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Estimation and Evaluate of uric acid , Cholesterol and Triglycerides Levels in Hypertension patients in Basra Governorate – Iraq

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ABSTRACT

The study included the measurement of the mean serum Uric acid, Cholesterol, Triglycerides levels in hypertension patients in Basra Governorate – Iraq. In this study a total number of (80) hypertensive patients (41 males, 39 females) classified according to (age, sex, family history, severity and smoking) as compared with (60) healthy (34 males, 26 females) subjects were investigated as control group. It was found that a highly significant increase is in uric acid, triglycerides and cholesterol in the blood of hypertensive patients as compared with healthy subjects. In accordance with age, severity, family history and smokers. There were significant changes in these variables between males and females. In conclusion hypertensive patients suffer a high degree of reactive oxygen species (ROS) formation causing considerable oxidative stress indicated a high levels of the serum uric acid, triglycerides and cholesterol.

Keywords: *MDA: Malondialdyhyde, ROS: Reactive oxygen species, UA: Uric acid, HT: Hypertension, Cholesterol, Triglyceride*

1. INTRODUCTION:-

Hypertension defines as elevation of blood pressure in value equal to 140/90 mmHg. However ,the incidence of cardiovascular disease particularly stroke is closely related to average BP at all ages In > 95% of cases , no specific underlying cause of hypertension can be found and such patients are said to have essential hypertension can be include (Genetic factor , high salt intake , Alcholexcess, obesity Lack exercise) .In-5% of cases hypertension results from a specific underlying disorder (secondary hypertension) causes include renal disease ,cystic kidney disease, endocrine disorders ,Pregnancy and drugs(corticosteroids, estrogen ,and bolicsteroids) . [1]

Uric acid is a naturally occurring of Purina metabolism [2] Urates are the ionized from of uric acid predominate in plasma extracellular fluid. uric acid production varies with Purina content of the diet and the rates of Purina biosynthesis and degradation .Normally two-thirds to three -

fourths of uric acid are excreted by kidneys and most of the remainder is eliminated through the intestines [3] uric acid is also found within cell and in all body fluids at Lower concentration than in plasma where it exists mostly in its ionized from urate at physiological pH [4]. Humans have no enzyme for further oxidation of uric acid so an access of uric acid is excreted by kidney. In normal condition the rate of synthesis of uric acid equals the rate of its consumption and excretion [5]. At physiologic pH, urate is a strong reducing substance, electron doner, and antioxidant, contributing half of the plasma antioxidative capacity [6] . Cholesterol is a type of steroid and an important molecule in animals, which accounts for most all of the sterol in the plasma and exists as mixture of unesterified (30%-40%) and esterified (60% -70%) forms [7]

Cholesterol is present in tissues and plasma, either as free cholesterol or storage a combined with Long-term fatty acid as cholesterol ester. In plasma both forms are transported in lipoprotein cholesterol and it is biochemically synthesized in many tissues from Acetyl-COA .A little more than half of the cholesterol of the body arises by synthesis (about 700 mg /day) and the remainder is provided by the average diet. The liver and intestine account for approximately 10% each of total synthesis in humans. [7]

Triglycerides are the most common lipid classes in nature which also Know as triacylglycerol are non charged esters of glycerol with three fatty acids, which are the major storage and transport form of fatty acids [8]. Liver and adipose tissues are also major sites of triacylglycerol synthesis, those synthesized in the liver being secreted into plasma as endogenous triacylglycerols; triacylglycerols are also transported from the liver in the form of VLDL to extra hepatic tissue. [9] The present study was undertaken to evaluate the uric acid, cholesterol, triglycerides concentrations in the serum of hypertension patients with several parameters (age, sex, and severity, smoking and family history) in Basra Governorate –Iraq.

2. Material and Methods

2.1 Samples

Eighty patients (41 males and 39 females) whose age ranged between (25-65) years for males and females are divided into three groups (25-40) years, (40-60) years, (>60) years. The following information was recorded for high blood pressure patients(age, sex smoking, family history and severity) Those patients were classified according to the severity into three groups (Mild , Moderate and Sever) sixty healthy subjects (34males and 26 Females) were investigated as a control group aged between (25-65) years blood samples (5mL) were collected from patients and healthy control by vane puncture using a sterile disposable syringe in plain plastic tube The blood was centrifuged at 3000 rpm for 10min the serum was collected and frozen at -20 C for estimation of uric acid and Triglyceride and Cholesterol.

2.2 Instruments

1- UV / VIS spectrophotometer APEL Japan
2- Centrifuge, HettichEBA 20 , Germany
3- Vortex stirrer, Gallen Kamp, Germany model SGP-301.
4- Water bath, Gallen Kamp, Germany, model YCW-01.

2.3 Estimation of serum uric acid

Uric acid was assayed enzymatically by uricase method using a kit from France biomerix (No.02160) depending on enzymatic method of Artctiss and Entwistle which is based on the following principle [10]

2.3.1 Principle

Uricase acts on uric acid to produce allantion, hydrogen peroxide and carbon dioxide. Hydrogen peroxide in the prescience of peroxides reacts with a chromogen (4-amino antipyrine and 3, 5 dichoro-2-hydroxybenzene suffocate) to yield quinoneimine a red colored complex. The absorbance measured at 520 nm is proportional to the amount of uric acid in the sample.

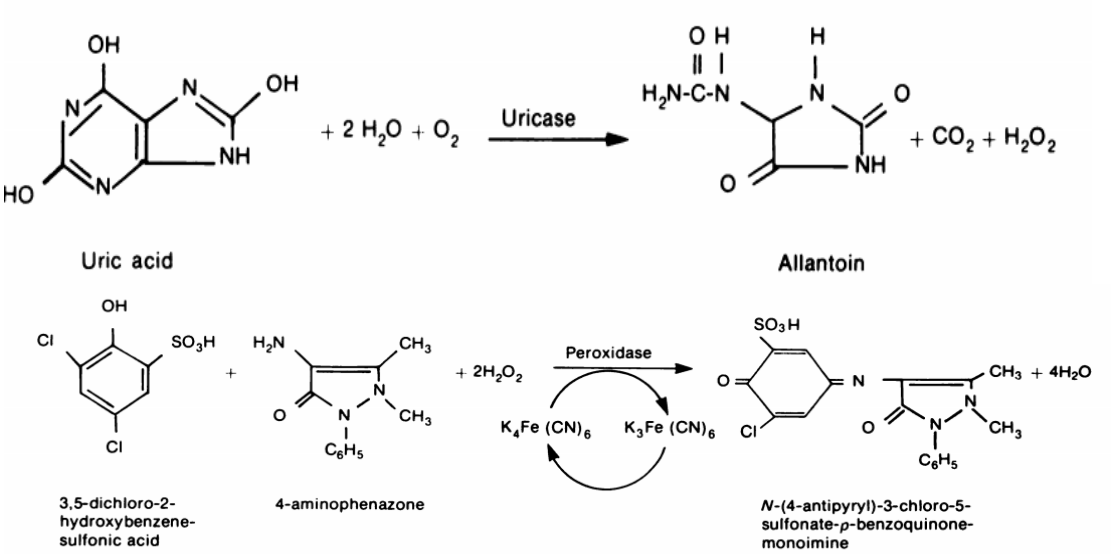


Figure (1): Chromogen (4-amino antipyrine and 3,5-dichloro-2-hydroxybenzene suffocate)(1) to form a stable, red colored N-(antipyryl)-3-chloro-5-sulfonate-p benzoquinone-monoimine (II)

2.4 Estimation of serum Triglyceride

The level of serum triglycerides was measured using the kit prepared by the France firm Biolabo. [11]

2.4.1 Principle

This method is based on the following interactions: Analysis of enzymatic triglyceride to glycerol.

1- Triglycerides are hydrolyzed by lipase enzyme to glycerol and fatty acids.

$$\text{Triglycerides} \xrightarrow{\text{Lipase}} \text{Glycerol + free fatty acid}$$

2- Glycerol is then phosphorylated to glycerol-3-phosphate by ATP in a reaction Catalyzed by glycerolkinase (GK).

$$\text{Glycerol + ATP} \xrightarrow{\text{Glycerol kinase}} \text{Glycerol -3- Phosphate + ADP}$$

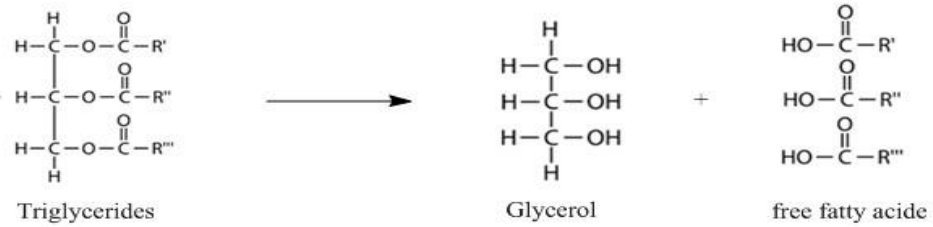
3- The oxidation of glycerol-3-phoshate is catalyzed by glycerol phosphate oxides (GPO) to form dihydroxyacetone phosphate and hydrogen peroxide

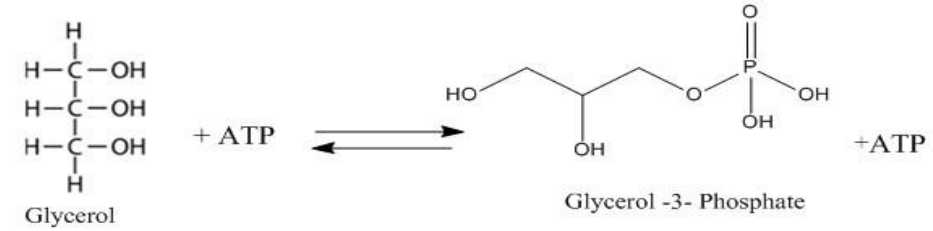
$$\text{Glycerol-3- Phosphate} + O_2 \xrightarrow{\text{Glycerol 3-P- oxidase}} \text{Dihydroxyacetone Phosphate} + H_2O_2$$

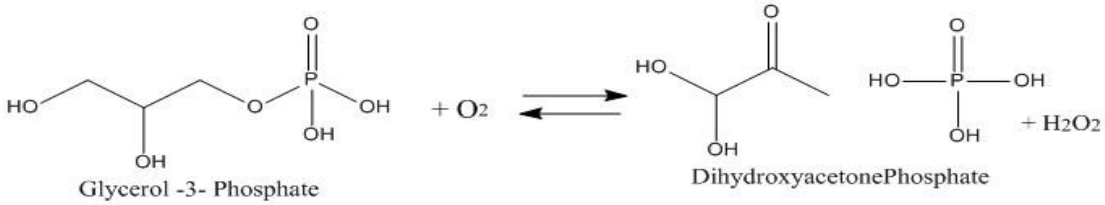
4- In the presence of peroxides (POD), hydrogen peroxide affects the oxidative Coupling of 4-chlorophenol and 4-aminoanti-quinonemine .

$$H_2O_2 + \text{4-Chlorophenol} + \text{PAP} \xrightarrow{\text{Peroxides}} \text{Quinoneimine (pink)} + H_2O$$

.







2.5 Estimation of serum Cholesterol

Measure the cholesterol level in the Serum using the Kit prepared by the France firm Biolabo[12]

2.5.1 Principle

Cholesterol concentration was measured by the enzymatic method described by Allain

1- Cholesterol esters are enzymatically hydrolyzed by cholesterol esterase (CE) to cholesterol and fatty acids

$$\text{Cholesterol esters} \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{free fatty acids}$$

2- Free cholesterol is then oxidized by

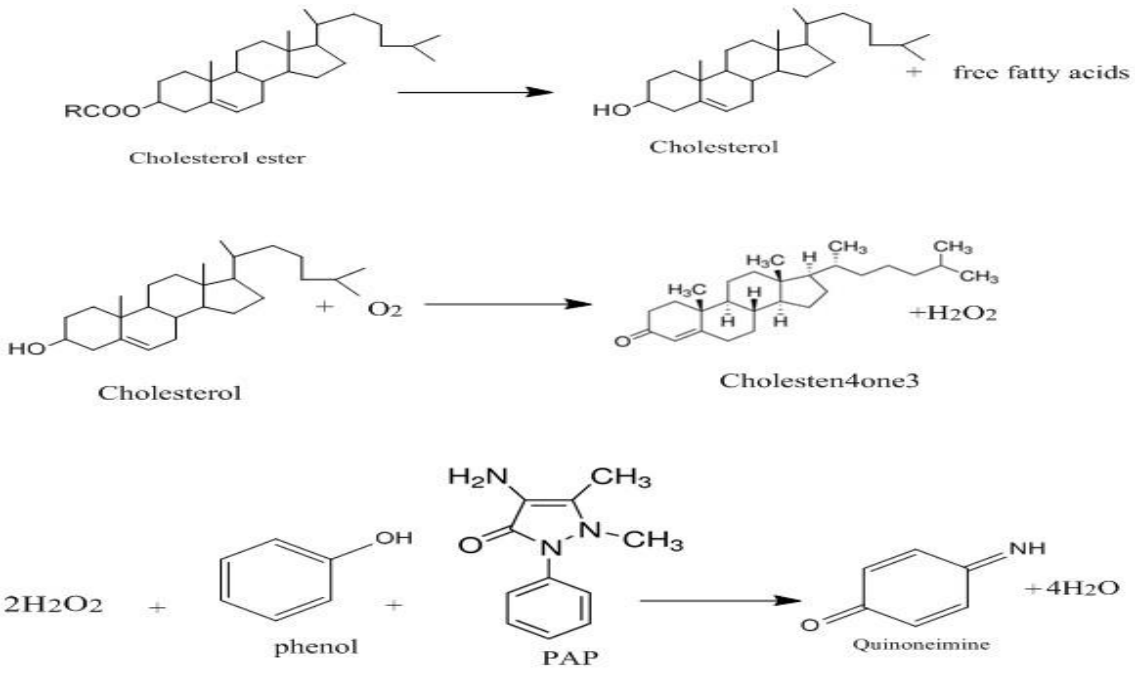
Cholesterol oxides (CO) to cholesten-4-one-3 and hydrogen peroxide.

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholesten 4 one 3} + H_2O_2$$

3- The hydrogen peroxide combines with phenol and 4-amino-antipyrine

(PAP) in the presence of peroxides (POD) to form quinoneimine dye.

$$2H_2O_2 + \text{Phenol} + \text{PAP} \xrightarrow{\text{Peroxides}} \text{Quinoneimine (pink)} + 4H_2O$$



3 .Procedure

Uric acid

To 25 µL of serum sample (T), standard uric acid (S) and distilled water for reagent blank (B), add 1 ml of the working solution were added, mixed and incubated at 25 °C for 5 minutes. Then the absorbance of serum and standard were recorded at 520 nm against the reagent blank (B).

Triglycerides

To 10 µL of serum sample (T), standard triglyceride (S) and distilled water for reagent blank (B), add 1 ml of the working solution were added, mixed and incubated at 37 °C for 5 minutes. Then the absorbance of serum and standard were recorded at 500 nm against the reagent blank (B).

Cholesterol

To 10 µL of serum sample (T), standard cholesterol (S) and distilled water for reagent blank (B), add 1 ml of the working solution were added, mixed and incubated at 37 °C for 5 minutes. Then the

absorbance of serum and standard were recorded at 500 nm against the reagent blank (B).

4. Calculations

Uric acid

The concentration of uric acid

$$(\text{mg/dL}) = \frac{AT}{AS} \times 10$$

AT: The absorbance of the sample serum.
AS: The absorbance of the standard.
10: the concentration of the standard in Mg/dL.

Triglyceride

The concentration of triglyceride

$$(\text{mg/dL}) = \frac{AT}{AS} \times 200$$

AT: The absorbance of the sample serum.
AS: The absorbance of the standard
200: the concentration of the standard in mg/dL

Cholesterol

The concentration of cholesterol

$$(\text{mg/dL}) = \frac{AT}{AS} \times 200$$

AT: The absorbance of the sample serum.
AS: The absorbance of the standard
200: the concentration of the standard in mg/dL

5. Statistical Analysis

The results were expressed as mean ± stander deviation (SD). The data was analyzed by one-way analysis (ANOVA) while the correlation between the data was tested statistically by simple linear test using computer SPSS program (No.24). was P -value less than 0.01 as highly significant. considered as statistic

6. Results and Discussion

Hypertension is one of the most important risk factor of stroke cardiovascular disease, renal disease and the risk of morbidity. The death increases as blood pressure exceeds the optimal levels. The present study revealed a highly significant increase ($P<0.01$) in uric acid serum level in all hypertension patients as compared to healthy control. These significant changes increase with age, sex, severity, smoking and family history. The mechanism by which elevated serum uric acid (SUA) levels in duce hypertension remains elusive. Oxidative stress, inflammation nitric oxide (NO) production impairment, vascular endothelial dysfunctions vascular smooth muscle proliferation, and rennin angiotensin system enhancement have been reported as mechanism for developing hypertension by hyperuricemia [13-14]. Crystallization of (UA) it self has also been reported to cause inflammation gouty kidney, and urinary tract calculi, and progression to renal failure[15-16].

In developing hypertension by hyperuricemia, two phase would be conceived. In the first phase inhibition of NO production and activation of the rennin-angiotensin system by hyperuricemia causes excessive vasoconstriction resulting in hypertension. This process is reversible and is a (UA) - dependent reaction where blood pressure can be reduced by lowering (UA). [17-18].

In the second phase, when hyperuricemia persists , the proliferation of vascular smooth muscle cells thickness vessel wall and increases blood pressure because of changes in the vascular structure. This step is(UA) independent and blood pressure dose not return to the original level any more by lowering (UA) as in the first phase [17-19].

In this study stronger relationship between (SUA) level and hypertension developed in subjects aged >60 years and The positive correlation between elevated (SUA) and hypertension has .The level in UA in male is higher than female this elevation could be due to the renal excretion or urate which may be affected

by sexhormone level These results are in agreement with the results previously reported [20-24].

on the other hand , there was significant difference in (UA) level between smokers and no smokers (P<0.05) as illustrated in(Table 1, Fig 2).Cigarette smoke contains high amount of free radicals such as superoxide anion $(O_2)^-$ and nitricoxide (NO) [25].,gas phase can react chemically to form highly reactive free radical peroxynitrite . In addition superoxide anion can react with hydrogen peroxide to form the more active hydroxyl free radical [26] Many researchers have reported increased level of superoxide anion from circulating neutrophils and the (SUA) level as illustrated in (Table 1 , Fig 2) . In this study we found a highly significant increase (p<0.01) in the Previous studies [27-29] that show an increase in the prevalence of dyplipidemia in hypertension patients . Also in our study we elevated the relation between serum cholesterol and risk factor. The increase in cholesterol level among hypertensive patients were found in correlation with older age group , more In female than male , sever hypertension . Age hypertension and hypercholesterolemia have a multicative effect on CVD risk and thought to be mediated through oxidative stress and endothelial dysfunction [30]. High cholesterol level in female is attributed to the lipid per oxidation as result of low level of sex hormones, which are important factor that act as antioxidants to protect against the oxidation of lipid [31]. Also a significant increase in cholesterol level between smoker non smoker hypertension patients (p<0.01) as illustrated in (Table 2.Fig3). The reason increased level of superoxide anion from circulation neutrophils and increased lipid peroxidation product malondialdhyde (MDA) in the plasma of smoker, supporting the concept of oxidative stress in these individuals

[27]The result of this study report that increase levels of (UA) with severity (p<0.05) in hypertension patients explained to the relationship between hypertensive severity to the extent of lipid peroxidation. [26]

Also Family history seems to influence the levels of (UA) as compared to positive and negative family history of hypertensive patients , there were a highly significant increase (p<0.01) in cholesterol serum level in hypertensive patients as compared to control group. This finding goes with may be due tube smoking that can cause free radicals and cause damage to DNA and proteins therefore smokers had high oxidative stress and posses a low level of anti oxidants [32]

on the other hand , there was a significant difference in cholesterol level between positive and negative family history (p<0.05) observed in this study as illustrated in (Table 2.Fig3) This study raveled a highly significant increase (p<0.01) in Triglyceride serum level in the hypertensive patients as compared to healthy control . In accordance with age, sex, and severity , serum level increases significantly(p<0.05) while with family history patients a highly significant increase is (p<0.01) as indicated in (Table 3,Fig4) These imply that hypertensive patients are exposed to considerable degree of lipid peroxidation , suggesting that oxidative stress affects simultaneously on lipid peroxidation [30]

Table (1): Serum level of Uric acid (mg/dL) in Hypertension patients classified according to (Age, Sex, Severity, Smoking and Family history) and healthy control.

Variable		Uric acid concentration(mg/dL) hypertension patients (80)		Uric acid concentration(mg/dL) control (60)		P – value
		No	Mean ±SD	No	Mean ±SD	
Age	25-40	15	5.19±0.72	33	2.97±0.44	P<0.01
	40-60	35	6.44±1.16	17	3.08±0.64	
	>60	30	8.32 ± 1.66	10	4.09± 0.76	
Sex	Male	41	7.23±1.89	39	3.44±0.68	P<0.01
	Female	34	6.66±1.59	26	3.11±0.70	
Family history	Positive	45	7.14 ± 1.61			P<0.01
	Negative	35	5.96 ± 1.55			
Smoking	Positive	24	7.35 ± 1.90	38	3.53± 0.72	P<0.05
	Negative	56	6.29 ± 1.53	22	3.34± 0.93	
Severity	Mild	17	5.31± 1.11			P<0.01
	Moderate	24	6.77 ± 1.32			
	Sever	39	8.01 ± 1.74			

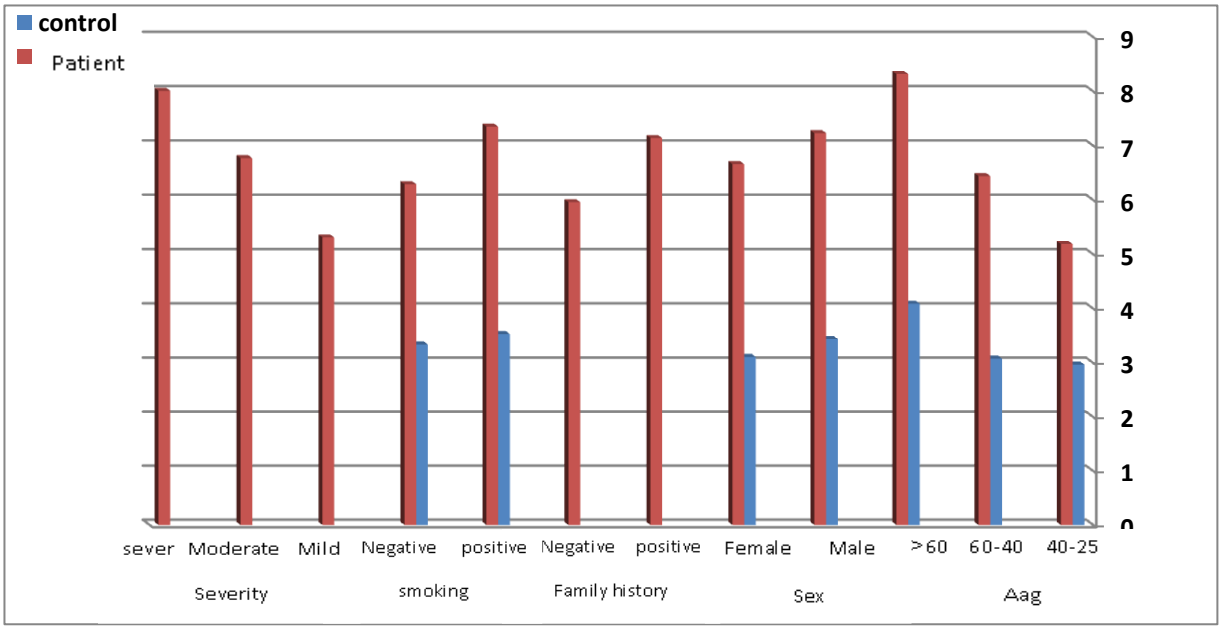


Figure 2. Serum Uric acid concentration (mg/dL) in Hypertension patients and healthy control classified according to (Age, Sex, Severity, Smoking and Family history).

Table (2): Serum level of Cholesterol (mg/dL) in Hypertension patients classified according to (Age, Sex, Severity, Smoking and Family history) and healthy control.

Variable		Cholesterol concentration(mg/dL) hypertension patients (80)		Cholesterol concentration(mg/dL) control (60)		P – value
		No	Mean ±SD	No	Mean ±SD	
Age	25-40	15	151.07 ±17.09	33	123.62 ± 9.38	P<0.01
	40-60	35	190.68± 25.49	17	129.58 ± 9.89	
	>60	30	199.71 ±20.63	10	134.18 ± 9.57	
Sex	Male	41	186.83 ± 31.60	39	126.43 ± 10.02	P<0.01
	Female	34	189.29 ± 24.32	26	128.02±10.69	
Family history	Positive	45	186.98 ± 25.40			P<0.05
	Negative	35	179.59±26.37			
Smoking	Positive	24	185.92 ± 29.67	38	187.53 ± 16.33	P<0.01
	Negative	56	126.92 ± 8.99	22	126.63± 11.91	
Severity	Mild	17	164.96± 28.93			P<0.01
	Moderate	24	187.43 ± 26.43			
	Sever	39	195.56 ± 25.64			

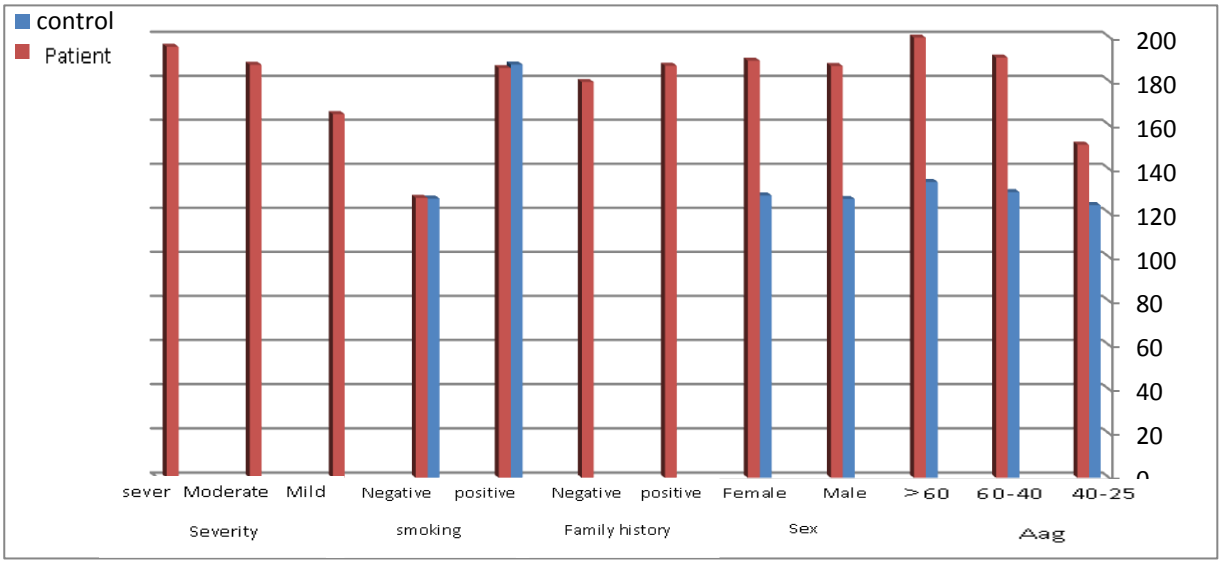


Figure3: serum Cholesterol concentration (mg/dL) in hypertension patients and healthy control classified according to (Age, Sex, Severity, Smoking and Family history).

Table (3): Serum level of Triglyceride (mg/dL) in Hypertension patients classified according to (Age, Sex, Severity, Smoking and Family history) and healthy control.

Variable		Triglyceride concentration(mg/dL) hypertension patients (80)		Triglyceride concentration(mg/dL) control (60)		P – value
		No	Mean ±SD	No	Mean ±SD	
Age	25-40	15	133.96 ± 8.76	33	83.54 ± 21.90	P<0.01
	40-60	35	149.41±17.24	17	84.21± 22.38	
	>60	30	155.58 ±39.04	10	100.58 ±17.28	
Sex	Male	41	150.47 ± 30.30	39	84.37± 21.14	P<0.01
	Female	34	142.67 ± 22.37	26	83.59± 20.55	
Family history	Positive	45	156.79 ± 21.44			P<0.01
	Negative	35	139.25 ± 17.51			
Smoking	Positive	24	149.31 ± 24.58	38	87.19± 22.84	P<0.05
	Negative	56	148.16± 29.18	22	81.13±19.59	
Severity	Mild	17	138.51 ± 14.07			P<0.01
	Moderate	24	142.65 ± 22.67			
	Sever	39	154.72 ± 33.10			

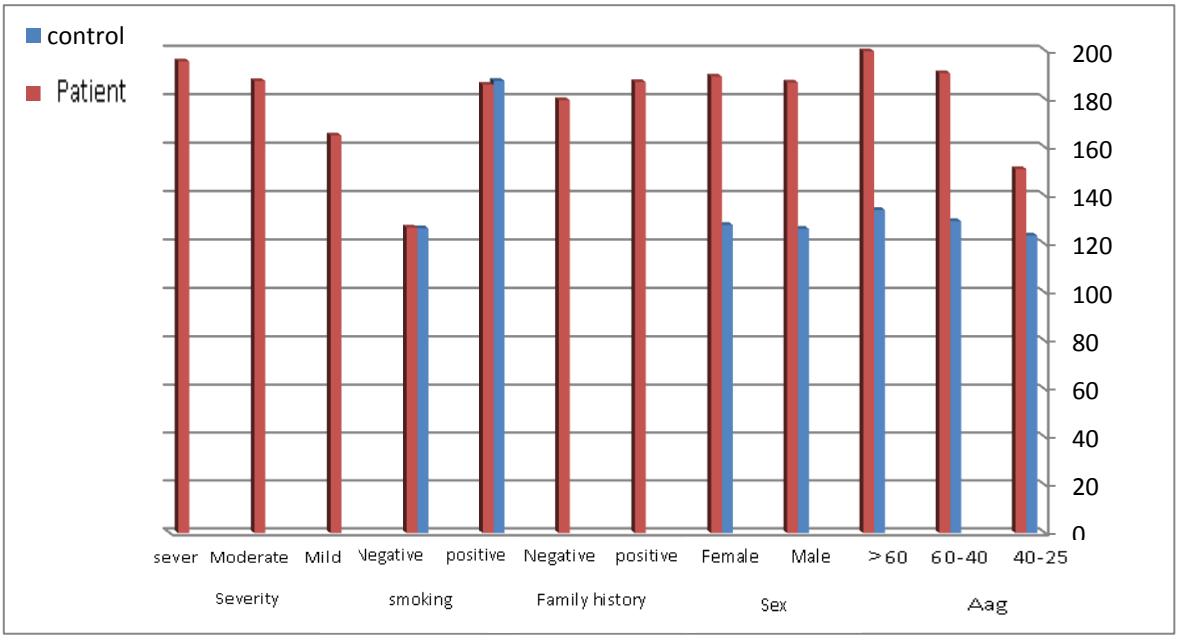


Figure 4 Serum Triglyceride concentration (mg/dL) in hypertension patients and healthy control classified according to (Age, Sex, Severity, Smoking and Family history).

Conclusion

Hypertension patients suffer a high degree of reactive oxygen species (ROS) formation causing considerable oxidative stress indicated by a high level of the serum uric acid, triglyceride and cholesterol.

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تقدير وتقييم مستويات حامض اليوريك ,الكولسترول و الكليسريدات الثلاثية في مرضى ارتفاع ضغط الدم في محافظة البصرة – العراق

ساهرة غريب صياح- حنين صلاح خليل

قسم الكيمياء – كلية التربية للعلوم الصرفة – جامعة البصرة – بصرة –العراق

الخلاصة

تضمنت الدراسة قياس معدل مستوى حامض اليوريك , الكليسيريدات الثلاثية ,الكولسترول لمرضى ارتفاع ضغط الدم في محافظة البصرة حيث تضمنت الدراسة (80)حالة مرضية (41 الذكور ,39 إناث) تم تقسيم المرضى على وفق مجموعة متغيرات تشمل العمر,الجنس, التاريخ العائلي , شدة المرض ,التدخين مقارنة مع (60) حالة للأصحاء (34 الذكور ,26 إناث) بينت الدراسة ارتفاع معنويا في مستويات حامض اليوريك , الدهون الثلاثية والكولسترول في دم مرضى ارتفاع ضغط الدم مقارنة مع الأصحاء ويزداد هذا الارتفاع بتقدم بالعمر , شدة المرض ,التاريخ العائلي ,ومع المدخنين ونستنتج من خلال نتائج هذه الدراسة بان لدى المرضى المصابين بارتفاع ضغط الدم درجة اعلي من تكوين الأنواع النشطة للأوكسجين والتي تسبب إجهاد مؤكسدا وزيادة في مستويات حامض اليوريك والدهون الثلاثية والكولسترول .

الكلمات ألمفتاحيه : المالون داي اليهايد, الأنواع النشطة للأوكسجين , حامض اليوريك , ضغط الدم , الكولسترول , الكليسريدات الثلاثية