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Effect of endothelin-1, Vimentin and some biochemical variables on men with type 2 diabetes mellitus, diabetic patients with hypertension, and diabetic patients with renal impairment

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Abstract

A comprehensive cross-sectional study is performed to assess the status of endothelin-1, Vimentin, and some biochemical variables. The study has included 140 blood samples from men, divided into four groups: Group 1 type 2 diabetes mellitus DM, group 2 type 2 diabetes mellitus and DM +P hypertensive patients, and group 3: type 2 diabetes mellitus and DM patients with - Renal insufficiency + R and the control group (C). Each group included 35 samples, and their ages ranged between (30-70) years. Samples are collected from patients and healthy people from Samarra General Hospital, outpatient clinics and Dalaza Hospital in Tikrit between 20/12/2020 - 20/4/2021. The current study includes measurement of body mass index, determination of serum endothelin-1, vimentin blood protein, fasting glucose-FSG, urea, creatinine, uric acid, and serum Hba1c measurement. The results indicated that the BMI value increased significantly (P≤0.05) in the DM and DM + P groups, while it decreased in the DM + R group compared with the control group, the FSG concentration increased significantly in groups of patients compared to the control group. , the Hba1c concentration increased significantly in the DM and DM + P groups, while DM + R did not show any significant difference compared to the control group, and the results showed that the urea concentration increased significantly in DM + P and DM + R, while DM did not show significant differences Statistic. Compared with the control group, while the creatinine concentration increased significantly in DM + R while DM and DM + P did not show any significant differences compared to the control group, the results also indicated that the uric acid concentration increased significantly in all patient groups compared to the control group. The concentration of nitric oxide decreased significantly in all groups of patients compared to the control group. The results also indicated that the concentration of Endothlen-1 increased significantly in all groups of patients compared to the control group, while the concentration of vimentin did not register any significant differences. In all patient groups compared to the control group. We concluded from the current study that hypertension with diabetes, pre-diabetes and kidney had a clear effect on biochemical parameters, while diabetes alone had a less pronounced effect.

Introduction:

Diabetes mellitus is a metabolic disorder characterized by hyperglycaemia and insufficient secretion or action of endogenous insulin [1]. High blood sugar in type 2 diabetes leads to a gradual development of complications including polyneuropathy, retinopathy, atherosclerosis, and many complications affecting blood vessels, kidneys, retina, lens, peripheral nerves and skin are common and costly. Extremely longevity and quality of life. Increased oxidative stress, which is an important cause of the development of diabetes and its complications [2], where the cells of the body become less responsive to the action of insulin, and the inability of muscles and fatty tissues to use insulin appropriately, or that the pancreas sometimes does not produce enough insulin Due to the body's need, most patients of this type suffer from obesity, and obesity in itself causes a degree of resistance of the body tissues to insulin, so this type of disease is characterized by the presence of insulin accompanied by a defect either due to the lack of secretion of the pancreas or a defect in its receptors, which leads to a lack of control over the body.

The level of glucose in the blood. Ketone bodies are rarely formed in this type of diabetes [3,4]. A case of high blood pressure is known when the value becomes greater or equal to (140/90) mm Hg continuously [5] Diabetes and high blood pressure are two concomitant diseases where high blood pressure is one of the dangerous factors, and advanced to diabetes [6]. Several studies have shown that high systolic pressure and diastolic pressure in diabetic patients; As a result of the increase in pressure on the glomerulus, it is offset by an increase in the protein micro albuminuria and a decrease in Creatinine clearance. [7] Chronic renal insufficiency is defined as a permanent and continuous deficiency and deterioration in the functions of the kidneys; It leads to a loss of its excretion, metabolic and hormonal activity [8]. "About 30% of patients with type 2 diabetes develop clinically overt nephropathy [9]. Studies have found that about 65% of chronic renal failure patients suffer from diabetes [10]. A study also indicates that about 38% of people with diabetes will subsequently suffer from decreased kidney function [11].

Nitric oxide NO is a relatively stable free radical that spreads easily from the production site and crosses cell membranes without the need for special carriers or receptors. [12,13]. It has a short half-life (a few seconds) and a short range of biological activity. Nitric oxide is the least molecular by weight among the biologically active molecules secreted by mammalian cells. Nitric oxide is an important molecule in the body's defense against diseases, as well as harmful and causing a wide range of diseases such as infections and autoimmune diseases [14].

Vimentin (Vim) is a filamentous protein with a molecular weight of approximately 57 k Dalton. [15]. It is the cellular component responsible for maintaining the integrity of the cell, and it is the most prevalent intermediate filamentous protein. And the first in formation in the stage of cell differentiation. It is formed within a wide variety of mesenchymal cells (fibroblasts), and in a number of other mesodermal cell types, in lymphocytes and phagocytes it produces Vimentin in very small amounts. Vimentin is found in cells derived from mesoderm, such as the kidney in Bowman's capsule and endothelium. It is also known to be an important marker for early detection of cancers such as bladder cancer, hepatocellular

carcinoma, and colorectal cancer [17,18]. Many malignancies, including gastric cancer, colorectal cancer, cervical cancer, and bladder cancer [17].

Creatinine is a muscle-wasting end product of Creatinine. It results from the breakdown of Creatinine phosphate in muscle at a constant rate (male 15-25 mg/kg bw/day female 10-20 mg/kg daily) and is often excreted by filtration without reabsorption. The tubules also play a role in the secretion of Creatinine [19]

Urea is a small water-soluble molecule with a molecular weight of 60 Da. It contains two nitrogen atoms and is the end product of protein-nitrogen metabolism. Urea is the compound with the highest concentration found in the blood of urea plasma patients. A less useful measure of kidney function because first, the rate of production is not constant (increases with high protein diets, tissue breakdown . Steroids, bleeding, decreased liver disease and low protein diets). Second, 40-50% of the filtered urea is reabsorbed into the proximal tubules [20].

Subject and Method

Sample collection and study design: the study was conducted on 140 blood samples (male), 35 samples for type 2 diabetes patients, 35 samples for type 2 diabetes patients and those with hypertension, 35 samples for patients with type 2 diabetes and those with kidney disease and 35 a sample of healthy individuals as a control group, age group (30-70) years. patient samples were collected from outpatient clinics in Samarra general hospital from the period between 20/12/2020 - 20/4/2021.

Serum samples were obtained from the blood collected after centrifugation and used to determine the biochemical parameters under investigation. 2 ml of blood was taken into anticoagulant tubes to make Hba1c. The total samples collected were divided into three categories:

Control group (C): It consists of 35 samples of healthy individuals

First Group DM (G1): It consists of 35 patients with type 2 diabetes.

The second group DM+P (G2): which consists of 35 samples of patients with type 2 diabetes and those with high blood pressure.

The third group DM+R(G3): which consists of 35 samples of patients with type 2 diabetes and those with kidney failure.

Methods: The current study includes identifying: Body mass index (BMI) was measured by measuring the body height and weight of sick and healthy people using a height scale in units of centimeters (cm) and weight using a sensitive person scale in units of kilograms (kg). BMI was calculated according to the following formula: ((kg) weight) / (m2) height [21]

Serum Vimentin and serum endothlen-1 protein using, Vimentin and Endothlen-1 test kit by ELISA method supplied by Melsin Medical, China. And the measurement of glycaemic haemoglobin in the blood from Stan Bio America [22] - fasting blood glucose - SFG and measurement of urea and Creatinine in the blood serum using a colorimetric method according to the procedure of glucose, urea and Creatinine kit provided by the Tunisian company Bio-Maghreb [23-25].

Serum uric acid measurement from Bio LABO France [26]. The measurement of nitric oxide in the blood is carried out according to the method of researcher Dervisevic [27].

Statistical analysis: Analysis of variance (ANOVA) was used to compare the groups of type 2 diabetics, groups of type 2 diabetics and those with hypertension, groups of type 2 diabetics and those with kidney disease, and the healthy group as a control group.

Results and Discussion:

The present study includes the determination of blood Hba1c, Vimentin, endothelin-1, glucose, urea, Creatinin, uric acid and nitric oxide levels in serum of patients with DM in G1, patients with DM and hypertension in G2, and patients with DM and kidney disease. G3 compared to the control group. The results obtained from this study are summarized in the Table.

Table (1): Mean ± standard deviation of Hba1c, serum FSG, urea and Creatinine In the four groups

Groups	BMI km/cm²	Glucose mg/dl	Hba1c %
Control	26.396±2.741 c	31.542±15.131 c	5.505±0.441 b
G1	28.509±3.820 b	328±43.611 a	9.762±1.812 a
G2	31.066±3.773 a	92.4857±46.815 a	9.18±1.753 a
G3	22.174±2.535 d	132.6±41.854 b	5.512±0.946 b

* Different letters indicate statistically significant differences, while similar letters do not have statistically significant differences.

*DM refers to the group of patients with type 2 diabetes.

* DM + P refers to the group of patients with type 2 diabetes, and hypertensive patients.

*DM + R refers to the group of patients with type 2 diabetes, and patients with kidney disease.

BMI

Table 1 shows the mean ± SD of BMI level (28.509 ± 3.820) kg/cm² in group DM, (31.066 ± 3.773) kg/cm2 in group DM+P, (22.174 ± 2.535) kg/cm² in group DM+R. compared The healthy subjects in the control group (26.396 ± 2,741) kg/cm². The results indicated that the level of body mass index in the blood increased significantly (P≤0.05) in the groups DM and DM+P, while it decreased in the DM+R group compared with the control group. shape 2

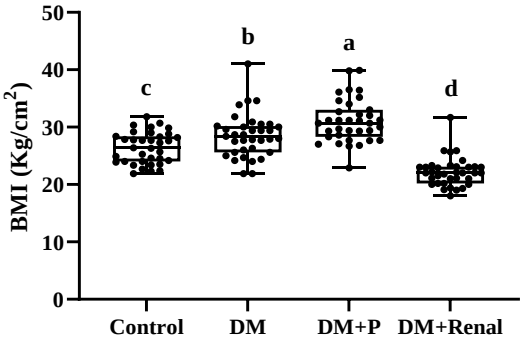

Figure 1: Measurement of BMI in the four groups.

Through the study, it was found that there is an effect of diabetes on body mass, as the BMI value increased with the diabetes, diabetes and pressure groups, as obesity has an effect on type 2 diabetes, the relative risk of T2DM increases with an increase in body mass index above 23 [28]. The risk of developing diabetes increases linearly with BMI; The prevalence of diabetes increased from 2% in those with a BMI of 25 to 29.9 kg/m, to 8% in those with a BMI of 30 to 34.9 kg/m, and finally to 13% in those with a BMI of 25 to 29.9 kg/m Body greater than 35 kg/m [29]. So Obesity has become a global epidemic. The relationship between obesity and diabetes is based on complex genetic and environmental factors. Including behavioral changes and lifestyle changes that lead to an increase in body weight through eating a lot and not consuming energy. There are many studies that confirmed a rise in the body mass of diabetics and pressure patients, in a study that showed that the possibility of developing prediabetes and diabetes is higher among individuals who are overweight and obese compared to individuals who are not overweight [30].

It also increased significantly in the pressure and diabetes group, where a study found that there is a gradient of blood pressure increase with higher levels of BMI. It showed that BMI may cause a direct effect on blood pressure, regardless of other clinical risk factors. [31].
While body mass has decreased in diabetic and renal patients, this may be due to dialysis reducing body mass.

Glucose level

Table 1 shows the mean ± SD for serum glucose (175.828 ± 43611) mg/dl in the DM group, (192.4857 ± 46.815) mg/dl in serum in the DM+P group, (132.6 ± 41.854) mg/dl in serum in the The DM + R group. Compared with the healthy group, the control group (80.632 ± 22.378) mg/dl. The results indicated a significant increase in the level of glucose in the blood ($P \leq 0.05$) in all groups of patients compared with the control group, with no significant difference between the groups DM and DM+P despite the slight increase in the level of glucose in DM+P compared with DM. Figure 2.

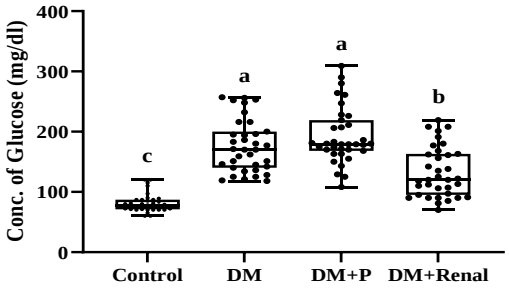

Figure 2: Serum glucose concentration in the four groups

The blood sugar level increased in diabetic patients in the DM group. The increase in sugar in this group is due to diabetes which causes insulin resistance, and therefore sugar builds up in the blood [32]. This high level of glucose is due to the inability of the pancreatic cells to

produce enough insulin [33], or a decrease. Tissue sensitivity to it, as diabetic patients suffer from metabolic disorders, and thus the level of glucose in the blood rises, and insulin cannot perform its function due to increased resistance by reducing its receptors on the surface of target cells [34] or due to a defect in the receptors of cells responsible for consuming It is believed that the cause of this imbalance is due to genetic factors or acquired factors as a result of a deficiency in the level of insulin or its absence, which leads to high blood sugar [35].

The result of the current study in DM agreed with the results of many researchers on how to assess the SFG of diabetes patients in Samarra [36-39], as well as T2DM patients with obesity, so the insulin secretion is insufficient as a result of obesity [40], as well as the high Sugar in DM+P agreed with Jaeger's study (and his group, where they reported a significant increase in blood glucose in hypertensive patients compared to healthy controls [41]. This may be due to the fact that high blood pressure is associated with intracellular insulin resistance, the resistance increases with the increase in fat and cholesterol in the blood, and the reason for these changes lies in the role of the hormone aldosterone secreted by the adrenal glands in raising blood pressure, as well as working to increase insulin resistance, which is the cause of type II diabetes [42,43] as well. Elevated DM+R Factors that make glycemic control in dialysis patients very difficult are due to therapeutic difficulties using anti hyperglycemics, monitoring difficulties, and dialysis exacerbating hyperglycemia. with blood [44].

Hba1c %

Table 1 shows the mean Hba1c ± SD estimation of glycosylated hemoglobin level (9.762 ± 1.812) in blood DM, (9.18 ± 1.753) in blood DM+P, (5.512 ± 0.946) in blood DM+R and (5.505 ± 0.441) in control group blood The results indicated that the level of Hba1c in the blood increased significantly (P≤0.05) in DM and DM+P, while DM+R did not show any significant differences compared to the control group with no significant difference between DM and DM+P in the level of Hba1c. Figure 3

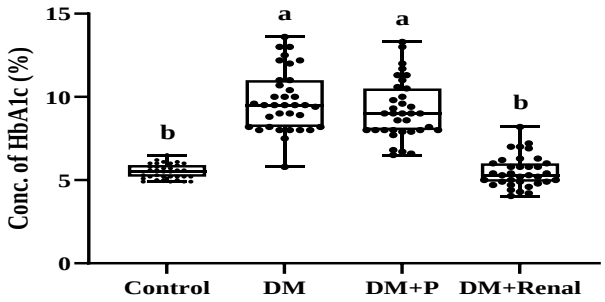

Figure 3: blood Hba1c Concentration in the in the four groups

The % of HbA1c increased significantly in the groups DM and DM+P in conjunction with the rise in the level of hemoglobin HbA1c with the level of glucose in the blood, and this came in support of what was obtained [45,46] and we believe that the reason for this is the existence of a relationship between the concentration of HbA1c formed and patients diabetes mellitus; The close relationship depends on the concentration of blood glucose to which the red blood cells are exposed during their time in the bloodstream, which in turn leads to the binding of the hemoglobin molecule. [47] These findings are also consistent with studies by a number of

researchers as well as many observations made by others, including [48-50]. This is also due to the fact that diabetics are exposed to increased oxidative stress. As a result of several reasons including spontaneous oxidation of glucose and non-enzymatic breakdown of glycoproteins., a spontaneous chemical reaction that occurs between glucose and amine groups in proteins, resulting in the formation of fatty bases and more stable amino compounds, Hba1c provides information on average blood glucose concentrations within 2- 3 months, such as indicated by [51] and its HbA1c group is an indirect measure of blood sugar and a guide to blood sugar levels over a 6-8 week period.

Table (2): - Mean ± standard deviation of serum urea, Creatinine and Uric acid in groups under investigation

Groups	Urea mg/dl	Criatinin mg/dl	Uric acid mg/dl
Control	6.265±5.355 C	0.854±0.146 B	5.166±1.058 C
G1	38.508±3.82 C	0.973±0.17 B	5.740±1.386 Bc
G2	91.066±3.773 B	1.169±0.372 B	5.991±1.518 B
G3	92.174±2.534 A	7.111±1.621 B	7.431±1.6367 A

Urea level

Table 2 shows the mean ± SD of serum urea level (30.371±6.193) mg/dl in serum DM, (38.657±9.152) mg/dl in serum DM+P, (119.885±32.816) mg/dl in serum DM+R and (26.265±5.355) mg/dl in the serum of the control group, the results indicated that the level of urea in the blood increased significantly (P≤0.05) in DM+P and DM+R , while DM did not show any significant differences compared to the control group Figure 4.

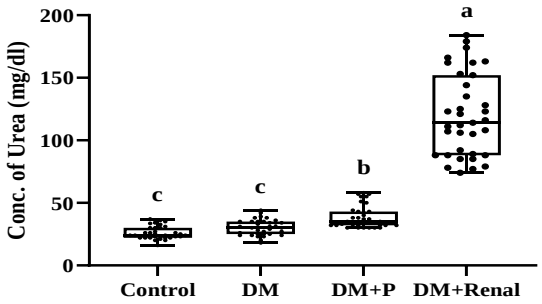

Figure.4: concentration of serum Urea in the four groups

The urea concentration did not rise significantly in the DM group, which may be due to the short duration of diabetes, as diabetes did not cause complications for the kidneys, and this contradicts the study of the researcher Chen 2009 [52].

While it rose in group DM+P. The results are in agreement with a study by Sumpio et al. That the urea concentration rises in patients with high blood pressure, which was attributed to the reason that high blood pressure leads to a failure to excrete urea due to the stimulation of angiotensin II, which causes an increase in the growth of smooth muscle in the lining of the vessels and thus reduces the filtration rate [53].

And it rose in DM+R that the high level of urea in patients with renal insufficiency is consistent with what was obtained by researcher Pillitteri. The reason for the high concentration of urea is attributed to the fact that urea is the basic nitrogenous substance resulting from metabolic waste that is formed and excreted to the outside through urine, and in the event of a defect and deficiency in the function of the kidneys This leads to a decrease in the excreted urea, which results in its accumulation and accumulation in the blood, and its concentration rises [54], and the lack of commitment of some patients to food regulation and their intake of high amounts of protein leads to an increase in their urea. [55].

The high level of urea in the blood of patients with chronic renal insufficiency and diabetes, its level is affected by the length of the disease and the lack of control it leads to the emergence of serious complications such as Diabetic Nephropathy, which causes a high rate of deaths, especially among young people with diabetes and that the lack of The efficiency of the kidneys results from an imbalance in the effectiveness of hormones such as angiotensin or a structural imbalance with a low number of nephrons or filtration area, which are related to the effectiveness of the glomerulus. [56].

Creatinine level

Table 2 shows the mean ± SD of serum Creatinine level (0.973±0.17) mg/dl in serum DM, (1.169±0.372) mg/dl in serum DM+P, (7.111±1.621) mg/dl in serum DM+R and (0.854±0.146) mg/dl in the serum of the control group. The results indicated that the level of Creatinine in the blood increased significantly (P≤0.05) in DM+R, while DM and DM+P did not show any significant differences compared to the control group. Figure 5

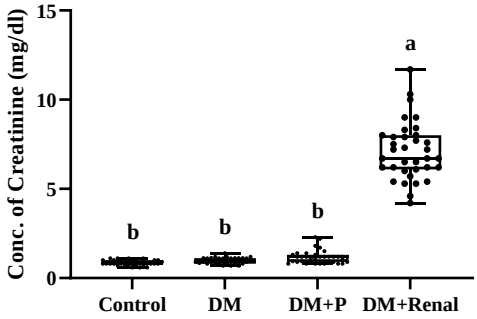

Figure.5: concentration of serum Creatinine in the four groups

The results did not show any significant differences between the groups DM and DM+P compared to the control group, while the DM+R group showed a significant increase. This is in agreement with a study by Rakopersingh et al on the relationship of both types of diabetes with chronic renal insufficiency that the increase in serum urea and Creatinine concentrations in patients with NIDDM is higher than in patients with IDDM. agrees [57] 'also agree with the study of Coachload et al [58], who found that serum Creatinine was very high in patients with chronic renal failure. It is possible that higher Creatinine and urea levels are evidence of decreased kidney function [59]. High blood urea causes severe disease unless it is removed from the bloodstream by the kidneys [60]. Decreased renal function can have an effect on the rate at which Creatinine is cleared by the kidneys and can be used as a measure of renal function. Because the kidneys are unable to filter Creatinine through urine excretion, kidney

function deteriorates resulting in increased serum Creatinine levels [61,62]. The reason for the high concentration of Creatinine in the serum of patients with chronic renal insufficiency may be attributed to the fact that Creatinine is a metabolic waste that is naturally excreted through diuresis. Glomerular filtration rate, a slight decrease in (GFR) leads to an increase in the concentration of Creatinine in the blood. [63], This increase in serum Creatinine level in patients with CKD is attributed to a decrease in the number of working nephrons, which reduces the glomerular filtration rate, which leads to a significant decrease in Kidney excretion of water and solutes. [64,65].

Uric acid level

Table 2 shows the mean ± SD of uric acid level (5.740 ± 1.386) mg/dl in serum DM, (5.991 ± 1.518) mg/dl in serum DM + P, (7.431 ± 1.6367) mg/dl in serum DM+ R compared to group Healthy subjects (5.166 ± 1.058) mg/dl in the serum of the control group. The results indicated that the level of uric acid in the serum increased significantly (P≤0.05) in all patient groups compared to the control group, with no significant difference between DM and DM+P despite the slight increase in the level of uric acid in DM compared with DM+P Figure 6.

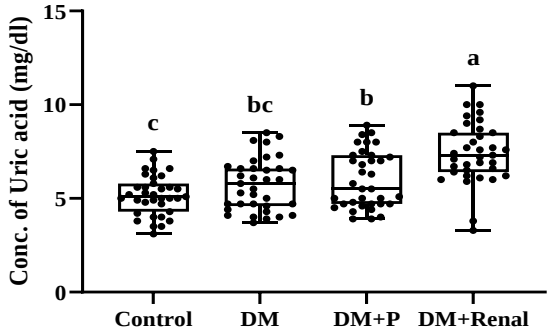

Figure.6: concentration of serum Uric acid in the four groups.

Uric acid increased in the DM group insignificantly as several clinical trials showed that uric acid was significantly associated with diabetes. As in an experiment, Bombelli et al. [66,67] They found that increased uric acid increased the risk of impaired fasting glucose (IFG) and serum uric acid was found to be an important predictor of the risk of metabolic syndrome, diabetes, and hypertension in adult males [68]. This is consistent with our results in group DM+P, where uric acid increased significantly in this group and the reason is that in recent years, human intake of foods (rich in purines), high added sugar (sucrose) and high fructose corn syrup has increased significantly. [69]. Fructose is the main component of added sugar. In contrast to other sugars, fructose can cause mitochondrial oxidative stress [70,71] and nucleotide turnover to a marked increase in serum uric acid [72]. In addition to causing gout, several studies have shown that hyperuricemia is closely associated with cardiovascular disease, metabolic syndrome, insulin resistance, and diabetes [73,74]. High levels of uric acid have been found to be closely associated with diabetes and its chronic complications.

UA levels are also associated with the occurrence and development of diabetic kidney disease and are risk factors for early kidney disease [75,76] and serum and urine albumin UA levels were significantly positively associated with kidney disease in patients with type 2 diabetes T2DM [77] . Patients with elevated SUA levels have impaired renal function, regardless of glaciated haemoglobin (HbA1c) or duration of diabetes [78] . In T2DM, there is a significant,

independent positive association between elevated serum UA and an increased risk of decreased glomerular filtration rate [79].

Table (3): - Mean ± standard deviation of serum nitric oxide, Endothlen-1 and VIM in groups under investigation

Groups	ric oxide µM/ml	Endothlen-1 pg/ml	entin ng/ml
Control	43.78±7.154 a	11.521±2.244 b	0.938±3.711 a
G1	1±2.689 b	13.731±2.967 a	12.036±2.32 a
G2	26.28±4.589 c	13.624±3.419 a	12.049±1 a
G3	11.41±3.205 d	14.159±3.224 a	1.117±2.793 a

Nitric oxide level

Table 3 shows the mean ± SD of nitric oxide level (36.04 ± 2.689) µM/ml in serum group DM (26.28 ± 4.589) µM/ml in serum group DM+P (11.41 ± 3.205) µM/ml in serum group DM+R and (43.78) ±7.154) µM/ml in the serum of the control group. The results indicated that the level of nitric oxide in the blood decreased significantly (P≤0.05 all patient groups compared to the control group Figure 7

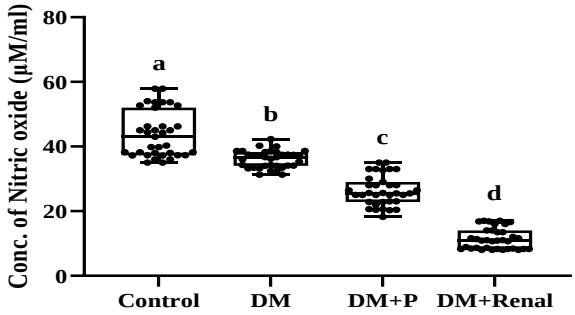

Figure.7: concentration of serum Nitric oxide in the four groups

its decrease in diabetic patients is due to the reason that diabetes is associated with high blood sugar, increased oxidative stress, and decreased nitric oxide production from endothelial cells. Increased oxidative stress affects a large scale in the development and progression of diabetes and its complications. [80.81] High glucose produces reactive oxygen species as a result of glucose auto-oxidation and metabolism [82]. Diabetes mellitus is characterized by a slow lesion in the development of both small and large micro angiopathy for diabetics, as micro angiopathy is retinopathy, nephropathy, and polyneuropathy. Most epidemiological studies have shown that chronic hyperglycaemia is the main cause of vascular and tissue damage. As sugar rises, oxidative stress increases and endothelial dysfunction develops [83]. Endothelial dysfunction is expressed in the lack of production or bioavailability of nitric oxide (NO) [84] due to reduced production and / or stopped

production by reactive oxygen species produced either by glycoproteins or directly from the endothelium of blood vessels [85]. Thus, it shows that the decrease was due to diabetes, which also plays a role for the rest of the groups that are related to diabetes, which are the diabetes and pressure groups, the diabetes group and the kidneys. between nitric oxide production and increased oxidative stress [86]. Also, this result is in agreement with the study Amrita at al Ghosh1 which found that nitrile oxide decreased significantly in the diabetic group compared with the control group.

Endothlen-1 level

Table 3 shows the mean ± SD for Endothlen-1 level (13.731±2.967) pg/ml in serum group DM, (13.624±3.419) pg/ml in serum group DM+P, (14.159±3.224) pg/ml in serum group DM+R and (11.521±2.244) pg/ml in the serum of the control group. The results indicated that the blood Endothlen-1 level increased significantly (P≤0.05) in All patient groups to the control group. Figure 8.

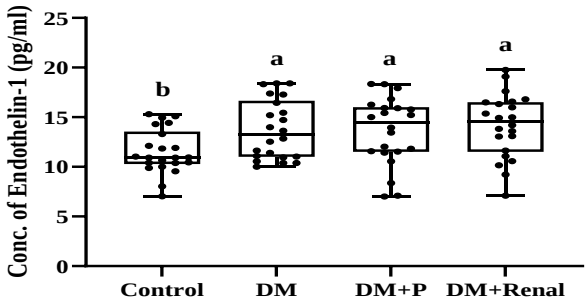

Figure.8: concentration of serum Endothlen-1 **in** the four groups

The level of endothelin-1 increased in All patient groups as endothelin-1 has an effect on diabetes, as the increase in production and increase in the functional effects of endothelin-1 changes significantly in cases of diabetes [87]. and high levels of insulin due to insulin resistance in The blood pressure increases the production and activity of endothelin-1, which promotes vasoconstriction and raises blood pressure. [88] It was also found that endothelin-1 impairs glucose uptake in skeletal muscle of insulin-resistant people, which leads to exacerbation of insulin resistance.

Oxidative stress also plays an important role: generation of reactive oxygen species (ROS) leads to synthesis of endothelin-1, meanwhile, endothelin-1 increases ROS generation via NAD(P)H oxidase [89] and levels of endothelin-1 are increased in patients with Diabetes mellitus and may contribute to endothelial dysfunction associated with diabetes. [90] 'This is consistent with our results in DM

And since ET-1 is considered an important pathogenic factor in high blood pressure, its level rises in patients with pressure and diabetes in the second group DM+P, while in DM+R its level rises and the reason is because endothelin-1 is responsible for the strong contraction of blood vessels [91] and thus increases renal vasoconstriction. An overall decrease in RBF in renal blood flow [92] The renal blood vessels are more sensitive to the vasoconstrictor effects of endothelin-1 than other blood vessels and thus increase in renal patients [93].

Vimentin level

Table 3 shows the mean ± SD for Vimentin level (12.036 ± 2.32) ng/ml in serum group DM (12.049 ± 1.958) ng/ml in serum group DM+P (11.117 ± 2.79) ng/ml in serum group DM+R and (10.938 ± 3.711) ng/ml in the serum of the control group. The results indicated that the level of Vimentin in the blood did not register significant differences (P≤0.05) in All patient groups to the control group. Figure 9.

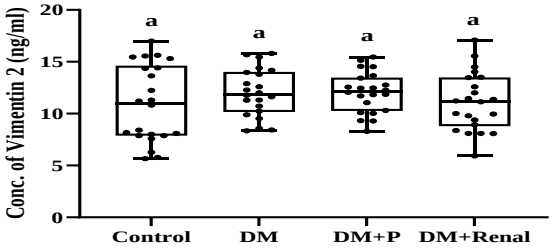

Figure.9: concentration of serum Vimentin in the four groups.

No significant differences were recorded between groups in our current study and the concentration of the Vimentin protein despite the slight increase in the groups compared to the control. Therefore, the Vimentin has no direct effect on the groups.

Conclusion: From all the above results we can conclude that diabetes, diabetes with stress and diabetes with kidney is a disease that increases oxidative stress through decreasing nitric oxide and it raises the level of endothelin-1, so that diabetes, hypertension and renal failure May increase morbidity and affect body activities.

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تأثير الاندوثلين-1 والفيمنتين وبعض المتغيرات الكيموحيوية على الرجال المصابين بداء السكري من النوع 2 وارتفاع ضغط الدم والمصابين بقصور كلوي

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الخلاصة:

تم إجراء دراسة شاملة لتقييم الاندوثلين-1و الفيمنتين وبعض المتغيرات البيوكيميائية. اشتملت الدراسة على 140 عينة دم من الرجال، مقسمة إلى أربع مجموعات: المجموعة 1 من داء السكري من النوع الثاني DM ، المجموعة 2 من داء السكري من النوع الثاني ومرضى ارتفاع ضغط الدم DM + P ، والمجموعة 3: مرضى السكري من النوع الثاني ومرضى المصابون - القصور الكلوي DM+ R ومجموعة التحكم .(C) ضمت كل مجموعة 35 عينة، وتراوحت أعمارهم بين (30-70) سنة. تم جمع عينات من مرضى وأصحاء من مستشفى سامراء العام والعيادات الخارجية ومستشفى الديلزة في تكريت في الفترة ما بين 2020/12/20 - 2021/4/20 . تتضمن الدراسة الحالية قياس مؤشر كتلة الجسم، وقياس تركيز الإندوثيلين -1، وبروتين فيمنتين ، الكلوكوز ، واليوريا ، والكرياتينين ، وحمض اليوريك ، و Hba1cفي الدم .أشارت النتائج إلى أن قيمة BMI ارتفعت بشكل معنوي (P≤0.05)في المجموعتين DM و DM + P ، بينما انخفضت في مجموعة DM + R مقارنة بمجموعة التحكم ، زاد تركيز الكلوكوز بشكل ملحوظ في مجموعات المرضى مقارنة بمجموعة الاصحاء مجموعة التحكم. زاد تركيز Hba1c بشكل ملحوظ في مجموعات DM و DM + P ، بينما لم يظهر DM + R أي فرق معنوي مقارنة بمجموعة الاصحاء ، وأظهرت النتائج أن تركيز اليوريا زاد بشكل ملحوظ في DM + P وDM + R ، بينما لم تظهر DM فروق ذات دلالة إحصائية. مقارنة بمجموعة الاصحاء، بينما زاد تركيز الكرياتنين بشكل كبير في DM + R بينما لم يظهر DM و DM + P أي فروق معنوية مقارنة بمجموعة الاصحاء، أشارت النتائج أيضا إلى أن تركيز حمض اليوريك ارتفع بشكل في جميع مجموعات المرضى مقارنة بمجموعة الاصحاء. انخفض تركيز أكسيد النيتريك بشكل ملحوظ في جميع مجموعات المرضى مقارنة بمجموعة الاصحاء. كما أشارت النتائج إلى أن تركيز الاندوثلين-1ارتفع بشكل معنوي في جميع مجموعات المرضى مقارنة بمجموعة الاصحاء، بينما لم يسجل تركيز الفيمنتين أي فروق معنوية. في جميع مجموعات المرضى مقارنة بمجموعة الاصحاء .