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Electrolyte Disturbance in Hemorrhagic and non-Hemorrhagic Stroke Patients in Al-Diwaniyah Teaching Hospital.

A Thesis

Submitted to The Council of the College of Medicine / University of Al-Qadisiyah in a partial Fulfillment of the Requirements for the Degree of Higher Diploma Equivalent to Master Degree in Family Medicine.

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بسم الله الرحمن الرحيم

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Supervisor's certification

I certify that this thesis entailed " electrolyte disturbance in hemorrhagic and non-hemorrhagic stroke patients in Al-Diwaniyah Teaching Hospital" was prepared under my supervision in the department of medicine as a partial fulfillments of the degree of Higher Diploma in Family Medicine.

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We , the examiner committee , after reading this dissertation and examining the candidate Zahraa Adel Aryan in its content , found that it meet the standards and requirement as adissertation in post graduate student in University of AL-Qadisiyah in partial fulfillment of higher diploma of family medicine rating.

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Dedication:

To my mother; the candle that lights up my world.

To my husband; for introducing the real support in my study and helped me in every step.

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Abstract

Background:

Among all neurologic diseases of adult, stroke graded first. Although there are many studies on stroke, but few studies on electrolytes disturbance have been done in our country. Electrolyte disturbances are frequently observed in hemorrhagic and non hemorrghic stroke and may potentially worsen outcome and prognosis.

Objective:

Our aim in this study is to investigate the level of serum potassium and sodium in acute stroke patients and their association with severity of acute stroke and the association between risk factors of stroke and type of stroke with comparison to patients with other disease than CVA.

Methods:

This study is a comparative cross-sectional study done to patients on the neurological center suffer from stroke and others in medicine department for any disease other than cerebrovascular disease consider control group. All in AL_Diwaniyh Teaching Hospital from April to July 2018. The level of potassium and sodium from all patients is estimated.

Results:

Diabetes mellitus was seen in 10 (17.5%), 3 (23.1%) and 4 (33.3) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.459). Hypertension was seen in 31 (54.4%), 6 (46.2%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.814). Smoking was seen in 33 (57.9%), 8 (61.5%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.971). Dyslipidemia was seen in 34 (59.6%), 7 (53.8%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.929). Overweight or obesity was seen in 19 (33.3%), 7 5 (38.5%) and 4 (33.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.938).

Family history was seen in 20 (35.1%), 4 (30.8%) and 5 (41.7%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference statistically was not significant (*P*= 0.848). Patients with ischemic stroke were classified into 9 (15.8%), 34 (59.6%) and 14 (24.6%) having Glasgow coma scores of 3-8, 9-12 and 13-15, respectively. Patients with hemorrhagic stroke were classified into 5 (38.5%), 7 (53.8%) and 1 (7.7%) having Glasgow coma scores of 3-8, 9-12 and 13-15, respectively. Patients with TIA were classified into 0 (0.0%), 5 (41.7%) and 7 (58.3%) having Glasgow coma scores of 3-8, 9-12 and 13-15, respectively. Significant difference was seen in distribution of patients according to GCS levels (P = 0.014). Mean serum sodium was significantly lowest in hemorrhagic stroke, then ischemic stroke, followed by TIA and the highest sodium level was seen in control group (P<0.001). The Mean of serum potassium was significantly lowest in hemorrhagic stroke, then ischemic stroke, followed by TIA and the highest sodium level was seen in control group (P<0.001). Mean serum to potassium ratio was significantly highest in hemorrhagic stroke, then TIA, followed by ischemic stroke and lastly by control group (P<0.001). The rate of hyponatremia was 2 (2.2), 20 (35.1), 5 (38.5) and 2 (16.7), in control, ischemic, hemorrhagic and TIA groups, respectively. The rate of hypokalemia was 1 (1.1), 4 (7.0), 2 (15.4) and 0 (0.0) in control, ischemic, hemorrhagic and TIA groups, respectively.

Conclusion:

This study reveals that in haemorrhagic stroke, the incidence of electrolytes imbalance was more than ischaemic and Transient Ischemic attack and which was mostly Hyponatraemia and Hypokalaemia . In our study we found that electrolyte disturbance could effects on the severity of stroke according to Glassgow com scale result. Thus Electrolyte imbalance may adversely affect outcome and prognosis of stroke.

List of abbreviations:

CVA: cerebrovascular accident

TIA: Transient Ischemic attack

ICH: intracranial hemorrhage

DVT: deep venous thrombosis

UTI: urinary tract infection

NCCT: non contrast computed tomography

SAH: subarachnoid hemorrhage

MRI: magnetic resonance imaging

MAP: mean arterial pressure

EVD: external ventricular drain

DMCH: Department of Medicine, Nilratan Sircar Medical College and Hospital.

AIS: acute ischemic stroke

OTT: onset time of treatment

GCS: Glassgow coma scale

RtPA: recombinant tissue plasminogen activatator

INR: international normalization ratio

AHA/ASA: American Heart Association/ American Stroke Association

ICU: intensive care unit

PEEP: positive end expiratory pressure

PIP: peak inspiratory pressure

FiO_2: Fractional inspired oxygen

Chapter one

Introduction

1. INTRODUCTION

1.1. Cerebro-vascular accidents (CVA):

It is a main public health problem. It is well-distributed in the whole world and is assorted to be the second top cause of death in the world. Stroke causes significant impairments particularly in the older age group and are amongst the major health matters in several countries.[1, 2]

"Stroke causes a great influence on disability rate. Stroke also has huge contribution to economic and social load for patients and their family[3,4].
In almost all neurological disorders, electrolyte disturbances are prominent. Electrolyte disturbance are commonly found in acute stroke events[5,6]. "
Recently, research with electrolyte disturbances focusing on risk factors of stroke, its prevalence and association with other medical condition not only on the neuroendocrine mechanism.[7,8] Many Stroke patients die either because of the primary disease or due to its consequences.[9] Management of stroke patient aimed not only on the treatment of the primary disease but also on the avoidance of severe consequences of stroke, including dyselectrolytemia, aspiration pneumonia, malnutrition, pulmonary embolism , DVT, bowel or bladder dysfunction, UTI , contractures, ,skin breakdown and joint abnormalities[10,11]. An early and accurate forecasting of stroke outcome in the emergency department is pivotal for decision-making, as well as in assessing patient's prognosis.[12, 13]

The reports on the association between electrolyte imbalance and severity of acute stroke are still in a limited number, although there are some data about large number of electrolyte disturbances in acute stroke events.[14, 15] There is a deficiency of data about this association especially from the developing countries. Electrolyte disturbances are usually present in acute stroke setting as hypokalemia or

hyponatraemia which is the commonest type of disturbance.[16, 17] Electrolyte disturbances such as hyponatremia resulting from either the syndrome of inappropriate antidiuretic hormone secretion (SIADH), inappropriate fluid intake and loss or high of Brain Natriuretic Peptides (BNP) these can result in consequences such as seizures or death. [18, 19]

The commenst presenting symptoms of patients with hemorrhagic stroke is headache and vomiting.[20]Vomiting also an important factor of electrolyte disturbance. Published reports about stroke suggest that CVD occurs with increasing frequency at all ages and in both sexes.[21,22] Prospective studies on acute stroke found that hypertension, diabetes mellitus, dyslipidemia, obesity, smoking and family history are important risk factors (RFs).[23,24]Regarding our country, many researches about stroke, its complications and their impact on stroke patient‘s prognosis are carried out, but a little studies about electrolytes abnormalities in stroke patients was done and far less in the other countries.[25,26] Therefore , the present study was designed to identify the common electrolyte disturbance in acute phase of multiple types of stroke patients and there relation with some usual clinical presentation & outcome.

Chapter two

Review of Literature

2. Review of Literature

2.1. Blood supply of the Brain

2.1.1. Main vessels**:**

The brain require good oxygen supplementation thus it have complicated network of vessels.

Common carotid arteries represent that major blood supply to brain and have two divisions. The face and scalp are supplied by external carotid artery. While the internal carotid division supply most of the anterior part of the cerebrum. The vertebrobasilar arteries are responsible for the supplying of the posterior two-fifths of the cerebrum, portion of the cerebellum, and the brain stem.[27]

Obstruction of one of the vertebral arteries may result in severe consequences, like the blindness, numbness and paralysis.

2.1.2. Circle of willis**:**

It represents a circle of anastomosing arteries at the base of the brain including the carotid and vertebrobasilar arteries. Also the anterior, middle and posterior cerebral artery arise from the Circle of Willis and supply different parts of the brain. [28]

If occlusion occurs to one of the main arteries in this circle, the other smaller arteries can supply from the other arteries (collateral circulation).

2.1.3. The anterior cerebral artery**:**

Anterior cerebral artery arises from the internal carotid artery and passes upward and forward direction. It supply frontal lobes and parts of the brain that responsible for logic thoughts, personality, and legs movement as a voluntary control.[29]

2.1.4. The Middle Cerebral Artery:

It is considered the biggest branch of the internal carotid. It supply part of frontal lobe and lateral area of temporal and parietal lobes, mainly supply the primary sensory and motor portions of throat, face, arm and hand also dominant hemisphere and speech center.[30]

The middle cerebral artery is main vessel that usually has closure in stroke.[11]

2.1.5. Posterior Cerebral Artery**:**

It has arisen mainly from a basilar artery but in some individuals may arise from the internal carotid artery. It is responsible for the supplying of occipital and temporal lobes in the right and left cerebral hemisphere . Occlusion of the posterior cerebral artery mainly results from embolism of the lower part of the vertebral basilar system or heart.[31]

The occlusion of the posterior cerebral artery results in clinical symptoms that depend up on the site of closure that includes the thalamic syndrome and thalamic perforate syndrome .Contralateral hemplegia, hemianopsia and also other different symptoms, like verbal dyslexia, color blindness and hallucinations. [32]

2.1.6. Lenticulostriate plexus**:**

This include small, deep perforating branching arteries from middle cerebral artery .The closure of those vessels or basilar arteries or penetrating arteries of the Circle of Willis will result in lacunar strokes. [33]

2.1.7. The veins

The brain's venous circulation is different from that of the body. In the brain,this is not the case. Main vein collectors are accumulated into the dura to through the venous sinuses. These receive blood from brain to internal jugular veins. Also superior and inferior sagittal sinuses mainly drain the cerebrum and the cavernous sinuses responsible for drainage of the anterior skull base. Eventually these sinuses drain into sigmoid sinuses, that pass through skull to form jugular veins.[34]

2.1.8. Collaterals:

The collateral circulation of the brain represents a vascular network that control cerebral blood flow when the main supply to brain fails due to occlusion. This anastomotic plexus lead to less-resistance connections that provide reversal of blood flow to assist primary collateral support to the anterior and posterior circulations. The pial plexus of leptomeningeal vessels represent secondary collaterals and it controls the distribution of flow when there is occlusion of an

artery after the circle of Willis. The capacity for collateral blood supply is mainly depend on the number and lumen caliber of the blood vessel that can be different in the leptomeningeal plexus. The collaterals veins help in augment drainage when the main pathways are occluded or when there is venous hypertension.[35]

2.2.1. Terms and definition:

Stroke occurs when the flow of blood to the brain is occluded. Deprivation of oxygen will result in dying of the brain cells after a few minutes. Also sudden bleeding within the brain can result in a stroke.

A stroke is a grave medical emergency that needs early intervention. A stroke can leads to lifelong brain damage, permanent disability, or even death.

Stroke has two types including ischemic stroke and hemorrhagic one .Ischemic is the major type of stroke.

The ischemic one result when there is blockage of the main artery that supplying the brain. Blood clots represent the main cause of the blockages that lead to ischemic strokes.

A hemorrhagic stroke develops if an artery either rupture or leaking blood. So the pressure which results from blood leaking will destruct brain cells.

Transient ischemic attack, (TIA or “mini-stroke”) develops if blood supply to a part of the brain becomes occluded transiently. So the effect on the brain cells not lasting long.[36]

2.3.1. Ischemic Stroke:

Ischemic stroke means that one of the brain arteries is blocked. The oxygen and nutrients are carried by blood to the brain, and replaced by CO2 and cellular out products. When there is blockage of an artery, energy of neurons diminished and stop working gradually. Artery blockage time is critical, when remains for period which may be only few minutes, the brain cells may die. [37]

2.3.2. Ischemic stroke types:

The two main types Ischemic strokes are *thrombotic* and *embolic*.
A thrombotic stroke caused by diseases of cerebral arteries that occlude due to deposition of a blood clot. This clinical condition referred as cerebral thrombosis or cerebral infarction, which is responsible for about 50 % of all strokes. Cerebral thrombosis also has two categories according to the site of the occlusion within the brain: main-vessel thrombosis and small-vessel thrombosis. Main -vessel thrombosis occurs when the occlusion involves one of the brain's major feeding arteries including the carotid or middle cerebral, while the small-vessel thrombosis caused by blockage of the cerebral smaller (one or more), deeper and perforating arteries. Lacunar stroke is the other name of this type of stroke.

Also embolic stroke result from an emboli impacted in the artery, but in this type its emerge away from the brain itself. Usually from the heart, these emboli will become lodged within brain vessels and cannot travel any farther. [38]

2.3.3. Pathophysiology:

An ischemic stroke occurs due to obstruction of vessels due to thromboembolic pathology .Ischemia leading to cell oxygen deprivation and deficient in cellular adenosine triphosphate (ATP). Depletion in ATP, result in decreasing of the energy necessary to control ionic balance through the cell membrane and cell depolarization. Entrance of sodium and calcium ions and spontaneous exchange of water through the cell may explain the cytotoxic edema.

Ischemic core and penumbra

Sudden vascular occlusions cause multiple areas of ischemia in the supplied vascular region.
Ischemic regions where cerebral blood flow is less than 10 mL/100 g of tissue/min are called the core. The death of these cells may occur within minutes of stroke onset.
The regions with low blood flow (less than 25 mL of 100g of tissue/min) are referred as ischemic penumbra. The penumbra's part can stay viable for hours due to minimal tissue perfusion.
Middle cerebral artery (MCA) infarction

The findings in CT scanning (with no contrast) include a massive acute ischemia in the MCA areas including the lateral portions of the left temporal, parietal, and temporal lobes, also the left insular and subinsular parts, with pressure feature and midline deviation to the right. The caudate lobe and area from the

lentiform nucleus with internal capsule will be spared, which supplied blood from the lateral lenticulostriate artery which is a branch of the M1 part of the MCA.

Anterior cerebral artery (ACA) infarction

CT scanning of the left reveals high signal in within frontal and parietal portions. Also there is ischemia of the lateral temporoparietal segments bilaterally, mostly on the left due to multiple vessels infarcted due to emboli.

Posterior cerebral artery (PCA) infarction

The CT images findings (noncontrast) include PCA terriority ischemia include the right occipital and inferomedial temporal lobes. Involvement of the thalamus also can be seen.

Hemorrhagic transformation of ischemic stroke

The hemorrhagic transformation means the transformation of an ischemic infarcted zone to an area of bleeding. Usually it occurs in about 5% of uncomplicated ischemic strokes, where the fibrinolytic agents are absent.[12, 53] The mechanism for this conversion, not fully understood, may include reperfusion of ischemic tissue, including the recanalization of a thrombotic vessel or the supply of the ischemic areas by collateral blood vessels.[7] The blood-brain barrier also will be disrupted, that will result in red blood cells extravasate from area of a weak capillary bed, leading into petechial hemorrhage or a frank intraparenchymal hematoma may result.

Post stroke cerebral edema and seizures

A major clinical cerebral edema may develop post anterior circulation ischemic stroke, but its incidence is uncommon (15-20%). Herniation and edema are the significant causes of mortality in patients with hemispheric stroke.[57, 68] While incidence of seizures range from 3-25% of patients during the early days post ischemic stroke. Chronic seizure disorders may develop in a number of stork's patients.[39]

2.3.4.Risk factors:

These classified as modifiable and non-modifiable factors. Non modifiable risk factors include the following:

- Age
- Race
- Sex
- Ethnicity
- History of migraine headaches

- Fibromuscular dysplasia
- Family history of stroke or transient ischemic attacks (TIAs)

.

Modifiable risk factors include the following [40]

- Hypertension (the most important)
- Diabetes mellitus
- Cardiac disease: including atrial fibrillation, heart failure, and valvular disease, structural deformities that permit right-to-left shunting (like patent foramen ovale).
- Hypertriglycerimia
- Transient ischemic attacks
- Social history of chronic alcohol intake, cigarette smoking, drug addict, low physical activity
- Obesity
- Using of oral contraceptive pills and postmenopausal hormone use
- Sickle cell anemia

2.3.5. The clinical picture of ischemic stroke:

Clinically Stroke syndromes presents as neurologic abnormality of acute onset. Symptoms will represent the infarcted part of brain. The clinical differentiation between ischemic stroke and hemorrhagic one is not sufficiently clear to permit clinical assessment of stroke type. So that, the imaging studies of the brain and neurovascular system in the acute phase is mandatory in all strokes. The left hemisphere stroke will present as aphasia, right hemiparesis and right hemianopia, while in the right hemisphere, left hemiparesis and left hemianopia. Supratentorial stroke (which represent 90% of stroke) symptoms will also include facial droop, drop of arm, speech and time disturbances. While in infratentorial stroke other additional symptoms, including diplopia, bulbar palsies, dysphagia, unilateral dysmetria and incoordination, with decrease in levels of consciousness. Stroke is typically painless. [40]

The typical clinical history of stroke syndrome is early onset of acute neurologic deficit that reaches peak usually in minutes, is regarding a stroke until proven otherwise.

National Institutes of Health Stroke Scale:

National Institutes of Health Stroke Scale used for determining neurologic deficit (Appendix 2). It helps in estimate the suspected location and the severity and of the stroke. It help in determining treatment option and which patient benefit from fibrinolytic drugs and patients who are at greater risk of complications result

by the stroke and the side effects that may associate with the reperfusion therapies. It is easily applicated; and consists of: Conscious level, Visual function, Motor examination, Sensation, Cerebellar function and Language.

It is a 42-score scale. Score of lower than 5 mean patients presented with minor strokes. 80% likelihood of proximal vessel occlusions (as determined by imaging studies) if score more than 10. The determination should be applied in determining the severity of the clinical deficit and the developed disability; as example, the NIHSS score will be 3 if a patient's single finding is mutism or loss of vision,. Additionally, the scale does not asses some deficits that related to the posterior circulation strokes (like vertigo, ataxia). [41]

Middle cerebral artery stroke (MCA):

MCA thrombosis usually produces these features: [42]

- Contralateral hyperesthesia
- Agnosia
- Ipsilateral hemianopsia
- Contralateral hemiparesis
- Gaze directed to the side of the lesion

This artery responsible for supply motor area of the upper limbs. Later on, weakness of the upper limb and face is becoming more severe than that of the lower extremities.

Anterior cerebral artery stroke(ACA):

Occlusions of the anterior cerebral artery early will affect frontal lobe function. Features include the following:

- Impaired judgment
- Disturbed mental status
- Primitive reflexes (like, grasping and sucking reflexes)
- Gait apraxia
- Contralateral weakness (more in legs than arms)
- Urinary incontinence
- Contralateral cortical sensory deficits

Posterior cerebral artery stroke(PCA):

Occlusions of the posterior cerebral artery (PCA) mainly will affect thought and vision. Presentations are as the following:

- Visual agnosia
- Contralateral homonymous hemianopsia
- Memory impairment
- Alteration of the mental status
- Cortical blindness

Occlusions of theVertebrobasilar artery are usually difficult to localize due to a wide features of brainstem, cranial nerve and cerebellar deficits and usually not conclusive. They include the following: [43]

- Vertigo
- Visual field deficits
- Nystagmus
- Dysarthria
- Diplopia
- Ataxia
- Facial hyperesthesia
- Syncope
- Dysphagia

The significant feature of the posterior circulation CVA is the developing of apposed findings: contralateral motor deficits and ipsilateral cranial nerve deficits. While only unilateral findings in the anterior stroke will be occur.

Lacunar stroke:

A lacunar stroke occurs due to the obstruction of the small, penetrating arteries in the deep subcortical regions of the brain. Size of infarcted areas is usually between 2-20 mm. Features of lacunar strokes include motor, mainly sensory and hemiparetic ataxic strokes. However, Lacunar infarcts usually not result into deficit of memory, speech, or conscious level this because of the small size and well-localized subcortical location. [44]

2.4.1.Hemorrhagic stroke:

Spontaneous intracerebral bleeding, or nontraumatic primary hemorrhage within the brain tissue, represents 10% to 15% of strokes cases in the United States.

2.4.2Risk factors:

1-genetics: the presence of an apolipoprotein E2 or E4 allele and ICH in the first-degree relative.

2- Increasing age.

3- Racial.

4-History of hypertension.

5-Tobacco smoking.

6-Chronic alcohol use: This risk appears to be dose-dependent.

7- Levels of cholesterol: where decrease levels of serum cholesterol regarding as a risk factors for ICH while ischemic stroke in which high cholesterol levels are a risk).

8- Abusing drugs: including sympathomimetic drugs, like cocaine, are risk factors for ICH.

The modifiable risk factors when change can result in decreasing an individual's risk of ICH. Multiple trials showing that lowering blood pressure can help in reducing the suspicion of ICH in patients with amyloid angiopathy and can have beneficial effect against ICH resulting from other causes

Primary ICH in many cases are the result as a type of severe small vessel disease including hypertensive vasculopathy and cerebral amyloid angiopathy (CAA) .In hypertensive vasculopathy is caused by longstanding hypertension leading to lipohyalinosis of minimum, deep perforating arteries.

CAA meaning amyloid deposition in the wall of brain vessel involving capillaries, arterioles, and small to medium arteries. Rupture of vessel, occurs as result of aneurysmal dilatation or dissection of the small arteries that result in injury by multiple mechanisms: [48]

1- Mass effect by the hematoma itself.

2- Activation of the coagulation cascade that causing activity of inflammatory cytokines, and damage to the blood-brain barrier

3- Continuous bleeding or hematoma expansion.

- **2.4.3.Pathophysiology:**
- Primary ICH is usually the result of small vessel pathology. The chronic hypertension will cause hypertensive vasculopathy that resulting in microscopic degenerative destruction at the levels of small-to-medium penetrating vessels; this is called as the lipohyalinosis.
- In cases of CAA, there is precipitation of amyloid-beta peptide (Aβ) in the small meningeal and cortical vessels walls.

- After the occurrence of vessel damage, the resulting hematoma leading to traumatic injury to the cerebral parenchyma. Development of edema at periphery during the first 3 hours from symptom development and reaching to peak within 10 to 20 days. [49]

- **2.4.4.Diagnosis:**
  - The definitive diagnosis of ICH is questionable. Early clinical symptoms may include sudden severe headache, vomiting, fit, and other focal or widespread neurologic symptoms. The distinguishing from ischemic stroke is difficult without neuroimaging. CT scan of the brain is the initial study in many centers for those with acute neurologic symptoms.
  - Etiologies of secondary ICH including aneurysm, arteriovenous malformation, masses, or hemorrhagic transformation of ischemic stroke. The points that raising the suspicion for secondary ICH includes lobar ICH, ventricular blood, and young age patents
  - Other imaging modalities that can help in detecting acute ICH:

1-CT angiography

CT angiography (CTA) can provides very good images of the larger arterial vessels.CTA also help to define other secondary causes.

2-Magnetic resonance scanning

MRI can diagnose the causing pathology such as tumor, and can give a better imaging for diagnosing perihematomal edema.

3-MR angiography

MR angiography (MRA) also helps in detecting vascular abnormalities like the aneurysm and arteriovenous malformation.

4-Digital subtraction angiography

Digital subtraction angiography (DSA) remains the main diagnostic tool to exclude other vascular abnormality in many centers.

2.4.5. Treatment:

- The admission may provide improvement to a specialized stroke unit. In critically ill stroke patients' long time admission at the emergency wards before transfer to ICU may suspect bad outcomes. The increased incidence of admission mortality has been shown in ICH patients. Delay in resuscitation predicts worse outcomes, even in patients with well cardiopulmonary function or those not in need of defibrillation; the more aggressive care generally will improves prognosis. [50]

Transiet Ischemic Attack:

A transient ischemic attack (TIA) is like a stroke, producing similar symptoms, but usually lasting only a few minutes and causing no permanent damage.

Often called a ministroke, a transient ischemic attack may be a warning. About 1 in 3 people who have a transient ischemic attack will eventually have a stroke, with about half occurring within a year after the transient ischemic attack.

A transient ischemic attack can serve as both a warning and an opportunity — a warning of an impending stroke and an opportunity to take steps to prevent it.

Symptoms

Transient ischemic attacks usually last a few minutes. Most signs and symptoms disappear within an hour. The signs and symptoms of a TIA resemble those found early in a stroke and may include sudden onset of:

- Weakness, numbness or paralysis in your face, arm or leg, typically on one side of your body
- Slurred or garbled speech or difficulty understanding others
- Blindness in one or both eyes or double vision

- Dizziness or loss of balance or coordination
- Sudden, severe headache with no known cause

Risk factors

Some risk factors for a transient ischemic attack and stroke can't be changed. Others you can control.

Risk factors you can't change

You can't change the following risk factors for a transient ischemic attack and stroke. But knowing you're at risk can motivate you to change your lifestyle to reduce other risks.

- **Family history.** Your risk may be greater if one of your family members has had a TIA or a stroke.
- **Age.** Your risk increases as you get older, especially after age 55.
- **Sex.** Men have a slightly higher likelihood of a TIA and a stroke, but more than half of deaths from strokes occur in women.
- **Prior transient ischemic attack.** If you've had one or more TIAs, you're 10 times more likely to have a stroke.
- **Sickle cell disease.** Also called sickle cell anemia, a stroke is a frequent complication of this inherited disorder. Sickle-shaped blood cells carry less oxygen and also tend to get stuck in artery walls, hampering blood flow to the brain. However, with proper treatment for sickle cell disease, you can lower your risk of a stroke.
- **Race.** Black people are at greater risk of dying of a stroke, partly because of the higher prevalence of high blood pressure and diabetes among blacks.
- **Regarding electrolytes:** Sodium regarding as the main cation within extracellular fluid, its main function includes the control and optimizes the osmotic pressure, fluid balance and the permeability of cellular membrane. Regarding potassium is the major cation of intracellular fluid its chief role is the maintaining of water electrolyte balance and osmotic pressure.

Chapter three

Material & Method

3. Material&Methods:

3.1. Study design &setting:

It is comparative cross-sectional study done on patients admitted to neurological center from April to July 2018 (122days) in comparison to patients admitted to medicine department for any cause other than CVA in the same hospital and at the same period.

3.2. The study population:

In this study the selection of a representative case group from patients with acute stroke events including Ischemic CVA, Hemorrhagic CVA and Transient ischemic attack (TIA). Stroke was diagnosed by clinical presentations and imaging studies such as CT scan of the brain.

The control group consisted of ninety patients, from the medicine departments for any cause other than CVA.

All stroke patients were treated in neurological center. Informations collected for this study were taken from meeting .Medical data was obtained from medical records. Patients with disturbed level of consciousness, the data collected from their next of kin. Diagnosis of stroke confirmed by clinical history, physical and neurological examination and documented by brain imaging studies. We measure serum potassium and sodium for all patients.

The level of consciousness assessment for patients with stroke included in the present study was performed according to Glasgow coma scale. The GCS includes three parts: eye opening, verbal and motor response .Each one have score according to patient condition. The summation of the score in each one of the 3 parts give assessment of patient condition, with a maximum score of 15 and a minimum score of 3.

3.3.Inclusion criteria:

Patients of either sex and age with acute stroke admitted within 48 hours of onset & fulfilling WHO definition of stroke and confirmation of stroke with imaging studies of the brain. No history of endocrine or kidney diseases, Patient was not in resuscitation phase and Patients are not in diuretic therapy.

3.4. Exclusion criteria:

Any patients have complication that could affect electrolyte level excluded from the study also if Patients have any neurological deficit secondary to head injuries, Subdural Hemorrhage (SDH) , Epidural Hemorrhage or an infarction which is caused by an infection/tumor (SOL) etc. also any patients having preexisting severe physical or cognitive disabilities.

3.5. The study tools:

3.5.1. The questionnaire:

Special form of questionnaire was planned to collect information from the patients and their families and also medical information taken from medical records . The questionnaire consists of:

SECTION 1: Personal history in term of patients: name, age, occupation, residency, marital status, next of kin and date of admission. This is for control and study groups.

SECTION 2: history of present illness, type of stroke, number of attack, past medical history, family history, drug history, smoking and alcohol history. This is for control and study groups.

SECTION3: level of consciousness according to Glasgow coma scale, degree of paralysis, speech disorder and sensory disturbance.

SECTION4: level of sodium and potassium, lipid profile and any other investigations done for patient in medical record. This is for control and study groups. These parameters were measured using photoelectric flame photometer (measuring Na and K).

3.5.2. Definition of variables:

***AGE:** any age

***OCCUPATION:** worker, Jobless

***RESIDENCY:** rural and urban.

***MARITAL STATUS:** married, single, Divorced, Widowed.

***HX OF PRESENT ILLNESS:** chief complaint and its duration.

***TYPE OF STROKE:** ischemic, hemorrghic and TIA.

***PAST MEDICAL HX:** DM, HT, Dyslipidemia.

***FAMILY HX:** if first degree relative have stroke or not and any chronic disease in the family.

***DRUG HX:** any chronic use of medication.

***TOBACCO HX:** active smoker or in the past.

***OVER WEIGHT OR OBESITY:** this done according to waist circumference (abdominal obesity) because limitation in movement for most patient with stroke.The measurement of abdominal obesity is either by waist circumference or waist-to-hip ratio and its sensitive ratio for adiposity and vascular risk. "Adiposity" means the amount of fat in the body, and its regarding as a precise mean than "obesity". Waist circumference > 102 cm in men and 88 in women is regarding as abdominal obesity)

***LEVEL OF CONSCIOUS:** according to scores of Glassgow coma scale13-15 mild, 9-12 moderate, 3-8 severe.

***LEVEL OF SODIUM:** 135-153 Meq/Lnormal, hyponatremia less than 135 Meq/L, Hypernatremia more than 153 Meq/.

*__LEVEL OF POTASSIUM:__ 3.5-5 Meq/L normal, below 3.5 Meq/LHYPOKALEMIA, above 5 Meq/Lhyperkalemia.

*__LIPID PROFILE:__ if patient had dyslipidemia previously and take treatment consider had abnormal lipid profile if not send for new one.

3.6. Statistical analysis:

Data were collected, summarized, analyzed and presented using two software programs; these were the statistical package for social sciences (SPSS)version 23 and Microsoft office excel2013.Numeric variables were presented as mean, standard deviation(SD) and range, where is categorical variables were expressed as number and percentage. Association between categorical variables was assessed using Chi-Square test. Comparison of mean values between groups was done using one way analysis of variance (ANOVA).The level of significance was considered at $P \leq 0.05$. Ethical consideration was obtained from Research Committee of AL-Diwaniyah Teaching Hospital.

Chapter four

Results

4. Results

4.1 Demographic characteristics of patients enrolled in the present study

4.1.1. Mean age and age range of control and study groups

The current study included 90 control subjects with a mean age of 57.92 ±5.79 years and an age range of 46 -68 years, 57 patients with ischemic stroke having a mean age of 57.30 ±5.64 years and an age range of 51 -67 years, 13 patients with hemorrhagic stroke having a mean age of 65.38 ±4.31 years and an age range of 60 -73 years and 12 patients with transient ischemic attack having a mean age of 60.50 ±3.61years and an age range of 55 -66 years. There was no significant difference in mean age among control subjects, patients with ischemic stroke and patients with TIA (P>0.05); however, patients with hemorrhagic stroke were significantly older than the rest of patients and also of control subjects (P<0.001), as shown in table 3-1.

Table 4-1: Mean age and age range in control and study groups

Group	***n***	**Mean age ± SD**	**Range (Min.-Max.)**	***P***
Control	90	57.92 ±5.79 B	22 (46 -68)	<0.001* HS
Ischemic	57	57.30 ±5.64 B	16 (51 -67)	
Hemorrhagic	13	65.38 ±4.31 A	13 (60 -73)	
TIA	12	60.50 ±3.61 B	11 (55 -66)	

n: number of cases; SD: standard deviation; min.: minimum; max.: maximum; TIA: transient ischemic attack; HS: highly significant difference; *: one way ANOVA; capital letters were used to indicate significant difference following post hoc LSD test; different letters indicate signficant5 difference at P≤0.05; similar letters indicate no significant difference at P≤0.05.

4.1.2 Distribution of patients and control subjects according to gender

The current study included 35 (61.4%) men and 33 (36.7%) women serving as control subjects. Ischemic stroke was represented by 35 (61.4%) men and 22 (38.6%) women, hemorrhagic stroke was represented by 9 (69.2%) men and 4 (30.8%) women, whereas, TIA was represented by 8 (66.7%) men and 4 (33.3%) women. There was no significant difference among control and study groups with respect to gender (*P*=0.952), as shown in table (3-2).

Table 4-2: Distribution of patients and control subjects according to gender

Gender	Control *n* (%)	Ischemic *n* (%)	Hemorrhagic *n* (%)	TIA *n* (%)	χ^2	P
Male	57 (63.3)	35 (61.4)	9 (69.2)	8 (66.7)	0.344	0.952 NS
Female	33 (36.7)	22 (38.6)	4 (30.8)	4 (33.3)		
Total	90 (100.0)	57 (100.0)	13 (100.0)	12 (100.0)		

n: number of cases; TIA: transient ischemic attack; $\chi^{2:}$ Chi-square statistic; NS: not significant

4.1.3 Distribution of control and study groups according to residency and occupation

According to residency, patients with ischemic stroke included 41 (71.9%) patients who were of urban residency and 16 (28.1%) patients of rural residency. Patients with hemorrhagic stroke included 7 (53.8%) urban residency and 6 (46.2%) patients of rural residency. Patients with TIA were distributed as following: 8 (66.7%) of urban residency and 4 (33.3%) of rural residency. No significant difference was encountered, with respect to residency among all stroke patients (*P*=0.446), as shown in table 3-3.

Patients with ischemic stroke were either employed 33 patients (57.9%), or unemployed, 24 patients (42.1%). Hemorrhagic stroke was seen in 7 employed

(53.8%) and in 6 unemployed or retired (46.2%), whereas, TIA was seen in 5 employed (41.7%) and in 7 (58.3%) unemployed or retired. There was also no significant difference in distribution of patients with respect of occupation (*P*=0.588).

Table 4-3: Residency and occupation of patients according to type of stroke

Characteristic		**Ischemic *n*= 57**	**Hemorrhagic *n*=13**	**TIA *n*=12**	χ^2	***P***
Residency	Urban; *n* (%)	41 (71.9)	7 (53.8)	8 (66.7)	1.616	0.446 NS
	Rural; *n* (%)	16 (28.1)	6 (46.2)	4 (33.3)		
Occupation	Employed; *n* (%)	33 (57.9)	7 (53.8)	5 (41.7)	1.061	0.588 NS
	Retired or unemployed; *n* (%)	24 (42.1)	6 (46.2)	7 (58.3)		

n: number of cases; TIA: transient ischemic attack; $\chi^{2:}$ Chi-square statistic; NS: not significant

4.2 Risk factors of stroke in study group

Diabetes mellitus was seen in 10 (17.5%), 3 (23.1%) and 4 (33.3) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.459). Hypertension was seen in 31 (54.4%), 6 (46.2%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.814). Smoking was seen in 33 (57.9%), 8 (61.5%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.971). Dyslipidemia was seen in 34 (59.6%), 7 (53.8%) and 7 (58.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.929). Overweight or obesity was seen in 19 (33.3%),

7 5 (38.5%) and 4 (33.3%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.938). Family history was seen in 20 (35.1%), 4 (30.8%) and 5 (41.7%) patients with ischemic stroke, hemorrhagic stroke and TIA patients, respectively and the difference was not statistically significant (*P*= 0.848), table 3-4.

Table 4-4: Risk factors according to type of stroke

Risk factor	**Ischemic *n*= 57**	**Hemorrhagic *n*=13**	**TIA *n*=12**	χ^2	***P***
DM; *n* (%)	10 (17.5)	3 (23.1)	4 (33.3)	1.556	0.459 NS
HT; n (%)	31 (54.4)	6 (46.2)	7 (58.3)	0.412	0.814 NS
Smoking; n (%)	33 (57.9)	8 (61.5)	7 (58.3)	0.058	0.971 NS
Dyslipidemia; n (%)	34 (59.6)	7 (53.8)	7 (58.3)	0.146	0.929 NS
Overweight or obesity; n (%)	19 (33.3)	5 (38.5)	4 (33.3)	0.128	0.938 NS
Family history; n (%)	20 (35.1)	4 (30.8)	5 (41.7)	0.331	0.848 NS

n: number of cases; TIA: transient ischemic attack; $\chi^{2:}$ Chi-square statistic; NS: not significant

4.3 Glasgow coma scale of patients within 24 hours of admission

Assessment of the level of consciousness of patients enrolled in the present study was done according to Glasgow coma scale. Patients with ischemic stroke were classified into 9 (15.8%), 34 (59.6%) and 14 (24.6%) having Glasgow coma scores of 3-8, 9-12 and 13-15, respectively. Patients with hemorrhagic stroke were classified into 5 (38.5%), 7 (53.8%) and 1 (7.7%) having Glasgow coma scores of 3-8, 9-12 and 13-15, respectively. Patients with TIA were classified into 0 (0.0%), 5 (41.7%) and 7 (58.3%) having Glasgow coma scores of 3-8, 9-12 and 13-15,

respectively. Significant difference was seen in distribution of patients according to GCS levels (as mild, moderate and severe impairment of consciousness) (P = 0.014). Median GCS score was significantly highest in patients with TIA, median and inter-quartile range of 13 (3), followed by ischemic stroke, median and inter-quartile range of 11 (3.5) and lastly by hemorrhagic stroke, median and inter-quartile range of 9 (3) as shown in table 3-5. Correlations between serum sodium, serum potassium and serum sodium to potassium ratios with clinical parameters are shown in table 3-6.

Table 4-5: Glasgow coma scale of patients according to type of stroke

GCS	Ischemic *n*= 57	Hemorrhagic *n*=13	TIA *n*=12	χ^2	*P*
3-8 severe	9 (15.8)	5 (38.5)	0 (0.0)	12.495	0.014* S
9-12 moderate	34 (59.6)	7 (53.8)	5 (41.7)		
13-15 mild	14 (24.6)	1 (7.7)	7 (58.3)		
Median (IQR)	11 (3.5) B	9 (3) C	13 (3) A	----	0.001† HS

GCS: Glasgow coma scale; *n*: number of cases; TIA: transient ischemic attack; $\chi^{2:}$ Chi-square statistic; NS: not significant; IQR: inter-quartile range;*: Chi-square test; †: Kruskal Wallis test; S: significant; HS: highly significant; capital letters were used to indicate significant difference following Mann Whitney U test; different letters indicate signficant5 difference at P≤0.05; similar letters indicate no significant difference at P≤0.05.

4.4 Serum sodium and potassium in control and study groups

Mean serum sodium and mean serum potassium in control and study groups are shown in table 3-6. Mean serum sodium was significantly lowest in hemorrhagic stroke, then ischemic stroke, followed by TIA and the highest sodium level was seen in control group (P<0.001). Mean serum potassium was significantly lowest in hemorrhagic stroke, then ischemic stroke, followed by TIA and the highest potassium level was seen in control group (P<0.001). Mean serum

sodium to potassium ratio was significantly highest in hemorrhagic stroke, then TIA, followed by ischemic stroke and lastly by control group (P<0.001). The rate of hyponatremia was 2 (2.2), 20 (35.1), 5 (38.5) and 2 (16.7), in control, ischemic, hemorrhagic and TIA groups, respectively. The rate of hypokalemia was 1 (1.1), 4 (7.0), 2 (15.4) and 0 (0.0) in control, ischemic, hemorrhagic and TIA groups, respectively, as shown in table 3-6.

Table 4-6: Serum sodium and potassium in control and study groups

Characteristic	**Control *n*= 90**	**Ischemic *n*= 57**	**Hemorrhagic *n*=13**	**TIA *n*=12**	***P*** †
Serum sodium	141.23 ±4.53 A	137.67 ±4.92 C	137.00 ±5.37 C	139.92±5.18 B	<0.001
Serum potassium	4.58 ±0.80 A	3.92 ±0.33 B	3.48 ±0.52 C	4.01±0.36 A	<0.001
Na/K ratio	31.06 ±5.54 C	35.12 ±2.92 B	40.20 ±6.59 A	35.23 ±4.21 B	<0.001
Hyponatremia	2 (2.2)	20 (35.1)	5 (38.5)	2 (16.7)	----
Hypoklemia	1 (1.1)	4 (7.0)	2 (15.4)	0 (0.0)	----

Table 4-7: Correlation between serum electrolytes (serum sodium and potassium) and clinical characteristics

Characteristic	Serum sodium		Serum potassium		Na/K ratio	
	r	***P***	**r**	***P***	**r**	***P***
Stroke type	0.206	0.064	-0.055	0.625	0.150	0.180
Age	-0.197	0.077	-0.328	0.003 *	0.301	0.006 *
Gender	-0.144	0.196	-0.214	0.053	0.110	0.326
Diabetes	0.170	0.126	-0.045	0.688	0.099	0.377
Hypertension	0.234	0.035 *	0.215	0.053	-0.131	0.241
Smoking	-0.085	0.448	-0.026	0.818	-0.002	0.986
Dyslipidemia	-0.012	0.918	-0.087	0.435	0.067	0.548
Overweight or obesity	-0.150	0.180	-0.088	0.432	0.030	0.789
Family history	0.092	0.409	0.103	0.356	-0.064	0.570
Residency	0.010	0.929	0.016	0.885	-0.019	0.865
Occupation	-0.049	0.659	0.095	0.396	-0.104	0.352
Glasgow coma scale	0.148	0.186	0.236	0.033 *	-0.181	0.104

r: correlation coefficient; *: significant at $P \leq 0.05$.

Chapter five

Discussion

5. Discussion:

The study was done in the Department of neurological medicine at Al Diwaniyah teaching hospital .Ninety control subject, 82 patients of stroke were selected from patients whom satisfying the inclusion criteria for this study through simple random selection. The findings and the derived and concluded data of this study are discussed below. In this present study and according to imaging studies there is 57 patients with ischaemic stroke, 13 with intracerebral hemorrhage and 12 with TIA.

The current study showing that hemorrhagic Stroke is most commonly occurred at the older age patients (significant difference with the rest of patients and control subject $P<0.001$) while Ischemic and TIA mainly occur at middle age group and no significant difference in mean age with the control subject ($P>0.05$).

According to study in Manado General Hospital Stroke is more commonly occurred in the middle age group and above also more in male than female. In this study we found that there is no significant difference between male and female among control and study groups ($P=0.952$).[51] In a community-based cross-sectional study in Bao'an district, Shenzhen, China ,there is more predominance of stroke in males than between females. The same results have been found in Saudi Arabia, and other countries, like in German, the United Kingdom, the US, Italy, Spain. [52] The multivariable studies indicated that gender was a significant factor in stroke; men have two times more risk to develop stroke than women. The best and significant explanation may be related to genetic factors' difference. Other possible explain may be due to the protective role of estrogen on the brain circulation[27]. In males, a high risk of hypertension, ischemic heart disease, and cigarettes smoking, all have been improved to increase the risk of cerebrovascular disease[53]. These findings make the males more in need for stroke prevention, also in need for more researching to know the biological mechanisms of stroke are affected by gender.

Our study revealed that residency is not a risk factor for CVA because no significant difference was encountered among all stroke patients ($p=0.588$). Also in this study there is no significant difference according to distribution of patients regarding their occupation ($P=0.588$).

The risk factors of stroke in study group have the same effect for all types of stroke because the difference in distribution among ischemic, hemorrhagic and TIA was statistically not significant.

The level of consciousness assessment for patients included in the present study was performed according to Glasgow coma scale.

The present study showed a significant difference in the distribution of patients according to GCS levels (as mild, moderate and severe disturbances of consciousness) (P = 0.014) .We detect that patients presenting with hemorrhagic stroke had a conscious level (median was 9) in comparative with ischemic and TIA (median 11, 13) respectively. These findings may help in the determining the stroke's type because Subarachnoid hemorrhage is usually associated with significant increase in intracranial pressure and lowering in cerebral perfusion pressure this causing intermittent reduction of cerebral blood flow thus the main presenting symptoms of subarachnoid hemorrhage (SAH) is loss of consciousness (LOC) that mean sever GCS.[54]

Distinguishing between ischemic and hemorrhagic stroke not depend on symptoms alone which are not enough specific. Any way generalized symptoms, like nausea, vomiting, and headache, in addition to altered level of consciousness, may give a hint to increasing intracranial pressure and usually found more in hemorrhagic strokes and large ischemic strokes.[55]

Disturbance of the normal values of sodium and potassium will control the fluid and electrolyte balance, edema and may result in disturbing the normal physiological function and the neurotransmission of the brain in the form of collection of fluid and exchange of nutrients like lactic acid and arachidonic acid which have mean role in the development of neurological signs and symptoms. The damage that results due to these consequences will result either in decreased cerebral blood flow or rupture of small vessels due to pressure imbalance. Study by Alam M N et al6 revealed that hyponatermia is the main electrolytes disturbances in cerebrovascular accidents, both in ischemic and hemorrhagic stroke. Other study by Kusuda K et al7 also showed hyponatremia, hypernatremia, hypokalemia and hyperkalemia in CVA. Study also stated that hypernatremia is more common in hemorrhagic stroke and 57% of patients with hypernatremia in his study end by dead within one month of hospital admission indicating the importance of electrolyte disturbances in these patients. [56]

In our study, we found that the hyponatremia and hypokalemia are more common in hemorrghic stroke followed by ischemic stroke then TIA and the highest level in control group. Low sodium level was mainly detected among patients with hemorrhagic stroke followed by ischaemic stroke patients and there is statistical significant association between hyponatremia and type of stroke. Many studies and researchers find that hyponatremia in acute CVA patient may affect the prognosis and severity of stroke negatively. Hypokalemia was more common within hemorrhagic stroke patients followed by ischaemic stroke patients.

In this study the dyselectrolytemia is more common in hemorrhagic stroke. As hyponatremia 38.5%

The main causes of mortality and morbidity following hemorrghic stroke may be divided into two categories: Neurologic and nonneurologic. Rebleeding, vasospasm, hydrocephalus, electrolyte disturbances, aspiration pneumonia, and venous thromboembolism are the main nonneurologic causes. The mortality and morbidity rate are higher with increasing age and about one-third of the survivors suffering from major neurologic affections.[57]

McGirt *et al*. reveal that an increase in the serum levels of cerebral natriuretic peptide (BNP) was independently associated with hyponatremia, and the occurrence of hyponatremia rising significantly within 24 h after the onset of delayed ischemic neurologic deficits. Hypokalemia occurring in haemorrhagic stroke patients more than ischaemic stroke patients in comparison to control group.[58]

Electrolyte disturbance have an effect on clinical characteristic revealed that there is negative relationship between age and serum potassium, when age increase serum potassium decrease, this may be the result of multiple causes. The normal physiological changes related to the aging affect the potassium normal values in the body. Also when the function of kidneys decrease, this will effect on the urine output and cause disturbance in the mechanisms that maintain reabsorption and excretion of nutrients. Thus high level of potassium will be execreated in urine. While at the level of gastrointestinal system, vomiting and diarrhea can affect the proper absorption of dietary potassium. Other medical problems such as Crohn's or Cushing’s disease, leukemia or magnesium deficiency cause large loss of

potassium. Some drugs like the diuretics, insulin, steroid, laxatives, antibiotics also theophyllines may change the normal absorption of potassium.

Notably, when there is increase in the levels of body sodium, blood pressure will increase. Renin-angiotensin system is affected by high salt diet. Damage to endothelial had a significant role in the effect of high sodium intake on blood pressure, although the exact mechanisms remain unknown. High blood pressure represents the major factor in the cardiovascular disease (such as stroke, cardiac failure, and coronary artery disease).[59]

Glasgow coma scale increase when there is an increase in serum potassium. Many studies detect that when there is serum potassium increase there is significant improvement in Glasgow coma scale.

Potassium concentration through membrane of cell has a significant effect in the membrane potential, so that when there is abnormal potassium level this may affect membrane potential in neuronal, cardiac and vascular tissues.[60] A study from Cheng et al. study show low potassium level can reduce the conductance hyperpolarization in potassium channel of skeletal muscle tissue. Severe muscle dysfunction, palpitations, cardiac dysrhythmias, and abnormality in neurological function may occur if there are few disturbances in potassium level . Potassium intake can lower the stroke risk and this is showed by multiple meta-analyses studies and the suggested mechanism could be that K^+ decreases the formation of free radicals and increase endothelial dysfunction . Gariballa et al. showed that low potassium level at admission may result in a three-month mortality rate of acute ischemic stroke while study from Fofi et al. revealed that the relationship between mortality and the serum potassium level not significant [61]. These differences may be due to discrepancy between the two studies in the population features. The study by Fofi et al. was focusing on AIS patients with an OTT of less than 6 h while Gariballa et al. included patients with multiple types of stroke. Other groups revealed that K^+ can suppress vascular smooth muscle cell proliferation [61]. A study performed at the First Affiliated Hospital of Xi'an Jiaotong University Department of Neurology reveal that serum potassium level less than 3.7 mmol/l on admission is an indicator of poor outcome for 3 months after stroke following acute ischemic stroke event.Thus hypokalemia is a significant factor for the worse outcome. So acute ischemic stroke patients should be monitored because they are at higher risk

of a poor prognosis by the effect of the serum potassium level at time of admission.[62]These findings will help physicians who usually monitor serum potassium with acute ischemic stroke and assess its level above 3.7 mmol/l. Because Mg^{2+} can improve the Na^{+}/K^{+} pump function so any disturbance in the serum Mg^{2}levels in patients with severe low potassium level should replace with Mg^{2+} inspite of the serum Mg^{2+} being normal.[19]Finally the limitations of this study was the sample size of this study was relatively small and there was no long-term follow-up of patient.

Conclusions&Recommendation

Conclusions:

1. This study showed that electrolyte imbalances are common problem. Hyponatremia & hypokalemia are the main electrolyte disturbances in both ischemic &haemorrhagic stroke.

2.The study also revealed that there was ultimate relation between hyponatremia or hypokalemia & stroke outcome. Electrolyte abnormalities may have an adverse impact on stroke outcome.

3. Regarding Glasgow coma scale outcome we found patients with normal electrolyte level at admission had better outcome and patient with dyselectrolymia on admission had worse outcome and it was statistically significant.

Recommendations:

All researches done uptill now had revealed that the electrolyte disturbance due to cerebrovascular accidents has severe effect on the brain functioning. Therefor its lead to severe complications likes organ failures and even death. Thus we suggest that all patients with acute stroke event should be assessed for electrolyte disturbance as early as possible, this can help the clinician for better prognosis and avoid complications. Also more researches is required about electrolyte disturbance and risk factors of stroke.

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Appendix-1

A-Study group

Questionnaire about electrolytes disturbances in hemorrhagic and non-hemorrhagic stroke patients

- **Name: Age: Address: Occupation: Marital status: Date of admission:**

- **Hx. Of present illness:**
- **Type of stroke :- ischemic ☐ hemorrhagic ☐ TIA ☐**
- **Past medical hx. : HT ☐ DM ☐ Epilepsy ☐ bleeding tendency ☐**
- **Drug history : Smokers ☐ alcoholics ☐**
- **Social hx. : rural ☐ urban ☐**
- **Family history of first degree relatives :**

  DM ☐ HT ☐ Stroke ☐

- **History of hospitalization :**
  Previous similar attacks yes ☐ no ☐
- **Level of consciousness :**
  Drowsiness ☐ delirium ☐ stupor ☐ coma ☐

- **Speech:**
  Normal ☐ dysphasia ☐ dysarthria ☐

dysphonia ☐

- **Cognition :**
  Oriented ☐ **disoriented** ☐

- **Degree of deficit :**

  Paralysis → **Rt. Upper limb** ☐ **left** ☐

  Rt. Lower limb ☐ **left** ☐

  Rt.upper &lower limbs ☐ **left** ☐

- **Fascial weakness**

- **Sensory disorders**

- **Visual disturbances**
- **Investigations :**

  CBP ☐ **B.urea** ☐ **S.creatinine** ☐

  S.Na^+ ☐

  S.K^+ ☐ **Brain CT** ☐ **Other IXS.** ☐

B-Control group

Questionnaire of control group

- **Name: Age: Address: Occupation: Marital status: Date of admission:**
- **Hx. Of present illness:**
- **Past medical hx. : HT ☐ DM ☐ Epilepsy ☐**

  bleeding tendency ☐
- **Drug history : Smokers ☐ alcoholics ☐**
- **Social hx. : rural ☐ urban ☐**
- **Family history of first degree relatives :**

  DM ☐ HT ☐ Stroke ☐
- **Investigations :**

  S.Na S.K Lipid profile

APPENDIX 2

National Institutes of Health Stroke Scale

	Category	Description	Score
1a	level of consciousness (LOC)	Alert Drowsy Stuporous Coma	0 1 2 3
1b	LOC questions (month, age)	Answers both correctly Answers 1 correctly Incorrect on both	0 1 2
1c	LOC commands (open and close eyes, grip and release normal hand)	Obeys both correctly Obeys 1 correctly Incorrect on both	0 1 2
2	Best gaze (follow finger)	Normal Partial gaze palsy Forced deviation	0 1 2
3	Best visual (visual fields)	No visual loss Partial hemianopia Complete hemianopia Bilateral hemianopia	0 1 2 3
4	Facial palsy (show teeth, raise brows, squeeze eyes shut)	Normal Minor Partial Complete	0 1 2 3

5	Motor arm left* (raise 90°, hold 10 seconds) (preferably with the palm facing up)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
6	Motor arm right* (raise 90°, hold 10 seconds) (preferably with the palm facing up)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
7	Motor leg left* (raise 30°, hold 5 seconds)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
8	Motor leg right* (raise 30°, hold 5 seconds)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
9	Limb ataxia (finger-nose, heel-shin)	Absent Present in 1 limb Present in 2 limbs	0 1 2
10	Sensory (pinprick to face, arm, leg)	Normal	0

		Partial loss Severe loss	1 2
11	Extinction/neglect (double simultaneous testing)	No neglect Partial neglect Complete neglect	0 1 2
12	Dysarthria (speech clarity to "mama, baseball, huckleberry, tip-top, fifty-fifty")	Normal articulation Mild to moderate dysarthria Near to unintelligible or worse	0 1 2
13	Best language** (name items, describe pictures)	No aphasia Mild to moderate aphasia Severe aphasia Mute	0 1 2 3
	Total	-	0-42

* For limbs with amputation, joint fusion, etc, score 9 and explain
** For intubation or other physical barriers to speech, score 9 and explain. Do not add 9 to the total score.

الخلاصة:

الخلفية:

تصنف الجلطه من بين جميع الامراض العصبيه للبالغ في المركز الاول. على الرغم من ان هناك العديد من الدراسات حول الجلطه الدماغية في قطرنا الا ان هناك لايوجد سوى القليل حول اضطراب ايونات الدم لدى مرضى الجلطه وايضا خارج البلد. اضطراب ايونات الدم لوحظت بكثره لدى مرضى الجلطه الدماغيه النازفه وغير النازفه ووجد لها تأثير فعال على نتائج حالة المرضى وتطورها.

الهدف:

لتقييم مستوى ايون الصوديوم والبوتاسيوم في الدم لدى المرضى الذين لديهم مختلف انواع الجلطه الدماغيه بالمقارنه لمجموعه اخرى من المرضى الذين لايعانون من الجلطه.

الطرائق:

تعد هذه الدراسه دراسه مقطعيه للمقارنة بين ايونات الدم الصوديوم والبوتاسيوم للمرضى الذين يدخلون الى مركز الجمله العصبيه واخرون الى قسم الباطنيه لاي مرض كان عدا الجلطه الدماغيه في مستشفى الديوانيه التعليمي من نيسان الى تموز 2018.

النتائج:

اظهرت الدراسه ان سكر الدم ,ارتفاع ضغط الدم,التدخين,خلل مستوى دهون الدم,السمنه والتاريخ العائلي للمرض هي عوامل خطوره على جميع انواع الجلطات الدماغيه وليس على نوع محدد فقط. وحسب مقياس درجة الوعي عند وصول مرضى الجلطه فقد وجد ان مرضى الجلطه النازفه هم الاقل وعيا عند وصولهم الى المستشفى وعند مقارنه ايونات الدم بين المجموعتين وجد ان انخفاض ايون الصوديوم والبوتاسيوم الاكثر عند مرضى الجلطه النازفه ثم الجلطه الغير نازفة ويليه مرضى الجلطات العابره والاكثر مستوى لهذه الايونات وجدت عند المرضى الذين لايعانون من الجلطه.

الاستنتاجات والتوصيات:

أظهرت الدراسه الحالية ان اضطراب ايونات الدم الصوديوم والبوتاسيوم كان اعلى نسبه لدى مرضى الجلطه النازفه وكان هذا الاضطراب هوه انخفاض في مستوى الصوديوم والبوتاسيوم وطبقا لهذه الدراسه فان هذا الاضطراب يؤثر على درجة المرض وشدته وعلى حالة المرضى الصحيه مابعد الجلطه لذلك يوصى الاطباء بضروره قياس مستوى ايونات الدم لدى جميع مرضى الجلطه الدماغيه فور تشخيصهم والعمل على تصحيح مستوى هذه الايونات في الدم الى المستوى الطبيعي لتفادي مضاعفات اضطرابها.

اقرار المشرف

اني الاستاذ الدكتور عقيل رحيم حسن المشرف على رسالة طالبة الدبلوم العالي (المعادل للماجستير) زهراء عادل عريان , قد اطلعت على رسالة الطالبة المذكورة والتي انجزت تحت اشرافي , اقر وأؤيد صلاحيتها للمناقشة لأستيفائها كافة المتطلبات العلمية لدرجة الدبلوم العالي.

التوقيع:

المشرف **: الاستاذ الدكتور عقيل رحيم حسن.**

مصادقة

اني رئيس فرع طب الاسرة والمجتمع في كلية الطب جامعة القادسية , اصادق على اقرار المشرف على رسالة طالبة الدبلوم العالي (المعادل للماجستير) زهراء عادل عريان واعتبر الرسالة صالحة للمناقشة من قبل اللجنه الممتحنه لهذا الغرض.

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