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Estimation Some Physiological Parameters in Renal Failure Patients in Babylon City

Shaymaa Hussein[1], Dhifaf Zeki Aziz[2], Zahraa Kamil Kadhim Lawi[3], Naser Jawad kadhum[4]

[1] Ph.D Scholar ,Department of Biology, Faculty of Sciences/ Kufa University.;
[2] Ph.D Scholar, Department of laboratory investigation, Kufa University;
[3]Ms.c. Scolar Department of Biology, Faculty of Sciences / Kufa University;
[4] assistant professor, Department of laboratory investigation, Kufa University. AL-Najaf Province/ Iraq
*Correspondence author: e-mail: shaymaa.alsaaedi@uokufa.edu.iq

Abstract

Chronic kidney disease (CKD) is a complicated syndrome that happens in exceedingly form of settings with clinical manifestations starting from a minimal elevation in blood serum creatinine to an body waste kidney failure. CKD conveys vital morbidity and mortality, could be a major risk issue of end stage kidney failure , and is so related to immense health and socioeconomic burdens Despite analysis advances within the past decades, however, the advanced pathophysiology of CKD isn't absolutely understood. an entire of thirty patients and twenty control subjects were collected between February to October 2016. several physiological parameters were calculable similar to (urea, creatinine , GFR ,SOD, and MDA). The results of present study illustrated that that the patients groups, had greater significant differences ($P \leq 0.05$) levels of SOD, MDA, Urea, Creatinine and GFR in contrast with control groups result at significant differences ($P \leq 0.05$).

Key wards:- antioxidant, SOD2 , CKD , Oxidative, Urea, GFR.

Introduction

Chronic kidney disease (CKD) is associated to oxidative stress that contribute to the event of diverse little- and long-standing complication together with cardiovascular disease, anemia, arteriosclerotic upset, medication disorder, hemostatic abnormality and impair immunity The occurrence of oxidative stress in CRF is established by accumulation of by-product of relations of reactive oxygen species (ROS) with different supportive or structural molecules inside the plasma and tissues of CRF humans and animals. as an example, several studies have shown prominent plasma concentration of macromolecule peroxidation creation, malondialdehyde, in humans and animals with CRF, more than a few factors supply to every variety of health consequences along with system disfunction, chronic inflammation and infection, condensed inhibitor

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levels, and increase of pathology toxins.The mortality is well higher in patients with CKD than inside the overall population, and distended oxidative stress has been exposed in patients with CKD [1,4.]

urea may be a little molecule that's made within the liver from proteins so as to you basically have been eating. it's unremarkably place out by the kidneys, therefore blood level increase as renal stop working. However alternative things modification the extent of urea in your blood stream also, in order that it's not asimple direct to renal perform. Here ar a number of the things:Blood loss into your gut - e.g. a bleeding lesion - raise ureaFluid ifyou are wanting solution (e.g. consumption terribly little), your renal stay moreurea within the bloodHow a lot of protein you've got ingested highprotein intake raises ureaLiver illness will stop urea being produced unremarkably urea remains a awfully helpful check once used along with creatinine. It also can be accustomed live however well dialysis is functioning to clear waste products[8].

Oxidative stress consequences from amplified concentration of reactive oxygen species and/or a reduction in antioxidants. The inhibitor enzymes, SOD (SOD), peroxidase (GPx) and enzyme type the first defence method alongside reactive species and oxidatives stress[2].

SOD change superoxide (O2) to hydrogen peroxide (H2O2) and 3 isoforming are known. SOD1 (CuZnSOD) is current in red blood cells (RBCs), SOD2 (MnSOD) is first and foremost mitochondrial and SOD3 is extracellular[3].

SOD (SOD) catalyzes the role of superoxide radical (O) to hydrogen peroxide (H O). although H O is 2 a pair of 2 2 2 not a radical, it's quickly converted by fenton result into OH radical that is extremely imprudent[5].

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Malondialdehyde (MDA) is major ending of essential response on membrane oily acids. although the cell is gifted with a lot of inhibitor systems to boundary the amount of macromolecules per oxidation, underneath bound circumstances defensive mechanism are often swamped, resulting in prominent tissue level of per oxidation yield, lipid per oxidation represent oxidative tissues damage cause by hydrogen peroxide, superoxide and grouping radical(Reactive oxygen Species,ROS), foremost to structural change of membrane with discharge

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of cell and organ inside, defeat of necessary fatty acids with configuration of cytosolic aldehyde and peroxide yield[6].

Material And Method:-

Sampling

Thrity blood samples were collected from patient with chronic kidney disease whom visit Dialysis center /marjan hospital /Babylon /Iraq and twenty samples as control.

Determination of superoxide dismutase (SOD) activity

superoxide dismutaswas determined by Winterbourne et al (1975).

Determination of Malondialdehyde (MDA)activity

Serum lipid peroxidation was determined by Ohkawa *et al.* (1979) after precipitating the protein according to the method of Satoh (1987). The result was expressed as n mol/ ml of serum[9].

Results

The data demonstrated that the patients groups, had greater significant differences (P≤ 0.05) levels of SOD2, MDA, Urea, Creatinine and GFR in contrast with control groups result as show in table(1). t-test.*P≤0.05.S.E: Standard error.

Table (1) serum superoxide dismutase, Malondialdehyde, Urea and Creatinine with measurement Glomerular Filtration Rate in patients and control groups.

Groups Parameter	Control (Mean±S.E)	Patients (Mean± S.E)	P-value
SOD2 (U/ml)	198.0±35.48	519.11±60.10	>0.0001*
MDA (µmol/l)	0.07±0.02	1.19±0.23	<0.0001*
Urea (mg/dl)	18.02±1.97	25.85±1.32	0.001*
Creatinine (µmol/l)	77.77±4.60	427.83±4.37	>0.0001*
GFR (mL/min)	15.76±4.42	101.3±5.31	>0.0001*

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Discussion

The consequences of estimation physiological parameters (SOD, MDA, Urea, Creatinine and GFR) illustrated that the patients groups,had greater levels in contrast with control groups result at significant differences ($P \leq 0.05$).

The p value of urea and creatinine was found to be 0.001, < 0.0001 respectively ($p < 0.05$), An amplify in urea height is seen when there is injure to the renal or the kidney is not performance correctly[10]. Creatinine may be a little, liberally drinkable material whose manufacture vary extremely small from day to daytime because it's resulting from the metabolism of muscles creatinene. Creatinine are often concealed by the proximal tubule from side to side correlate macrobiotic cation pathway. The concealed creatinine constituent confound GFR capacity since it will differ within people over time[11].

we should view of the information that eGFR (commonly employed in studies) is base on a patient's gender, along with other variable. the 2 the majority ordinary equation for assess eGFR, the MDRD (Modification of Diet in renal Disease) and CKD-EPI (Chronic renal disorder epidemiology Collaboration) equation, each employ gender as a changeable. they are support the thought that for a given creatinine level, men can have superior level of renal function than women due to elevated muscles accumulation and distended creatinine creation among men. Thus, in the require of study using the gold commonplace quantity of glomerular filtration rate (GFR) (which is that the mensuration of inulin), consequences relating to gender manipulate on CKD are usually prejudiced since of the consumption of gender needy equations[7].

GFR may be examination of what proportion the renal are filterings. it's unremarkably regarding a hundred mls/minute. this earnings that the renal are removings all the creatinine originate in 100mls of blood each minute nearly one hundred fifty litres per day! Most of this one hundred fifty litres is absorbed back to the body, so solely 12-tone music of the remains seems as urine. Creatinine clearance provides quite an good quality calculate of GFR, however needs a twenty four hour urine assortment for mensuration Knowing your GFR is incredibly helpful. It will provide you with associate ambit kidney operate, and be accustomed show your CKD stage[12].

within the present study level of MDA is considerably higher in cases

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when compared to controls. there is relationship between amplified levels of MDA and evolution of renal deficiency. MDA evaluation is one of the the majority frequently used technique for monitor lipid peroxidation in biological sample. The renal provide of reactive oxygen species(ROS) in tubular cells. present infiltrating cells (granulo-cyte-monocytemacrophage) and platelets present in infla-mmatory kidney process additionally turn out huge quantity of ROS. wholly dissimilar cellular enzymes as well as mitochondrial enzyme, lipoxygenase, cycloxygenase, myeloperoxidase, NADPH enzyme, organic compound enzyme, NO synthase are known as cellularsources of ROS arrangement[13].

ROS cause inflated lipid peroxidation. throughout this study level of MDA in blood serum of CRF is greater than control. amplified concentration of MDA have been report in plasma of hemodialyzed patient[14]. during this revision, a major increase in plasma MDA level was experiential in patients compare to control that were like to result of the researchers (Mimić-Oka et al., 1999) World Health Organization urged that the rise in MDA levels can be because of enlarged oxidative stress in kidney from varied source or reduce in inhibitor protection and the other way around[15].

Our results showed that SOD in CKD patients showed elevated, and arrive at to the many stage when put next to control grouping. This indicate that oxidative stress induced by high level of carbamide and creatinine might increase super compound major production with diabetic patients this result was in conformity with the opposite evidences [16].while alternative study showed SOD shrivelled value in HD patients ascompare to controls group. SOD is the enzymes of inhibitor protection in our body. As oxidative stress will increase, the protection reaction is powerless by the frequent offensive of the ROS, leading to lesser level of those enzymes. it's additionally doable that persons with CKD have a lesser concentration of antioxidant enzymes create them disposed to the illness within the main position[17].

Ethical Clearance :The study was approved by the Baghdad ethics committee of the Iraq ministry of health and environment. all participants in the study gave their written permission to access their medical records, blood sampling, and anonymous use of their samples.

Source of Funding : Self

Conflict of Interest: Nil

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