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Article

A Study on Ischemic Stroke Data Collected from Some Biochemical Analysis of Stroke Patients

Radwan, EH[1]; L Soltan[2]; M Eltody[1] and K Abdel Aziz[1]

1 Damanhour University, Faculty of Science
2 Alexandria University, Faculty of Medicine

Corresponding author: dr_eman_hashem@yahoo.com , radwan.eman@sci.dmu,edu,eg

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Abstract

The present study was to detect the effect of stroke on biochemical serum analysis including thyroid hormones. This study was conducted on 100 people (50 were normal control) + (50 were patients with ischemic cerebrovascular stroke based on clinical presentation and radiological findings) presented to Alexandria University Hospitals. circulation and memory and was purchased from (EMA Pharma pharmaceutical) from (Plaza building, Asma Fahmy st., Nozha, Nasr city, Cairo, Egypt). The present results of patients showed a statistical significance difference for SGPT, SGOT, Urea, Creatinine, Uric Acid, Cholesterol, Glucose, FT3, White Blood Cells, Red Blood Cells, Hct and Platelets count between patients and control humans. Results of patients showed a statistical significance difference for SGPT, SGOT, Urea, Creatinine, Uric Acid, Cholesterol, Glucose, FT3, White Blood Cells, Red Blood Cells, Hct and Platelets count between patients and control.

Keywords: environment, brain stroke, stress.

1. Introduction

A stroke may be a medical condition during which poor blood flow and injury. Signs associate with degreed symptoms of a stroke could embody an inability to dizziness or loss of vision. If symptoms last for two hours, it's referred to as a transient ischaemic attack (TIA) or mini stroke. Blood provide to a part of the brain is bated, resulting in the pathology of the brain tissue in this space (Donnan et al. 2008). There are two main kinds of hemorrhage (Goldstein and Simel 2005). Stroke symptoms generally begin suddenly, over seconds to minutes, and in most cases don't progress additional. In intracranial hemorrhage, the affected space could compress alternative structures. Most types of strokes don't seem to be related to a headache, except for subarachnoid hemorrhage (Kothari et al. 1999). Neuroprotection is an intervention, typically involving drug administration that acts directly on the intracellular mechanisms of the anemia cascade to affect the stroke (Teocchi 2010). Most variables area unit tightly controlled in laboratory experiments; thus, they will not mirror factors contributory to strokes within the human population as a full. In distinction, an individual's patient experiencing the associate anaemia method might not notice the symptoms or request medical help promptly (Wessmann et al. 2009).

The pathophysiology of cerebral anemia has been studied in animals with varied types of ischaemic lesions. These models have shown that metabolic alterations in reperfusion could result in cellular lesions in specific brain regions, betting on the length of the anemia (Farooqui et al. 2006). Regional destruction of the brain is followed by alterations in motor activity. although the recovery method begins bit by bit when the development of the lesion, the motor operate gift before the lesion won't essentially be recovered (Lessa 1999).

The use of various experimental models is beneficial for experimental studies on anemia, preventing the event of a customary surgical model. The best model has the characteristics of clinical relevancy, simple experimental execution, reliableness. many strategies of anemia induction are represented, as well as surgery, blood vessel embolism, and occlusion of (3 or 4) cervical vessels. Variation in time of anemia contributes to the variety of the experimental models used, the foremost used technique for inducement anemia is occlusion by middle artery occlusion (Calloni 2006). In tests of motor behavior, animals given different degrees of useful defects on the contralateral aspect of the anemia. Histologically, middle artery occlusion produces tiny death central and apoptotic peripheral regions (Mendez-Otero et al. 2009). Occlusion of the central artery is that the most ordinarily used surgical technique of manufacturing stroke. By 1st damaging neural structure structures and so damaging animal tissue structures, this occlusion mimics human striatocapsular infarcts in terms of size and therefore the structures affected. Striatocapsular infarcts have an effect on the bulk of the basal ganglia or adjacent nervous tissue. These lesions are caused by occlusion of the transient middle artery with early reperfusion or, if the occlusion persists, with smart collateral be due to the anterior or posterior arteries to the animal tissue middle artery territory (Rossmeisl et al. 2007).

The maidenhair tree leaf extract has stepped into the seasoning spotlight principally attributable to its established edges for treating presenile dementia (Yao et al. 2004). It conjointly seems promising as a therapeutic for several different chronic and acute sorts of diseases (Izzo and Ernst 2001). The 2 main pharmacologically active teams

of compounds gift within the maidenhair tree leaf extract the flavonoids and also the terpenoids (Smith and Luo 2004). Flavonoids, conjointly known as phenylbenzopyrones or phenylchromones, square measure a gaggle of low relative molecular mass substances that square measure wide unfold within the kingdom. Flavonoids gift within the maidenhair tree leaf extract square measure flavones, flavonols, tannins, biflavones (amentoflavone, bilobetol, 5-methoxybilobetol, ginkgetin, isoginkgetin and sciadopitysin), and associated glycosides of quercitin and kaempferol hooked up to 3-rhamnosides, 3-rutinosides, or p-coumaric esters. The flavonoid content within the leaf is varied between seasons; larger amounts square measure found in fall than in spring (McKenna et al. 2001). These compounds are glorious to act principally as antioxidants/free-radical scavengers, catalyst inhibitors, and ion chelators (DeFeudis and Drieu 2000).

The bioavailability of flavonoids is comparatively low because of restricted absorption and fast elimination. Flavonoids within the glycosidic type are poorly absorbed within the intestine; solely within the aglycone type will they're absorbed directly (Goh and Barlow 2004). Unabsorbed flavonoids that reach the colon is also subject to metabolism by microorganism enzymes, and so absorbed (DeFeudis and Drieu 2000). Once absorbed, flavonoids reach the liver wherever they're metabolized to conjugate derivatives. It is well-known that the biological activities of flavonoid metabolites don't seem to be continuously equivalent as those of the parent compound (Manach et al. 2004). Two sorts of terpenoids are gift in gingko as lactones (nonsaponifiable lipids gift as cyclic esters): ginkgolides and also the bilobalide (Smith and Nilotic 2004). Ginkgolides are diterpenes with five varieties A, B, C, J, and M, where A, B, varieties and C account for around three. One per cent of the whole gingko leaf extract (DeFeudis and Drieu, 2000). Bilobalide, a sesquiterpene trilactone, accounts for the remaining of nine percent of the whole standardized gingko leaf extract (Smith and Nilotic 2004). The counseled dose of standardized extract, EGb 761, is forty to sixty mg, three times daily supported clinical trials (Mahady, 2001). For chronic conditions the German commission recommends a minimum 8-wk intake to watch the helpful effects of the Gingko leaf extract (McKenna et al. 2001).

The aim of this work is on laboratory on patients, trying to find some possible correlations through laboratory study to detect the effect of stroke on biochemical serum analysis including thyroid hormones.

2. Materials and Methods

This study was conducted on 100 people (50 were normal control) + (50 were patients with ischemic cerebrovascular stroke based on clinical presentation and radiological findings) presented to Alexandria University Hospitals. The reagent Ginkgo biloba is a dietary supplement for improving circulation and memory and was purchased from (EMA Pharma pharmaceutical) from (Plaza building, Asma Fahmy st., Nozha, Nasr city, Cairo, Egypt).

Work design: Comparing data collected from biochemical analysis of stroke patients with that of control.

Determination of the RBCs count, (Wintrobe, 1976) . Determination of haemoglobin, (Hgbcontent Dacie and lewis, 1975). Determination of Haematocrit value (Packed Cell volume ((Oser, 1979).Determination of Platelets (PIT count ((Seivered, 1983) .Total leucocytes count investigated by using the haemocyto -meter method (according to Miale, 1972.: Determination of serum blood sugar enzymatic colorimetric method (GOD-POD(Young et al., 1972). Determination of lipid profile in the serum, Determination of

serum total cholesterol (Allain et al., 1974).Determination of serum triglycerides (Fassati and Prencipe, 1982). Determination of liver functions in the serum: Determination of alanine aminotransferase; ALT; Kachmar and Moss,1976. Determination of aspirate transaminase activity; AST; Kachmar and Moss .,1976. Determination of kidney function tests; Determination of urea concentration; Urea level in serum was assayed by using commercial kit that was supplied by Diamond, Egypt. Urea was estimated according to the method of Patton and Crouch .(1977).Determination of Creatinine concentration: ; The assay is based on the reaction of Creatinine in alkaline solution with picrate to form a red colored complex. The intensity of color formed is direct proportional to Creatinine concentration in the sample Bowers and Wong. (1980). . Determination of Uric acid concentration;The assay is based upon the methods of modified trinder peroxidase assay using 3,5-dicholoro-2hydroxybenzenesulfonic acid ; DCHB; Fossati et al., 1982.Determination of thyroid function tests; Determination of Free T3 concentration according to Maes, (1997.Determination of Free T4 concentration according to Thakur, 1997.Determination of Thyroid Stimulating Hormone ;TSH concentration according to Morimoto, 1998.

RESULTS:

This study was conducted on one hundred people. Fifty of them were patients suffering from ischemic cerebrovascular stroke based on clinical presentation and radiological findings presented to Alexandria University Hospitals. Thirty-one (62.0%) of them were males while the other nineteen (38.0%) were females. Their ages ranged between 41 and 76 years with a mean of (59.53±9.16). The other Fifty subjects were selected as control group matching in age and sex with the patients group.

Table (1) shows that, sixty-two patients (62%) were males while thirty-eight patients (38%) were females. Their ages ranged between 41 and 76 years with a mean of 59.53 ± 9.16.

Table (1): Distribution of the studied cases according to demographic data (n=50)

Demographic data	No.	%
Sex		
Male	31	62.0
Female	19	38.0
Age (years)		
≤50	15	15.0
>50	85	85.0
Min. – Max.	41.0 – 76.0	
Median	59.50	

Table (2): Comparison between different studied groups regarding liver function tests.

	Patients group "n=50"	Control group "n=50"
SGPT(U/L) Range Mean SD	 17-40 26.98 6.52	 15-29 21.70 3.63
T-test P value	2.54 0.007*	
SGOT(U/L) Range Mean SD	 19-45 31.56 7.37	 13-30 21.80 4.23
T-test P value	3.65 0.003*	

t = student t-test, P was significant if ≤ 0.05

** Highly Significant at level 0.01.

Table (3): Comparison between different studied groups regarding kidney function tests:

	Patients group "n=50"	Control group "n=50"
Urea(mg/dl) Range Mean SD	 22-57 36.92 7.44	 19-43 31.48 7.05
T-test P value	2.89 0.011*	
Creatinine (mg/dl) Range Mean SD	 1-1.7 1.31 0.21	 0.8-1.5 1.17 0.19
T-test P value	2.01 0.020*	
Uric Acid (mg/dl) Range Mean SD	 3.9-9 6.08 1.37	 2.4-5.9 4.15 0.93
T-test P value	2.89 0.004*	

t = student t-test ,P was significant if ≤ 0.05,** highly Significant at level 0.001.

Table (4): Comparison between different studied groups regarding lipid profile

	Patients group "n=50"	Control group "n=50"
Cholesterol(mg/dl) Range Mean S.D.	 135-263 191.54 28.88	 134-198 175.70 16.65
T-test P value	3.08 0.0064*	
Triglycerides (mg/dl) Range Mean S.D.	 73-173 109.10 26.65	 72-152 105.66 16.50
T-test P value	0.82 0.220	

t = student t-test , P was significant if ≤ 0.05, ** highly Significant at level 0.001,N.S. not significant.

Table (5): Comparison between different studied groups regarding Glucose test:

	Patients group "n=50"	Control group "n=50"
Glucose (mg/dl) Range Mean S.D.	 83-201 136.48 32.17	 72-110 92.44 11.89
T-test P value	4.05 0.001**	

t = student t-test , P was significant if ≤ 0.05,* Significant at level 0.05, ** Highly significant at level≤ 0.001.

Table (6): Comparison between different studied groups regarding thyroid hormones results

	Patients group "n=50"	Control group "n=50"
F.T3(pg/ml) Range	1-4.4 2.97	2-4.4 3.38

Mean S.D.	0.98	0.74
T-test P value	2.08 0.027*	
F.T4(ng/dl) Range Mean S.D.	 0.91-2 1.21 0.29	 0.93-1.7 1.30 0.28
T-test P value	1.78 0.056	
TSH(mIU/ml) Range Mean S.D.	 0.4-4.1 2.68 0.98	 0.27-4.2 2.40 1.05
T-test P value	1.61 0.082	

t = student t-test, P was significant if ≤ 0.05,** highly Significant at level 0.001,N.S. not significant.

Table (7): Comparison between different studied groups regarding blood picture test:

Blood picture	Patients group "n=50"	Control group "n=50"
WBCs (thousands/cmm) Range Mean S.D.	 8.1-14.3 11.51 1.56	 4-11 5.95 1.56
T-test P value	5.01 0.001**	
RBCs (Millions/cmm) Range Mean S.D.	 4-6 4.93 0.42	 3.8-5.2 4.53 0.41
T-test P value	1.98 0.046*	
Hb (g/dl) Range Mean S.D.	 13.2-16.1 14.72 0.75	 12-16 14.27 0.86
T-test P value	1.69 0.085	
Hct(%) Range Mean	40-55 44.82 3.63	34-46 40.88 2.75

S.D.		
T-test P value	1.97 0.039*	
Plt.(thousands/cmm) Range Mean S.D.	 316-464 402.54 35.42	 183-300 257.64 37.86
T-test P value	5.11 0.001**	

t = student t-test , *P was significant if ≤ 0.05, ** highly Significant at level 0.001.

DISCUSSION

Stroke may be a focal medical specialty deficit caused by an alteration in circulation within the brain within the last decade, this term has evolved to incorporate injuries caused by hemodynamic disturbances and natural action that can't be detected in arteries or veins (John et al. 2011). Stroke morbidity is St Martin's Day in girls and eight.4% in men and is additional current among blacks than whites area unit, particularly within the younger age teams (O'sulivan and Schimitz 2004). The most common type of stroke is atherothrombotic brain infarction, which accounts for approximately 61% of all strokes . The second most common type of stroke is embolic stroke, at 22%. Most stroke survivors develop lasting symptoms, such as physical and intellectual limitations, leading to high social costs (Wang 2009).

Encephalic vascular accident (EVA) occurs in four different forms:1) Ischemic and transitory, 2) Ischemic and complete, with neurologic deficits, 3) Progressive, 4) Hemorrhagic (Arthur et al. 2008). The present study has analyzed the results of ischemic stroke patients and its severity regards some biochemical analysis and serum thyroid hormones level. The liver participates to a variable extent in the acute phase of ischemic stroke in patients, producing enzymes in response to signals coming from cerebral infarct and proportional to its size. An important signal is likely represented by inflammatory cytokines (Campos et al. 2011).

The present study showed that there was a statistical significance of liver enzymes ALT, AST after the occurrence of ischemic stroke, which were probably influenced by inflammation. These results were inconsistent with Campos et al. (2011) who found that AST is the only liver enzyme directly associated with the ischemic cerebral lesion independently from inflammation. Possibly this enzyme, neutralizing the toxic glutamate, might play a protective role, as some reports on its favorable prognostic significance suggest (Sobrino et al., 2011).

The association of kidney dysfunction with post stroke outcomes may be because of several possible factors. Renal impairment in patients with stroke may indicate end-organ damage from common risk factors, such as uncontrolled hypertension or other comorbidities (MacWalter et al. 2002). Renal impairment may cause endothelial dysfunction, homocystenemia, coagulation disorders, extravascular coagulation, and higher levels of inflammatory cytokines (El Husseini et al. 2014). The present results showed a statistical significant difference of kidney; (Urea, Creatinine) after ischemic

stroke. The present findings further extend and strengthen previous studies suggesting that renal dysfunction may be associated with increased post stroke mortality (Putaala et al. 2011). These results were consistent with MacWalter et al., (2002) who reported that high serum Creatinine, and high urea concentrations post stroke were associated with a higher risk of all-cause mortality.

Serum uric acid (SUA) is well known to be associated with cardiovascular risk factors such as chronic kidney disease. However, it is difficult to show the causality between the SUA level and cardiac and cerebrovascular diseases because the former is easily affected by food (Kuwabara 2016). The present results showed a statistical significant difference of serum uric acid after ischemic stroke with probability of value <0.05 (p=0.004) between normal control and patients with stroke. The present study was consistent with Masoud et al. (2012) studies who said that prevalence of hyperuricemia is significantly higher in patients with acute ischemic stroke than normal population.

Some studies reported a positive independent relationship between uric acid and stroke whereas others demonstrated that uric acid did not relate significantly to stroke occurrence (Hariklia et al. 2008). It is difficult to establish an independent relation of hyperuricemia with ischemic stroke. Some studies revealed hyperuricemia as a protective factor of ischemic stroke (Chamorro et al. 2004). A high cholesterol level is a powerful risk factor for coronary heart disease (Wattanakit et al. 2005).

Associations between high serum total cholesterol (TC) levels and an increased risk of ischemic stroke have been reported (Benfante et al. 1994). Most of brain cholesterol originates from local synthesis rather than plasma lipoproteins and serum cholesterol does not necessarily correlate with its content in the CNS, it should be kept in mind that cholesterol is the essential constituent of plasma membranes, and regulates their fluidity and permeability (Murphy and Johnson 2008). The present study showed a statistical significant difference of serum cholesterol between control and stroke patients. This was consistent with a study in America indicated a positive association of total cholesterol levels with atherothrombotic infarction (Ohira et al. 2006).

High triglycerides are associated with several abnormalities of the body's clotting systems, which may contribute further to their association with cardiovascular disease (Tanne et al. 2001). Austin et al., (1998) found that the mean average of TG was 169.71 mg/dl in patients and 148.68 mg/dl in controls. Bowman et al. (2003) found that the mean average of TG was 192.3±155.9 mg/dl in patients and 157.0±93.0 mg/dl in controls. By provoking anaerobic metabolism and free radical production, hyperglycemia may exert direct membrane lipid peroxidation and cell lysis in metabolically challenged tissues (Kernan et al. 2002). The present study showed that there was a statistical significant difference of blood glucose level between control and stroke patients. The value of glucose in patients group range (83-201) mg/dl. The present results were consistent with Scott et al. (1999) study that showed that elevated blood glucose is common in the early phase of stroke. Hypothyroidism can cause hypertension, hypercholesterolemia, cardiac dysfunction, and both hypo- and hypercoagulability, all of which are risk factors for stroke (Bai et al. 2014). Hyperthyroidism is also associated with atrial fibrillation, which is a common cause of cardio embolic stroke (Chen et al. 2014). Elevated concentrations of thyroid hormones are associated with an increase in energy and oxygen demand, which would be expected to impair ischemic tolerance in the brain. However, more studies that are detailed are required to determine the impact of thyroid

function on cerebral ischemia (López et al. 2010).

Tri-iodothyronine (T3) has an ability to induce hypothermia, anti-inflammation (Li et al. 2017). Free triiodothyronine (FT3), the bioactive form of T3, also plays an important role in neurogenesis in all stages of brain development (DEG et al., 2004). patient monitoring of thyroid status may be important, as higher levels of FT3 have been associated with better functional outcomes (Winter et al. 1988). The present results showed a statistical significance difference of FT3 level. Patients group range (1-4.4) pg/ml; (2.97±0.98), control group range (2-4.4) pg/ml; (3.38±0.74). Wang et al. (2018) reported that the subgroup analysis indicated that in the acute phase of ischemic stroke, TSH was associated with severity of PSF in the groups with euthyroidism ($p<0.001$), subclinical hypothyroidism ($p<0.001$), and low-T3 syndrome ($p = 0.008$). Higher TSH was associated with better Fatigue Severity Scale scores in patients with low-T3 syndrome 6 months after the index stroke ($p = 0.01$).

The present results were in consistent with O'Keefe et al. (2015) who found that low Thyroid Stimulating Hormone (TSH) and free triiodothyronine (FT3) were associated with poor function at 3 months, and that low TSH and fT3 were associated with higher rates of death in the hospital. Zhang et al. (2019) results had indicated that the lower FT3 in patients with acute ischemic stroke (AIS) was associated with greater baseline severity of ischemic stroke on admission. Leukocytes are the first cells that arrive in the stroke region (s), and they increase in peripheral blood. Leukocytosis on admission was related to initial stroke severity but not to outcome. Leukocyte count on admission seems merely to reflect initial stroke severity and is most likely a stress response with no independent influence on outcome (Kazmierski et al. 2001).

Previous stroke studies had demonstrated that patients with greater inflammation have larger volumes of injured tissue where leukocytes can accumulate (Christensen and Boysen 2004). The persistence of leukocytosis can ultimately lead to worse neurologic outcomes. Leukocyte migration and accumulation was measured using leukocytes labeled with radioactive markers and scintigraphy or single-photon emission computed tomography. The present study, along with several others, has evaluated the efficacy of using WBC count as a prognostic marker among patients with acute ischemic stroke. Current study showed a statistical significance between normal control and ischemic stroke patients. Patients group range (8.1-14.3) thousands/cmm; (11.51±1.56), Control group range (4-11) thousands/cmm; (5.95±1.56).

Hematological parameters are used for diagnosis and prognosis of numbers of hematological diseases (Nadkarn et al. 2009). Hematological analysis are found to be useful for prognosis and can be of immense value for stroke patients; like erythrocyte sedimentation rate (ESR), platelet count and leukocyte count in blood sample collected at the time of admission for prediction of stroke outcome (Yoon and Zheng ,2005). Researchers found that anemia was present in about a quarter of patients with stroke upon admission and was associated with a higher risk of death for up to one year following either ischemic stroke or hemorrhagic stroke. Elevated hemoglobin levels were associated with poorer outcomes and a higher risk of death (Raphae et al. 2016). The present study showed the statistical significant difference between control group and patients group with probability of value $p<0.05$ in RBCs and hematocrit concentration, but without any statistical significance in Hb% ($p>0.05$). Circulating platelets play critical role in the development of ischemic stroke by acting as mediator for other circulating cells through

facilitating activation (Thomas and Storey 2015).

The present results showed the elevating in platelets count in patients group with statistical significant difference between normal control and ischemic stroke patients (p=0.001). Consistent with present study, Du et al. (2016) found a positive correlation between elevated PC level and the risk of stroke recurrence. Inconsistent with the present results, some studies indicated that platelets count (PC) was significantly lower in patients with ischemic stroke and myocardial infarction compared with healthy controls (Ranjith et al. 2009). Some studies have presented a positive correlation between PC level and platelet-induced pro-thrombotic (Li et al. 2016). It is possible to speculate that the two mechanisms, platelet consumption and platelet-induced inflammation, reached lower activity at intermediate PC level, making platelet count can be of prognostic significance for improved risk stratification of adverse clinical outcomes in ischemic stroke and TIA patients (Elkind et al. 2014).

The use of animal models has provided a better understanding of the pathophysiologic mechanisms of strokes. Mice and rats are the most commonly used species, to better study the disease and its treatments (Teocchi 2009). A human patient experiencing an ischemic process may not notice the symptoms or seek medical assistance promptly. Precise and rapid identification of symptoms enables improved treatment options and outcomes (Wessmann et al. 2009). Another important difference between animal models and humans is the rigorous control of the animals used. Typically, young, healthy, genetically similar animals of the same sex or age groups are used, especially in studies involving rodents. The typical stroke patient is elderly, with many risk factors, and may present additional complications, such as coronary disease. Age is a primary risk factor for stroke pathologies (Joutel et al. 2010). Animal models of cell death in stroke are designed to generate reproducible infarcts in a high throughput manner with a minimum of surgical manipulation to determine mechanisms of cell death (Yang and Lin 2002). Zhang et al. (2018) reported that liver disease and stroke have a negative correlation. In experimental studies, after 10 minutes of the simulation of stroke and hyperthermia, decrease in activity of superoxide dismutase and increase of free radicals in the liver were detected (Yang and Lin 2002).

Two hours after brain ischemia/ reperfusion in rats with a fatty diet, a sharp decline in the activity of antioxidant enzymes (superoxide dismutase, glutathione peroxidase) and increased levels of malondialdehyde and free calcium in the liver were observed (Parikh et al. 2017). In patients with traumatic and ischemic brain damage, changes of biochemical parameters of liver function are seen (Tziomalos et al., 2013). Abdeldyem et al. (2017) study showed that the initiation of inflammatory response was detected only on the 5th day of the experiment. Covic et al. (2008) study showed that renal function on admission is a strong independent prognostic factor for long-term mortality and new cardiovascular morbidity following stroke over a 10-year period.

In the Mc Walter et al. (2002) study, a similar high proportion of patients who presented within 48 h of ictus had renal dysfunction despite the differences in the risk profile of patients. In the present study on experimental rats there was a statistical significance difference of Urea level value in stroke rats with probability of value (p=0.001), (rats with ischemic stroke) range (22-39) mg/dL; (32.20±4.44) while control group range (13-26) mg/dl; (19.87±3.58). It was consistent with previous studies. This might happen because urea concentration in blood is more affected by meals, which contain most protein, water, daily activity and sleep. The

present experimental study on rats regarding Uric acid level showed a statistical significance between control normal group and stroke rats group.

Cholesterol is present in all tissues and lipoproteins plasma. It exists in the form of free cholesterol or a combination of long chain fatty acids. Free cholesterol is expelled from tissues by HDL and transported to the liver to be converted into bile acids (Murray et al. 1999). The state of Hypercholesterolemia is characterized by increased blood cholesterol levels above normal. In Wistar rats, normal blood cholesterol level is 10-54 mg/dl (Smith and Mangkoewidjojo 1998). The present study on experimental rats showed a statistical significant difference between control and induced stroke rats, Control group range (47-73) mg/dl; (59.20±8.74). Rats with ischemic stroke group range (43-106) mg/dl; (79.33±18.53), the probability of value (p=0.005). According to Ganong (2002), estrogen effects blood cholesterol. A study in America indicated a positive association of Total Cholesterol levels with atherothrombotic infarction Ohira et al. (2006).

The complex process of aging is associated with changes in thyroid hormones metabolism and action in all tissues. During aging, the disruption of circadian rhythm leads to a reduction in thyroid stimulating hormone (TSH) secretion (Campos et al. 2013) and circulating TH levels, in particular T3, in humans (Bensenor et al. 2012) and rodents (Visser et al. 2016). Hypothyroidism and decreased TH availability to the brain has been considered a risk factor for the development of neurodegenerative diseases (Villanueva et al., 2013). Recent epidemiological studies have associated low levels of T3 with poor functional outcome after acute ischemic stroke (Dhital et al. 2017; Li et al. 2019). The present study on rats showed that there is a statistically significance for TSH (p=0.001). Control group range (1.03-2.28) mIU/ml; (1.64±0.35). Stroke patients with subclinical hypothyroidism were more prone to suffer a non-fatal stroke and minor adverse events (Chaker et al. 2016). It is likely that hypothyroid episodes prior to stroke only delayed neuronal death, due to decreased metabolic demand of neurons, decreased glutamate production and delayed oxidative stress (Rastogi et al. 2018).

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