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Relation of Lipoxygenase Activity with Some Biochemical Parameters in Epilepsy Patients

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Abstract

Epilepsy is a define as a generally disorder in neurological that affects more than 50 million people in the world, also is referred to as acute encephalitis, or a disorder in its system that leads to an over excitation of nerve tissues. Lipoxygenase [EC 1.13.11.12] catalyzes the bi-oxidation of polyunsaturated fatty acids. This enzyme pathway leads to the production of leukotrienes and fatty acid hydroperoxide that have essential role in many inflammatory diseases such nerve disorders.

In this study, 40 samples of epilepsy patients and 60 healthy of both sexes were taken. The activity of the lipoxygenase, polyamine oxidase and lipid profile triacylglycerol, total cholesterol, high density lipoprotein, low density lipoprotein and very low-density lipoprotein in addition to the level of glucose, uric acid, albumin, bilirubin and total protein as well as the level of vitamin E and C, malondialdehyde and glutathione were measured.

The results illustrate a significant increase in of Lipoxygenase glucose, uric acid, albumin and insignificant for polyamine oxidase, bilirubin and total protein in epilepsy patients compared with healthy. The level of MDA was a significant increase meanwhile GSH and vitamin E there was a significant decrease, and vitamin C was insignificant changed. All Lipids profile were a significant increased except high density lipoprotein was a significant decrease in epilepsy patients compared to healthy. The correlation of lipoxygenase appears clear negative with uric acid, malondialdehyde, vitamin E in epilepsy patients and positive correlate in healthy. No effect of sex except for lipoxygenase, uric acid, GSH, and albumin in females compared with males. while MDA increased significantly in male compared femal. Also, no effect found of age on all the measured variables. This study indicates a significant increase of LOX activity in epilepsy patients compared healthy, and a clear negative relationship with uric acid, MDA and vitamin E.

Key word: Lipoxygenase. Oxidative stress. Epilepsy, Neurological disorder .

Introduction

Epilepsy is define as a generally disorder in neurological that affects more than 50 million people in the world [1]. The essential reason that responsible for the occurrence of an epileptic seizure is the excessive discharge of the abnormal nerve cells in a specific in the brain and the imbalance in the brain leads to the occurrence of many different behavioral disorders [2]. The epilepsies are a category of disorders a group of neurological disorders in which a person suffers from an underlying brain dysfunction causes a decrease in the intrinsic seizure threshold, resulting in a higher risk of spontaneous recurrent seizures [3]as indicates the occurrence of a seizure that lasts for a certain period of time or is repeated more than once, which leads to the production of a permanent epileptic condition.The first classification of seizures was published by ILAE in 1960, and then it was updated in 1981 and the last update published in 1989, in 2016 was presented a report by ILAE for classification and terminology provided some changes in concepts and definitions of seizures and epilepsy [4]. According to ILAE, seizures can be classified as focal or generalized depending on whether one or both hemispheres of the brain are responsible for the initial symptoms appearing when the seizure activity occurs [5]. Epilepsy is classifying to three levels: type of seizure, epilepsy, and the syndrome at each stage. The reasons and symptoms associated with three levels must be identified because of its important therapeutic.

Lipoxygenase (EC 1.13.11.12) LOXs are a group of dioxygenases that contains non-heme iron and catalyzes the bi-oxidation of polyunsaturated fatty acids (PUFAS) containing 1-cis-4-cis-pentadiene to convert them into fatty acids, hydroperoxides [6]. Among these enzymes, 5-LOX is the most important factor contributing to the formation of lipid peroxidation and involved in the

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biosynthesis of leukotrienes that have essential role in many inflammatory diseases such as arthritis, asthma cancer, and allergic [7] Studies were elucidating that the inflammation is one of the mechanisms causing of epilepsy and its developing were many inflammatory molecules such as cytokines, chemokines, prostaglands and others are released by the brain, brain endothelial cells has been notice effects on nerve stimulation [8].

AA is released from the membrane phospholipids by cytosolic phospholipase A2 which is depending calcium [9]. AA is enzymatically metabolized to eicosanoids through three metabolic pathways Cytocrome P450, Cycloxygenase (COX) Lipoxygenase (LOX) pathways. In the LOX pathway, AA is metabolized to 8,12,15-(hydroperoxyeicosatet- raenoic acid) HPETE by 12 and 15-LOX or to 5-HPETE by 5-LOX and the 5-LOX activity protein (FLAP). A previous study showed that an acetazolamide has a more inhibitory effect than other antiepileptic drugs of epilepsy patients LOX activity [10**].**

Experimental

Samples

Blood samples were collected with the consent of all patients and their relatives to participate in this study. A study was included 40 epilepsy patients volunteers (22 male and 18 female) from Bin-Sina Teaching Hospital in Mosul/Iraq and 60 healthy subject (35 male and 25 female) with no accidental or chronic diseases. Two groups aged between 10-60 year

Collecting blood samples

Venous blood samples were collected with 5ml in plane laboratory tube with ethylenediamine tetraacetate as anticoagulant, centrifuged with 3500g for 10 minutes at 4ºC to obtained serum and the following laboratory parameters were performed.

Biochemical analysis

Assay of LOX activity: LOX activity was determined by the following procedure of [11] depend on oxidation of the linolic acid by the LOX enzyme to add two oxygen atoms, the increase in the absorbance which resulting from the formation of the conjugated dines is followed up at 234 nm for 5 min.

Assay of Polyamine oxidase PAO activity: enzyme activity was measured by dependent colorimetric method [12] by the following of the change in absorbance at 410 nm for 1min by using spermine as a substrate.

Serum glucose: Glucose was determined by using analysis tool kit. Glucose is oxidized to gluconic acid and hydrogen peroxide by the glucose oxidase, in the presence of the peroxidase with phenol as a hydrogen-donor, substrate is oxidized to the Quinoneimine, which is the color product Proportional to glucose concentration [13].

Uric acid U.A: U.A was determined using analysis tool kit. Uricase is oxidized U.A, CO_2 and H_2O_2 were produced by peroxidase, hydrogen peroxide reacts with chromogen (4-aminophenazone) to produced complex which absorption intensity at 520 nm, the color is proportional to the uric acid amount in serum [14].

Creatinine: Creatinine was estimated using an enzymatic method that used Spanish biosystem analysis kit. Creatinine in the sample reacts with picric acid and in the presence of sodium hydroxide, which results in a yellow complex, the absorption at 500nm is imply a direct proportion to the creatinine content in serum [14].

Total protein: Protein was estimated using the Biuret method [15] using French biolabo analysis kit. The principle based to interaction of the peptide bonds in the protein with a copper base potassium tartrate solution called the Biuret reagent to give a complex of purple color, absorbance at 550nm depends on the protein bonds number.

Albumin: Albumin concentration was estimated using the Bromocresol Green using French biolabo analysis kit, the method depends on the content of albumin associated with the green bromocresol reagent (5',5,3',3-tetrabromo -metacresol phthalene sulfate to be a green-colored Albumin-BCG complex was measured at 630nm [16].

Bilirubin: bilirubin estimated using analysis kit, the method is based interaction of bilirubin with the reagent Diazotized sulfalinic acid to form the color azobilirubin. Dimethyle sulfoxid was added to estimate the total bilirubin to determine total bilirubin at 550nm [17]and

Malondialdehyde MDA: Lipid peroxidation in the blood serum is estimated by measuring the amount of malondialdehyde as the final product of super oxidized lipids, the procedure depends on the interaction between lipid peroxides MDA and thiobarbituric acid (TBA) in an acidic medium. Colored product is measured at (532nm) [18].

Glutathione GSH: Antioxidant GSH in blood serum was detected using the modified method [14]by using of (Ellman's reagent) containing [5.5'-dithio bis (2-nitrobenzoic acid)] DTNB as a reagent interacts with glutathione and is reduced by the thiol group (SH) to form Colored compound measured at (412 nm).

Vitamin E (alp-tocopherol): Vitamin E was estimated using the redox and labeled method By(Emmeric-Engle reaction). This reaction includes oxidation of tocopherol to tocopherol quinone by ferric chloride which will give a red complex with α, α- Dipyridyl with xylene an organic solvent to extract tocopherols and carotenoids from the serum at the beginning, and then the absorbance

is read at(460 nm) in order to estimate carotene, then ferric chloride ($FeCl_2$) is added. The absorbance at (520 nm) taken to measure vitamin E[19].

Vitamin C (ascorbate): **Ascorbate** was estimated by oxidation of ascorbic acid with copper to form dihydro-ascorbic acid and diketo gluonic acid, then these products reacted with 2,4-Dinitro phenyl hydrazin presence of thiourea formed bis-2,4-dinitro phenyl hydrazine derivative, with the addition of sulfuric acid, a product measured at 520 nm [20].

Lipid profile amount: Total cholesterol, triacylglycerol and high density lipoproteins was measured by enzymatic reaction[13] using French Bio labo kits. Very low-density lipoprotein (VLDL-C) calculated by the equation

$$\text{VLDL-C} = \frac{\text{TG}}{5}$$

, Meanwhile low-density lipoprotein (LDL-c) by the equation

LDL-C = Total Chol. – (HDL-C + VLDL-C) [21].

Statistical analysis: Statistical significance was assessed using Student's t-test. A P-value of less than 0.05 was accepted as the significance level. All values are reported as mean ± SE.

Results and Discussion

The results in Table (1) indicate a significant increase in lipoxygenase activity ($p \leq 0.05$) of epilepsy patients (0.163 ± 0.014 U/ml) compared with control(healthy) (0.07 ± 0.005 U/ml). Also indicated a non-significant elevate in the level of polyamine oxidase ($p \leq 0.05$) in serum of epilepsy patients (0.134 ± 0.05 U/ml) compared to control (0.11 ± 0.09 U/ml).

Table 1. LOX and PAO activity in epilepsy patients and healthy

Enzyme activity[U/ml]	Control N=60		Patients N=40		*P ≤0.05*
	Mean	S. E	Mean	S. E	
Lipoxygenase	0.07	0.005	0.163	0.014	S
Polyamine oxidase	0.111	0.016	0.134	0.05	n.s

S= significant, n.s= non-significant

The high level of the lipooxygenase enzyme LOX in the blood of epilepsy patients Table (1) may be due to exposure to external effectors, acquired environmental influences, genetic factors and the occurrence of infections, as it leads to an increase in the immune system response to released polyunsaturated fatty acids such as arachidonic, these acids are substrates for LOX [9]. Arachidonic acid plays an important role in promoting the seizure because it directly strengthens the excitability of neurons [22]. Thus, the oxidation process begins and the LOX enzyme pathway is activated, which ultimately to leads the production inflammatory molecules such as cytokines and the formation of leukotrienes and (interleukotrienes 1-beta IL). -1β) Inter leukotren 1-beta), factor alpha (TNF-α) addition Interferon gamma (IFN-)) by the brain, brain endothelial cells, surrounding blood cells, and that the high level of these molecules affects the nerve excitation leads to Recurrent seizures in epilepsy [23]. According to study in rats, levels of spermine, spermidine, and putrecine increased due to the occurrence of severe seizures and were more clearance in putrcine, and thus they participate in the stimulation of neurons in the brains of epileptic patients [24]. This increase may be explained the high level of basic substances for polyamine oxidase and increase its activity epilepsy patients.

Where the results illustrate a significant increase in the level of glucose ($p \leq 0.05$) in serum of epilepsy patients (7.13 ± 0.36 mmol/L) compared to control group (5.78±0.18 mmol / L) as shown in figure 2 and table (2).

Table 2. Glucose, Uric acid and Protein profile in epilepsy patients and healthy

Biochemical variable	Control N=60		Patients N=40		*P ≤0.05*
	Mean	S. E	mean	S. E	
Glucose mmol/L	5.78	0.18	7.31	0.36	S
Uric acid mmol/L	3.26	0.14	4.6	0.34	S
Total protein mg/ml	68.57	2.06	72.78	2.54	n.s
Bilirubin µmol/L	7.02	0.64	5.11	0.58	n.s
Albumin mmol/L	1.17	0.018	1.01	0.092	S

S= significant, n.s= non-significant

A significantly increased in the level of uric acid ($p \leq 0.05$) in epilepsy patients (4.60 ±0.34 mg/dL) compared to control (3.26±0.14 mg/dL), whereas insignificant rise of total protein concentration ($p \leq 0.05$) in the serum of epilepsy patients (72.78±2.54 mg/ml) was found compared to control group (68.57±2.06 mg/ml) as shown in Figure 2 and table (2). Results illustrated insignificant decrease in the level of bilirubin concentration ($p \leq 0.05$) in the serum of epilepsy patients (5.11 ± 0.58 µmol/L) compared with healthy (7.02 ± 0.64 µmol/L). The results were indicated a significantly decreased in the level of albumin ($p \leq 0.05$) in the serum of epilepsy patients (1.01±0.092mmol/L) of the control (1.17±0.018 mmol/L).

A significant increase in the level of glucose table (2) may contributing the process of transporting glucose

across the blood-brain barrier to nerve cells is important and impairment of GLUT1 Glucose transporter 1 encoded by (SLC2A1) leads to epilepsy and intellectual disability [25]. Increasing in uric acid are identical previous studies have shown that high uric acid promotes an epileptic seizure [26].

The increase in total protein may be due to a change in the blood brain barrier as a result of diseases of the central nervous system that increase white blood cell transmission and then increasing in protein leakage, and plasma protein proliferation [27]. On the hand some specific proteins were studied such as haptoglobin an referred to participate in generalized epilepsy with idiopathic [28].

Insignificant decrease in bilirubin was obtained in the study whereas Ibrahim and Alhashemi [29] found a significant decrease. The reason may be due to bilirubin acts as an antioxidant function that can prevent the oxidation of fats, and it is affected by epilepsy patients [30].

Albumin as an antioxidant and its ability to eliminate free radicals resulting from inflammation and It is a powerful antioxidant in the blood, as a result, it may lead to a decrease in its amount in the blood [15]. These results are consistent with a study conducted on epilepsy patients being treated with Depakene, where it showed a low level of albumin concentration in the blood [31].

This study was indicating a significant increase in the level of the concentration MDA ($p \leq 0.05$) in the serum of epileptic patients (8.07±0.81 µmol/L) compared to the control group (3.31±0.35) µmol/L. GSH was a significant decrease ($p \leq 0.05$) in blood serum patients of epilepsy (1.94±0.21 µmol/L) compared with the control group (3.18±0.39 µmol / L) table (3).

Table 3. Oxidant and antioxidants in epilepsy patients and healthy

Biochemical variable	Control N=60		Patients N=40		*P ≤0.05*
	Mean	S. E	Mean	S. E	
Malondialdehyde µmol/L	3.31	0.35	8.07	0.81	S
Glutathione µmol/L	3.18	0.39	1.94	0.21	S
A-tocopherol µmol/L	8.86	1.42	3.26	0.32	S
Ascorbic acid µmoll/L	0.21	0.047	0.185	0.06	n.s

S= significant, n.s= non-significant

Vitamin E level was a significant decrease in epilepsy patients µmol/L (3.26 ± 0.32) compared with the control group (µmol/L 8.86 ±1.42). While the results showed a non-significant decrease in the level of vitamin C concentration at a probability level($p \leq 0.05$) in the serum of epilepsy patients (0.185±0.06µmol/L) compare control group (0.21 ± 0.047 µmol/L). The level of total cholesterol was a significant elevate ($p \leq 0.05$) in epilepsy patients (6.22±0.34 mmol/L) compared with control group (4.45±0.2 mmol/L) as shown in table (4), also indicated a significant increase ($p \leq 0.05$) in the level of triglycerides T.G at a probability level in the serum of epilepsy patients (2.07 ±0.25mmol/L) compared to its level in the serum of the control group (1.40 ± 0.15 mmol/L). Where HDL-C concentration decreased significantly ($p \leq 0.05$) in the serum of epilepsy patients (2.04±0.1 mmol/L) comparing to the control group (2.34 ± 0.18 mmol/L) as shown in table (4). Also, LDL-C was a increasingly significant ($p \leq 0.05$) in epilepsy patients (4.03 ±0.36 mmol/L) compared to the control group (2.29±0.36 mmol/L). Also the results indicated a significant increase ($p \leq 0.05$) in the level of VLDL-C levels in epilepsy patients (0.39 ± 0.05 mmol /L) compared with its serum concentration level in the control group (0.27±0.03 mmol/L)

Table 4. lipids profile in epilepsy patients and healthy

Biochemical variable	Control N=60		Patients N=40		*P ≤0.05*
	Mean	S. E	Mean	S. E	
T.C mmol/L	4.45	0.2	6.22	0.34	S
T.G mmol/L	1.4	0.15	2.07	0.25	S
HDL-C mmol/L	2.34	0.18	2.04	0.10	S
VLDL-C mmol/L	0.27	0.03	0.39	0.05	S
LDL-C mmol/L	2.29	0.36	4.03	0.36	S

S= significant, n.s= non-significant

Increase in MDA and decrease in GSH contributing to imbalance of an antioxidants due
to their depletion to scavenging free radicals, in this case unable to protect the fatty acids from oxidation, and the main product of lipid peroxidation is MDA [32]

This result is in agreement with [33], as free radicals generate epilepsy and thus decrease levels of glutathione in the epilepsy patients.

These results are agreeing with the studying as vitamin-E levels where much lower in epilepsy patients compared to the control group [34]. Vitamin E plays as an antioxidant, as it prevents the oxidation of polyunsaturated fatty acids (PUFAs) by protecting them and prevents the formation of free radicals [35], Where vitamin E reduces seizure activity as it prevents the effects of oxidative stress in brain tissue, it is an antioxidant that removes free radicals that have a role in the brain injury of epilepsy due to long seizures due to hyperoxia [36].

Vitamin C is an antioxidant accumulated in the nervous system, and it has been proven that vitamin C can be easily transported across the blood-brain barrier, as it participates with other antioxidants by reducing the injury of nerve cells (hippocampus) during seizures. In addition, vitamin C works on neuroprotection by integrating cell membranes and reducing lipid peroxidation [37]. The oxidative stress and generation of active oxygen species are closely related to a number of disorders including epileptic seizures [38].

Effect of the drugs CBZ Carbamazepine (Tricitol), VPA valproic acid (Dibacaine) and Phenytoin PB were among the most studied drugs, according to studies conducted on 4126 people illustrated that these drugs are involved in causing significant changes in plasma lipid levels for epilepsy patients who takes these drugs, cholesterol levels were high in these patients, most antiepileptic drugs, including CBZ, VAP and PB, are metabolized in vivo, mainly in the liver. [39].

Increasing of the hepatic lipase, may be contributing in this influence where the high-density lipoprotein is rich in T.G, and becomes a basic material for the of the hepatic lipase action, and this will lead to the removal of HDL-C and decreasing its level from the circulatory system [14]. Long-term treatment with antiepileptic drugs such as sodium valproate and carbamazepine increase levels of fats in the blood, including LDL-C and apo-lipoprotein A [40]. This may be due to an increased synthesis of this type of lipoprotein VLDL-C and decreased plasma clearance [41] No effect or significant contributing of age with parameter between two groups, whereas Shenta shows showed decreasing in antioxidant with an increase in age [42].

Effect of sex on some biochemical parameters

When studied the effect of sex on the biochemical parameter the results were illustrate in table (5)

insignificant increase in LOX activity in female compared to male epilepsy patients. While MDA was increased significantly in male patients compared with epilepsy female, and GSH increase in female compared male.

Also, albumin and uric acid was increasing significantly in female compared with male epilepsy patients. The reason for may cause by the difference in muscle protein breakdown between them.

Also the results indicate a non-significant influence in the level of each (PAO, glucose, creatinine) in female patients with epilepsy compared with male patients, and the results also the level of antioxidants (vit.E, vit.C, total protein and bilirubin). Also, serum lipid levels (T-Cho, TG, HDL, LDL-C and VLDL-C) were insignificantly higher in male epileptic patients compared with female patients.

Table 5. Effect of sex on biochemical parameters

Biochemical variable	Male	Female	
	Mean ± S.E	Mean± S.E	*p≤0.05*
LOX	0.15±0.01	0.19±0.03	s
PAO	0.07±0.01	0.30±0.22	n.s
Glucose	7.56±1.0	7.41±0.80	n.s
MDA	7.9±2.1	5.04±0.7	s
GSH	1.5±0.41	1.98±0.33	s
U. A	2.4±0.21	4.86±0.37	s
Albumin	0.71±0.12	1.14±0.11	s
T-Cho	6.41±0.75	6.46±0.43	n.s
T.G	1.53±0.33	2.14±0.33	n.s
LDL-C	4.36±0.77	3.95±0.42	n.s
VLDL-C	0.30±0.06	0.42±0.06	n.s
HDL-C	1.73±0.23	2.04±0.13	n.s

S= significant, n.s= non-significant

Coloration between LOX activity and biochemical parameters

The relationship of the lipooxygenase with the biochemical variables showed in table(6) that there is a weak direct corolate R = 0.03 between LOX and PAO activity in patients with epilepsy, while the results showed that there is a clear inverse relation between (MDA, vit.E, and UA) R value equal to (-0.35, -0.55, -0.41) , respectively with LOX in epilepsy patients. Also, the results showed that the weak opposite relationship between (vit.C, Albumin, T.Cho, GSH and glucose) with LOX activity (-0.12, -0.08, -0.12, -0.14, -0.12), respectively in epileptic patients, while the relationship was positive for (vit.C , Albumin, glutathione and glucose) in healthy subjects. The results also showed a

positive relationship between LOX activity and serum lipids (T.G , HDL-C , VLDL-C and LDL-C) with R values (0.23, 0.03, 0.23, 0.08) respectively in patient group except T.Cho was opposite, while it was reversible in healthy subjects. The results showed that there was a positive relationship R (0.46) between BMI and LOX, while it was opposite in healthy subjects (-0.02.).

Through the study of the relationship, distinct relationships were found between the infec ted, compared to healthy subjects, illustrated in the following figures.

Table 6. The Coloration between LOX activity and biochemical parameters

Parameters	Control	Epilepsy
PAO	**0.24**	**0.03**
Glucose	**0.01**	**-0.12**
Uric acid	**0.02**	**-0.41**
Bilirubin	**-0.42**	**-0.32**
Total protein	**0.12**	**0.03**
Albumin	**0.05**	**-0.08**
MDA	**0.19**	**-0.35**
GSH	**0.16**	**-0.14**
Vit.E	**0.21**	**-0.55**
Vit.C	**0.05**	**-0.02**
T-Cho	**-0.35**	**-0.12**
T.G	**-0.38**	**0.23**
HDL-C	**0.4-**	**0.03**
VLDL-C	**-0.35**	**0.23**
LDL-C	**-0.51**	**0.08**
BMI	**0.46**	**-0.02**

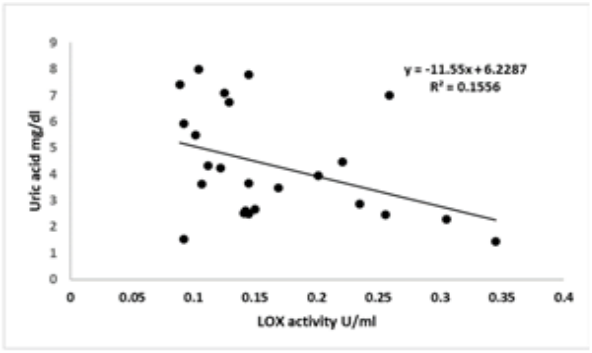

Fig1: Correlation between LOX activity and Uric acid in patients

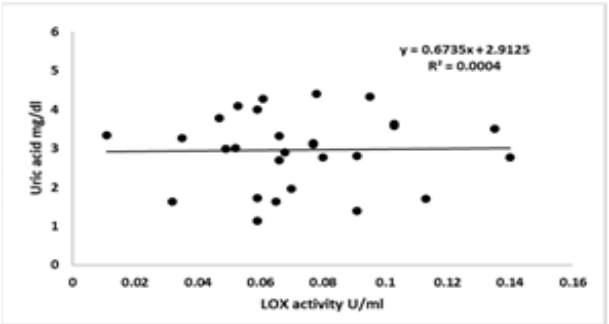

Fig2: Correlation between LOX activity and Uric acid in healthy

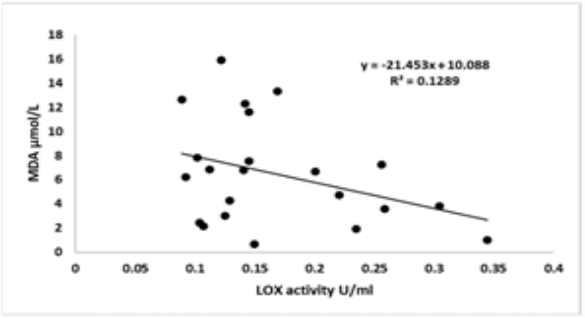

Fig3: Correlation between LOX activity and MDA in

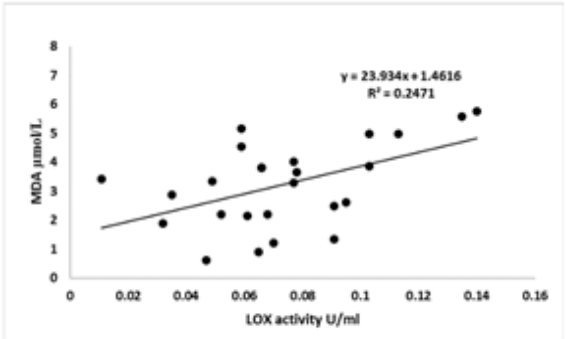

Fig4: Correlation between LOX activity and MDA in healthy

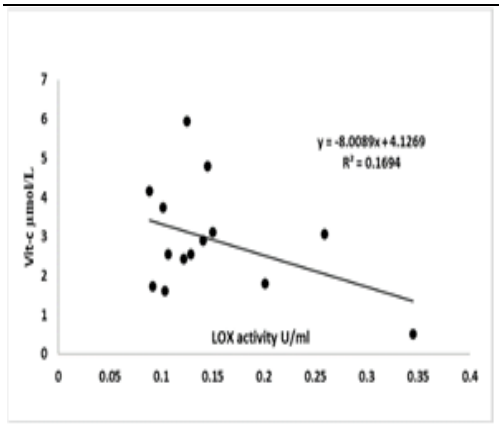

Fig5 : Correlation between LOX activity and Vit-E in patients

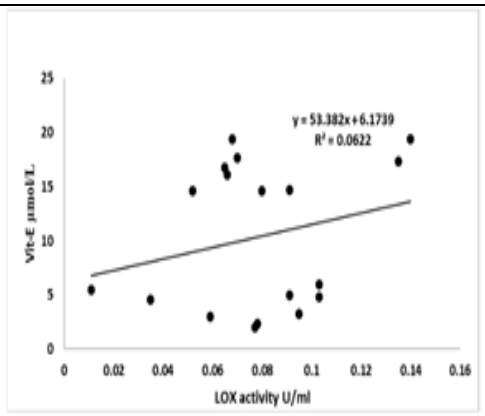

Fig6: Correlation between LOX activity and Vit-e in healthy

By study the effect of age on the biochemical variables in epilepsy and control group, the results in table (7) showed no significant difference in the level of lipooxygenase enzyme activity and all parameters

Table 7: Effect of age on the parameters in epilepsy patients

Parameters	Epilepsy patients Age 15-25 Mean ± S. E	Age 26-35 Mean ± S. E	*p≤0.05*
LOX[U/ml]	0.18±0.03	0.15±0.02	n.s
PAO[U/m]	0.21±0.10	0.09±0.01	n.s
Glucose [mmol/L]	5.82±0.04	5.96±0.5	n.s
Uric acid[mg/dl]	4.08±0.60	3.97±0.93	n.s
Bilirubin [µmol/L]	6.46±2.06	7.80±2.42	n.s
Total protein [mg/ml]	78.02±6.19	78.38±6.28	n.s
Albumin [mmol/L]	0.92±0.17	0.96±0.19	n.s
MDA [µmol/L]	6.48±1.41	5.72±1.80	n.s
GSH[µmol/L]	1.94±0.38	1.4±0.32	n.s
]mol/L[µVit.E	2.75±0.47	3.41±0.51	n.s
]g/ml[µVit.C	48.32±6.13	57.48±10.6	n.s
T.C [mmol/L]	6.12±0.56	6.55±0.80	n.s
T.G [mmol/L	1.69±0.41	1.61±0.35	n.s
HDL-C [mmol/L]	2.22±0.40	2.12±0.63	n.s
VLDL-C [mmol/L]	0.34±0.08	0.32±0.06	n.s
LDL-C [mmol/L]	4.17±0.62	4.01±0.77	n.s

S= significant, n.s= non-significant

Conclusion: In this study found a significant increase of LOX activity in epilepsy patients compared healthy, and a clear negative relationship with uric acid, bilirubin, MDA vitamin E and BMI.

There was no effect of sex except for LOX enzyme and uric acid in addition to albumin, also was no effect of age on all the measured variables

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