2.2	0.39	0.8	9.1	0.21	0.06
Employer	2.2	0.30		8.7	0.36	
Paterfamilias	2.1	0.28		9.0	0.26	
Pensionary	2.1	0.28		8.6	0.21	
Other	2.0	0.28		8.6	0.34	

-The above mentioned table presented that there were non-significant correlation (P-value>0.05)

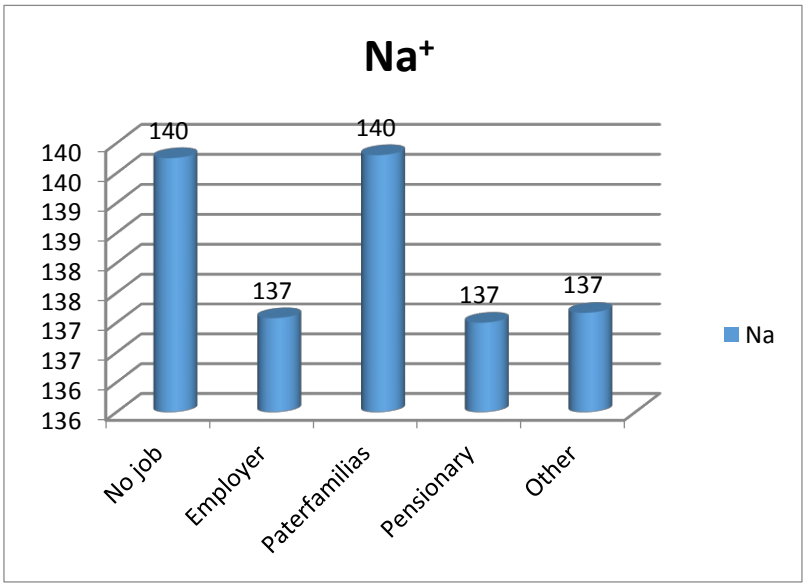

figure (4.3): mean and Std. Deviation of Na^+ with job

-non- significance difference (p> 0.05)

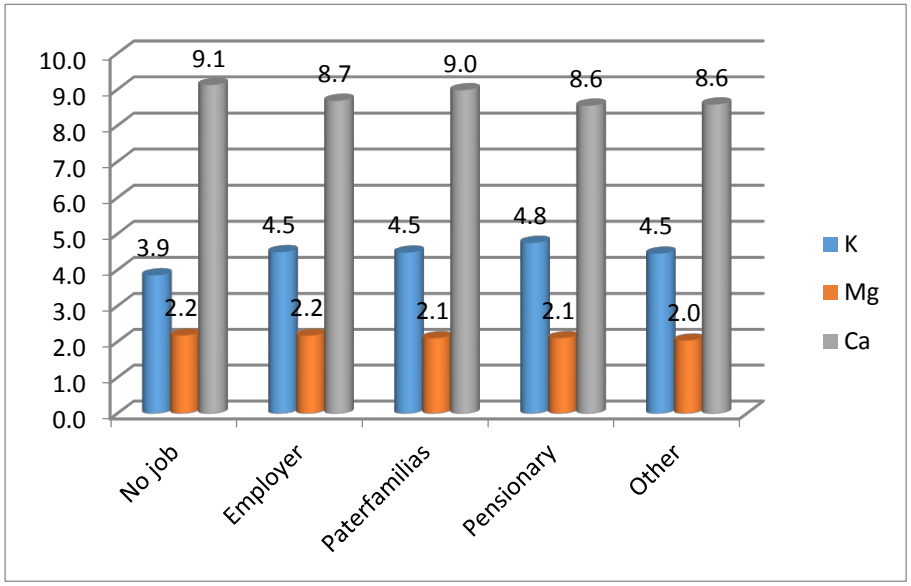

Figure (4.4): Mean and Std. Deviation of, K^{+}, $Mg^{2+}$and $Ca^{2+}$with job

- The above figure denoted that there was non- significance difference ($p > 0.05$).

Table (4.6): Mean and Std. Deviation of Na^+ and K^+, with education and economic statue

		Na^+			K^+		
		Mean	Std. Deviation	P.value	Mean	Std. Deviation	P.value
Education	primary	138	3.27	0.4	4.5	1.07	0.5
	secondary	140	2.39		4.2	0.41	
	university	137	3.89		4.4	0.73	
	post graduate	138	3.73		4.6	0.66	
Economic	low	139	1.83	0.4	4.3	0.47	0.5
	Medium	138	3.57		4.4	0.76	
	High	136	3.63		4.7	0.76	

-The above table indicated that, there were non-significance correlation (P->0.05).

Table (4.7): Mean and Std. Deviation of $Mg^{2+}$and Ca^{2+} with education and economic status

		Mg^{2+}			Ca^{2+}		
		Mean	Std. Deviation	P.value	Mean		P.value
Education	primary	2.0	0.29	0.7	8.9	0.49	0.5
	secondary	2.0	0.25		8.8	0.22	
	university	2.2	0.33		8.8	0.44	
	post graduate	2.2	0.26		8.6	0.26	
Economic	low	1.9	0.16	0.01	8.8	0.24	0.7
	Medium	2.2	0.29		8.8	0.34	
	High	2.3	0.26		8.7	0.53	

-Above table show significance difference with economic status and Mg^{2+} level($p<0.05$)

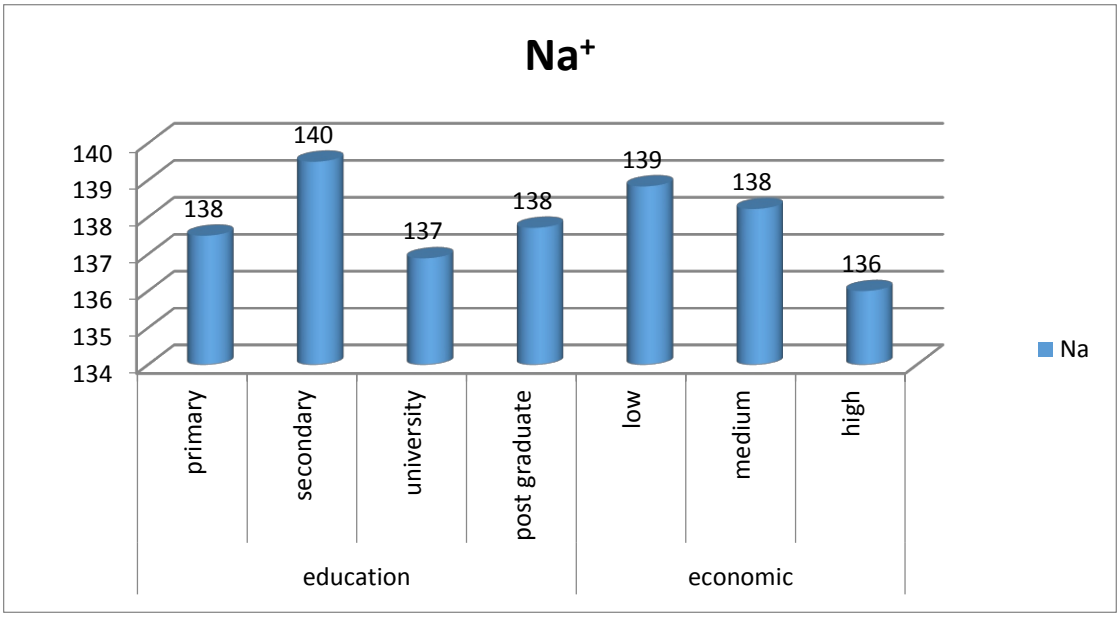

Figure (4.5): Mean and Std. Deviation of Na^+ with education and economic status

- non-significance difference ($p > 0.05$).

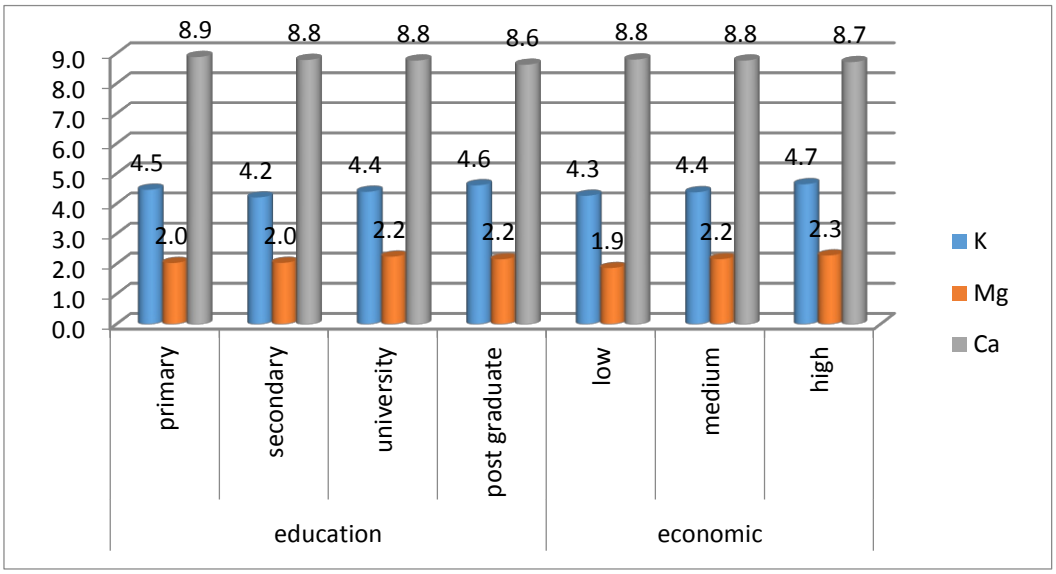

Figure (4.6): Mean and Std. Deviation of K^{+}, Mg^{2+} and Ca^{2+} with education and economic status.

-Above Figure Show non-significance difference between education and serum level of K^{+}, Mg^{2+} and Ca^{2+} ($p > 0.05$).

With economic status show significance difference for Mg^{2+} ($p<0.05$) and non-significance difference for of K^{+} and Ca^{2+} ($p > 0.05$).

Chapter Five

Discussion

Conclusion

Recommendations

5.1 Discussion:

The study covered (30) individuals in Khartoum State with hypertension (19 females, 11male), with age range from (30-80) years, and duration form (1-20) years. The results of Na^+ showed normal concentration with mean (138 mmol/l) compared with normal range (135 – 145 mmol/). Results of K^+ show mean (4.4 mg/dl) which was normal concentration compared with normal range (3.5 – 5.0 mmol/l). Results of Mg^{2+} indicated mean (2.2 mg/dl) which was normal concentration compared with normal range (1.7 – 2.7mg/dl), also results of Ca^{2+} demonstrated normal concentration with mean (8.8 mg/dl) compared with normal range This result was in agreement with other similar study carried in china by (Jiang He and GretheS.Tell *et al*1991) whom were reported non-significant change in serum level of Sodium, magnesium and calcium of tested group with normal range, But was in disagreement with potassium level of tested group whom was found to have a negative significant change.

Also the findings of this study denoted a non-significant difference between the serum levels of sodium, potassium, magnesium and calcium of tested group to age , gender, duration, social status and job ($p > 0.05$) also there was a non-significant correlation between education status and serum level of electrolytes ($P > 0.05$), and non- significant correlation between economic status and serum level of Na^+, K^+, Ca^{2+} ($P > 0.05$), but presented a significant correlation between serum level of Mg^{2+} and economic status ($P<0.05$).

5.2 Conclusion:

The study demonstrated the non-significant correlation between serum electrolyte and hypertension tested group compared with normal range. But prevailed significant correlation with economic status of tested group and level of magnesium serum.

5.3 Recommendations:

1. A study on large sample size should be done to obtain accurate results about serum electrolytes in hypertensive Sudanese male and female**.**
2. Estimation of electrolytes in hypertensive patients should be done regularly as part of care assessment**.**

Chapter Six

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بسم الله الرحمن الرحيم

Shendi University

Faculty of Graduate Studies and Scientific Research

Research questionnaire

Assessment the Associations between serum Electrolytes in Sudanese Hypertensive Peoples in Khartoum State

Questionnaire identification data

01 QUESTIONNAIRE IDENTIFICATION NUMBER |___|___|___|

02 CITY----------

03 HOSPITAL----------

04 INTERVIEWERS: Name_____

05 DATE OF INTERVIEW: __\ ____ \ _____

06 CHECKED BY SUPERVISOR: Signature _____ Date _____

Background characteristics:

Q1. Age ….

Q2. Gander: male ( ) female ( )

Q3. Education: ……..primary () secondary ( ) bachelor ( ) postgraduate()

Q4. Job: ……...student( ) employer ( ) paterfamilias ( ) pensionary ( ) no job ( )

Q5. Marital status: married () single ( )

Q6. Economic status: low ( ) high () medium ( )

Q7. Duration of diseases ……. month\ year

History of other disease: □ Yes □ No If no escape the following questions; but if yes, go to the following questions:

Other disease:

□ Renal disease □ Liver disease □ Thyroid disease

□ Other disease

If there is positive history of any of the above mentioned diseases, we excluded this patient from our study

Measuring blood pressure:

Systolic…. Diastolic ……

Results:

Test	Na^+	K^+	Mg^{2+}	Ca^{2+}
Result				