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Effect of Curcmin on Chronic Kidney Disease of Expermintal Rats.

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Abstract

The objective of this study was to study the effect of curcumin (CUR) on rats with chronic kidney disease. Forty-two male albino rats weighing 160 ± 5 g have been used in this study. The first group (n = 6) was fed on a basal diet (control -ve). To induce CKD in rats, adenine was added to the diet by about 0.75% w/w "adenine diet". Rats in this study (n = 42) were divided into 7 groups (n=7 rats) and fed on a tested diet for 6 weeks: group1 : fed on a normal diet, and used as a normal control group, group2 : fed on adenine diet, and was used as a CKD control group, group 3 : fed on low protein diet "12.5% protein" containing 0.75% adenine "low protein adenine diet", group 4 & 5: fed on adenine diet containing 100 and 150 mg curcumin/kg diet, respectively, group 6 & 7 : fed on low protein adenine diet containing 100 and 150 mg curcumin/kg diet, respectively. Results have shown that all chronic kidney disease groups receiving 100 mg and 150 mg curcumin have produced varying increases in body weight gain, feed intake, and feed efficiency ratio. The results showed that there was a significant

decrease ($P \leq 0.05$) between the groups which were treated with an adenine diet or low protein adenine diet containing 100 mg and 150 mg curcumin in internal organ weights, as compared to the positive control group. Liver enzymes including (aspatate amino transferes (AST), alannine amino transferes (ALT), and alkaline phosphatase (ALP)), kidney functions (uric acid, urea nitrogen, and creatinine), lipid profile (cholesterol, triglycerides, LDL-c, VLDL-c), and malondialdehyde increased significantly in the positive control group, while HDL-c and the glutathione decreased significantly, as compared to the negative control group. All treated groups with adenine diet or low protein adenine diet containing 100 & 150 mg curcumin/kg diet showed significant decrease in these parameters except HDL, as compared to the positive control group. So, this study recommended using curcumin for kidney diseases.

Introduction

Chronic kidney disease (CKD) encompasses a spectrum of pathophysiological processes associated with the abnormal renal function (such as proteinuria) and a progressive decline in glomerular filtration. CKD is a major health problem and its prevalence is increasing at least in part worldwide due to an increase in the prevalence of systemic diseases such as metabolic syndrome that affect kidney function *(**Trivedi et al., 2002, Dirks et al., 2005 and Ghelani et al., 2019**).* It is well documented that cardiovascular disease, such as heart failure or coronary artery disease, is one of the leading causes of death in CKD patients. As a result, most CKD

patients die from cardiovascular disease before dialysis becomes necessary ***Yamamoto*** and ***Kon, 2009 and Vaziri, 2014).***

CKD is associated with cardiovascular complications, such as dyslipidemia, atherosclerosis and myocardial infarction (***Vaziri, 2014***). Patients and experimental animals with CKD have a high plasma concentration of lipids, such as cholesterol, triglycerides and fatty acids ***(Chen et al., 2013 and Bulbul et al., 2018).*** Current therapeutic regimens, including the use of statins and fibrates, have limited success in the treatment of associated CKD dyslipidemia and do not address the underlying causal factors ***(Harper and Jacobson, 2008).*** Although statins may be effective in slowing CKD progression in patients with mild to moderate CKD, they have consistently failed to reduce HDL deficiency ***(Weiner and Sarnak, 2004, and Harper and Jacobson, 2008)***. Fibrates are indicated when hypertriglyceridaemia is the primary lipid abnormality in the CKD patient and may significantly reduce triglyceride levels ***(Weiner and Sarnak, 2004).*** However, fibrates are excreted by the kidney and may cause myositis, especially when used in conjunction with statins ***(Broeders et al., 2000).***The development of novel therapies to either slow or reverse the deterioration of renal function, as well as to improve the metabolic dyslipidemia of CKD, is therefore essential. Natural products have shown significant potential for improving hepatic lipid metabolism in experimentally induced CKD ***(Nakagawa et al., 2004; Ahmed, 2010; Ali et al., 2013 and Manivannan et al., 2013).***

Curcumin is a natural product of a polyphenol class that is abundantly present in Curcuma longa (turmeric rhizome) ***(Akomolafe et al., 2021).*** Several reports have shown its wide range of pharmacological properties including chemopreventive, anti-inflammatory, antioxidant and neuroprotective activity in different experimental models ***(Kuhad et al., 2007; Strimpakos and Sharma, 2008 and Nabavi et al., 2012)***. Research studies on the therapeutic effect of curcumin in KBrO3-induced renal dysfunction have not, however, been conducted. The study, therefore sought to investigate the modulatory role of curcumin in renal arginase and adenosine deaminase, as well as oxidative stress parameters and histopathological changes in treated groups induced renal damage in rats.

Materials and Methods

Materials:

Curcumin was obtained from the (Shana company in Shepen Elkom, Monofia, Egypt).

Rats:

Albino adult male rats (n=42) of Sprague-Dawely strain weighing 160 ± 5 g were purchased from Helwan Farm of Experimental Animals, Helwan, Egypt. Cholesterol has been obtained from VACSERA, Dokki, Egypt.

Chemicals:
Casein, vitamins, minerals and cellulose have been obtained from Elgomhoria Company, Egypt. The kits have been purchased from the Gama Trade Company, Dokki, Egypt.

Biological Assay:

A group of 42 rats were housed in hygienic conditions and fed a basic diet ***(Reeves et al., 1993)*** for one week of adaptation. After that week, rats were randomly divided into tow groups as a following:

Group (1) : (6 rats) fed on a normal diet, and used as a normal control group

Group (2) : (38 rat) were divided into 6 sub groups as following:

Sub group (1): (6 rats) fed on a normal diet containing 0.75% adenine "adenine diet", and used as a CKD control group.

Sub group 2: (6 rats) fed on low protein diet "12.5% protein" containing 0.75% adenine.

Sub group 3 & 4: (6 rats) each group fed on adenine diet containing 100 and 150 mg curcumin/kg diet, respectively.

Sub group 5 & 6: (6 rats) each group fed on low protein adenine diet containing 100 and 150 mg curcumin/kg diet, respectively.

At the end of this experiment (6-week), two blood samples were taken from each rat, one sample was centrifuged to obtain serum for biochemical analysis, while the second sample (whole blood) was used to determine hematological parameters.

Feed intake (FI) was determined, feed efficiency ratio (FER), body weight gain (BWG%) and organs relative weight were calculated according to ***Chapman et al., (1959).***

Biochemical Analysis:

Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were estimated according to (***Thomas, 1998***). Serum alkaline phosphatase (ALP) was determined according to (***Roy, 1970***). Determination of Serum Creatinine (Cr) Creatinine was determined using the method described by ***Henry (1974)***.

The serum urea concentration was determined using the method described in ***Fossati et al. (1980)***. Serum malondialdehyde (MDA) was measured by (***Sinha (1972 and Draper and Hadly, 1990***); ***Fossati and Praneipe, 1982*** and ***Young, 2001***, respectively). VLDL-c and LDL-c were determined using the method described in ***Friadwald et al., (1972).***

Statistical Analysis:

The data was analyzed in accordance with the SPSS program. The ANOVA test was used to compare groups and the P-value <0.05 was considered to be significant (**SPSS, 1986**).

Results and Discussion

Effect of Low Protein Diet and Curcumin on Body Weight, Feed Intake and Feed Efficiency Ratio of Rats with Chronic Kidney Disease

The data in Table (1), the data showed that the percentage of body weight gain in the control group + ve decreased significantly ($p < 0.05$) compared to the negative control group (152.20 ± 10.0 VS 169.80 ± 4.324) The observed decrease in body weight gain, feed efficiency, feed intake and growth rate in rats fed adenine may be due to a decrease in the palatability of the feed mixture. The percent BWG increased significantly in In all tested groups compared to the positive control group ($P < 0.05$). Food consumption decreased in the negative control group compared to the positive control group. The best results were the markings in a groups 6 and 7. Non-significant in FER were observed between healthy and all tested groups .

In this study, the body weight of rats treated with curcumin was not significantly improved compared to CKD rats. The reason for the observed difference in body weight between curcumin-treated and untreated CKD rats is not clear. The possible explanation for this discrepancy is a decrease in food intake in CKD rats compared to control rats (***Ghelani et al., 2019***).

Effect of curcumin on Relative Organs Weight of Rats with chronic kidney disease

The result of changes in the relative weight of the organs is shown in table (2). The positive control group of rats administered adenine diet (CKD) diet showed a decrease in the mean relative weights of the liver and kidney significantly compared to the -ve group. Treating the CKD groups with (low protein diet, two levels from curcumin and low protein diet with the two levels from curcumin) sowed significant increase $p \leq 0.05$, as compared to the positive

control group. The data in this Table revealed that, non-significant differences were observed in liver and kidney between all tested groups.

Effect of Curcumin on Serum Liver Functions of Rats with Chronic Kidney Disease

The results of Table (3) showed the curative effect of curcumin on alanine aminotransferase activity (ALT). The positive control group and group 3 had the significant highest values of serum ALT level of 68.60 ± 0.45 u/l and 56.40 ± 1.817u/l, respectively. The results showed that when rats gave curcumin in their diet at any level the ALT level decreased in serum when compared with positive control group.

The results of Table (3) demonstrated the curative effect of curcumin on the serum activity of aspartate aminotransferase (AST). The data showed that AST activity increased significantly when animals received adenine and adenine and low protein (positive control group) with a mean value of 126.00 ± 1.00 u/l, 105.00 ± 6.55 u/l, respectively when compared with the negative control group (36.60 ± 2.07 u/l). However, when rats were treated with a balanced or low protein diet containing two levels of curcumin, there was a significant decrease in serum AST activity, as compared to the positive control groups which were fed on a basal or low protein diet. Data in this table showed non-significant changes in AST enzymes between all tested groups.

However, when rats were treated with a balanced or low protein diet containing two levels of curcumin, there was a significant decrease in serum AST activity, as compared to the positive control groups which were fed on a basal or low protein diet. Data in this table showed non-significant changes in AST enzymes between all tested groups.

Liver damage is the most common toxic effect observed with the overloading of iron. Excessive deposition of iron in hepatocytes causes fibrosis and cirrhosis (W***eintraub et al., 1985, Deugnier et al., 2008 and Espinoza and Muriel , 2009***). Dietary curcumin lowers the lipid peroxidation induced by Fe21 intoxication to rats by enhancing the activity of antioxidant enzymes (***Reddy and Lokesh, 1994***)[a]. In addition, curcumin and other spice ingredients have been shown to inhibit the oxidation of Fe21 by H2O2 in the Fenton reaction (***Reddy and Lokesh, 1994***)[b], which produces OH radicals involved in the initiation of lipid peroxidation (***Girotti and Thomas, 1984***).

This study in the same line with ***Reddy and Lokesh , (1996)***[a] who indicated that the injury caused by iron intoxication to hepatic parenchyma could be normalized by curcumin. The authors found that the administration of 30 mg/kg p.o. of curcumin daily to Wistar rats for 10 days significantly decreased the degree of iron-induced lipid peroxidation 30 mg/kg, intraperitoneal (i.p.). Serum enzyme activity of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are very important adjuncts to diagnosis and measurement of liver injury; iron administration significantly increased hepatic lipid peroxide and serum AST, ALT and lactate dehydrogenase. Curcumin significantly prevented serum levels of

AST and ALT in iron-treated rats, indicating that this spice principle reduced iron toxicity by reducing lipid peroxidation (R***eddy and Lokesh, 1996***).

Other curcumin targets, such as NF-kB inactivation (S***ingh and Aggarwal, 1995***), cannot be discarded to contribute to the beneficial effects of curcumin on iron toxicity.Curcumin has been shown to reduce the toxic effects of iron loading in rat liver epithelial cells . In summary, there is evidence that curcumin has beneficial effects on iron toxicity; however, further studies are needed to confirm this effect. New studies should include histopathological analysis and more in-depth investigation of the mechanism of action of curcumin in the light of its many and new described properties (***Goel et al., 2008 and Strimpakos and Sharma, 2008***).

Effect of Curcumin on Kidney Functions of Rats with Chronic Kidney Disease

Table (4) shows the curative effect of curcumin on renal function (serum concentrations of urea, creatinine and uric acid) in rats with chronic kidney disease. When adenine and adenine + low protein When adenine diet and low protein adenine diet were received in rats, the serum urea concentration increased significantly with an average value of 40.80 ± 1.05 mg/dl35.80 ± 1.92 mg/dl, respectively compared to the control group negative (28.40 ± 1.81 mg/dl). While the group of rats fed adenine, adenine + low protein and curcumin supplementation at each intake showed a significant

decrease in serum urea levels until they reached normal levels compared to the positive control group.While the groups of rats which were fed on an adenine diet or a low protein adenine diet containing the two levels of curcumin showed a significant decrease in serum urea levels until they reached normal levels compared to the positive control group.

Table (4) showed that the positive control group fed adenine, adenine and low protein had an increased concentration of creatinine The mean values of serum creatinine increased significantly in the positive control groups which fed on an adenine diet or low protein adenine diet 1with an average value of 1.86 ± 0.55 and 1.02 ± 0.130 mg/dl, respectively compared to the control group negative (0.70 ± 0.022 mg/dL). The group of rats received a basic diet and there was a significant decrease in the serum creatinine concentration compared to the positive control group in either crcumin or adenine, adenine and low protein acid complemented with curcumin. When feeding CKD groups on the tested diet which used in this study, serum creatinine decreased significantly, as compared to the positive control groups. All treated groups showed non-significant changes in serum creatinine, as compared to the negative control group.

In the same table uric acid level was highest in the positive control group when compared with the negative control group with a mean value of 4.95 ± 0.23 mg/dL, 3.18 ± 0.08 mg/dL, respectively. In addition, the rest of the groups (3.4.5.6 and 7) serum uric acid decreased significantly when compared with the positive group.

The kidney is susceptible to oxidative stress due to its low level of antioxidant defense systems, including antioxidant enzymes. ***Khan et al., (2012)*** reported that there is an elevation of serum levels of MDA with low antioxidant enzymes (catalase, superoxide dismutase, glutathione peroxidase, etc.) in rats' kidneys. Similar results were obtained in this study as SOD, CAT, GPX activity and GSH levels were significantly lower in treated groups when compared with the positive control group. In addition, treated groups have a significant increase in the MDA level in rats' kidneys compared to the negative control. Low antioxidant activity and high MDA levels observed suggest oxidative damage to rat kidney tissue. There are indications that increased urea and creatinine levels, as well as high arginase activity, may lead to redox imbalance and free radical production (***Akomolafe et al., 2021***).

Siddhartha et al., (2014) demonstrated that curcumin was as effective in reducing the inflammatory cytokines TNFα and IL-1β and effectively abated both proteinuria and kidney injury as manifested by glomerulosclerosis and tubulointerstitial injury . The presence of curcumin is a significant reduction in the cytokines was noted . However, creatinine and blood urea nitrogen (BUN) biomarkers for CKD did not significantly change but there was a marked reduction of inflammatory markers TNFα, IL-6 and C-reactive protein. It is recorded that these patients also had significant reduction of their BMI.

The positive control group shows a significant increase (P≤0.05) in the TC, TG, VLDL-C and LDL-C levels as indicated in the table (6). However, the HDL-C level was significantly lower compared to the negative control group . Diets complemented by curcumin showed a significant reduction in the mean values of the serum lipid profile, except serum HDL-C Which recorded significant increase compared with the +ve control group . Groups of rats with CKD which were fed curcumin improved the lipid profile compared to the positive control group (+V). These changes indicate the importance of a curcumin-based diet in the treatment or prevention of chronic kidney disease.

Studies recommended that when patients have CKD they have impaired metabolism of triglyceride rich lipoprotein (i.e. VLDL) which reduces the fatty acids in the adipose tissue and reduces the energy storage capacity and therefore contributes to weight loss, waste and cachexia in CKD conditions. In the present study, however, curcumin could not reverse the weight reduction induced by CKD, although it did reduce impaired triglyceride metabolism in CKD rats. In addition, CKD rats increased water intake as the kidneys lost the ability to concentrate urine and excrete more water (which is evident by the increased urinary volume of CKD rats). CKD rats drink more water because of thirst. Curcumin treatment, however, normalized the intake of water, which may be due to its protective effects on kidney damage and therefore retains the ability of the kidneys to concentrate urine (which is evident by the decreased urinary volume of curcumin- treated rats). CKD animals have marked increase in total serum LDLand VLDL-cholesterol. Serum total

cholesterol elevation may be due in part to improved biosynthesis of cholesterol by up-regulation of the HMG-CoA reductase enzyme (***Vaziri and Liang, 1995, Liang et al., 2005*** and ***Ghelani et al., 2019***) as well as to a relative reduction of hepatic cholesterol elimination by down-regulation of the cholesterol in CKD animals (***Pandak et al., 1994*** and ***Pahl et al., 1998***). In addition, increased serum LDL-cholesterol in CKD rats may be due to the down-regulation of the LDL receptor in response to CKD (***Vaziri and Liang, 1996***). Previous studies involve inefficient translation and/or increased LDL-receptor protein turnover (***vaziri et al., 2003*** and ***liu*** and ***Vaziri, 2014***). However, our results showed that chronic curcumin supplementation with CKD rats effectively reduced serum and hepatic total cholesterol and serum LDL-cholesterol and were found to be consistent with previous curcumin studies using various dyslipidaemic animal models.

Effect of Curcumin on Serum Malondialdehyde and Glutathione of Rats with Chronic Kidney Disease

As showen in Table (6) the mean value of malondialdehyde activity increased significantly in the positive control group compared with the normal group (P>0.05). Rats fed adenine, adenine+ low protein diet showed a significant decrease (P>0.05) in average glutathione activity (Table 6) compared to the normal control group. In addition, the mean values of glutathione activity due to the feeding of rats with curcumin increased significantly in all groups compared to the corresponding values of the positive control group (P>0,05). The mean value of malondialdehyde activity decreased significantly (P>0.05) due to nephrotoxicity in rats with curcumin compared to the

+ ve control group .The curcmin sample tested had beneficial effects on both glutathione and malondialdehyde activity.

The kidney is susceptible to oxidative stress due to its low level of antioxidant defense systems, including antioxidant enzymes. (***Akomolaf, 2021*** and ***Khan et al 2012***) demonstrated is an elevation in MDA level and a decrease in antioxidant enzymes (catalase, superoxide dismutase, glutathione peroxidase, etc.) in rats' kidneys. Similar results were obtained in this study as SOD, CAT, GPX activity and GSH levels were significantly lower in treated groups resulted compared to control. In addition, the positive group had a significant increase in the MDA level in rats' kidneys compared to the -ve control. Low antioxidant activity and high MDA levels observed suggest oxidative damage to rat kidney tissue. There are indications that increased levels of urea and creatinine, as well as high activity of arginase, may lead to redox imbalance and free radical production. (***Modaresi et al., 2015***).

Curcumin is an extremely potent lipid soluble antioxidant and has been suggested to act through its pro-oxidant/antioxidant effects, because, formation of ROS by curcumin and curcuminoids correlates with their apoptotic activity on tumour cells . The free radical scavenging activity of curcumin can arise either from the phenolic OH group or from the CH2 group of the β-diketone moiety. A reactive free radical may undergo electron transfer or abstract H-atom from either of these two sites (***Mishra et al., 2005***).

Table (1): Effect of Low Protein Diet and Curcumin on Body Weight, Feed Intake and Feed Efficiency Ratio of Rats with Chronic Kidney Disease

Parameters / Groups	Body weight Gain (%)	Feed intake (g/day/rat)	Feed efficiency ratio
G(1):Control (-Ve)	$169.80^{a} \pm 4.32$	$12.60^{b} \pm .53$	$12.97^{b} \pm .0.59$
G(2): Control (+Ve)	$152.20^{c} \pm 10.03$	$13.19^{a} \pm 0.86$	$11.01^{bc} \pm 0.31$
G(3): 0.75% w/w adenine+ low protein 12.5%	$160.60^{b} \pm 4.61$	$9.22^{c} \pm .733$	$16.56^{a} \pm 1.19$
G(4):0.75% w/w adenine+100mg curcumin	$161.80^{b} \pm 9.78$	$13.24^{a} \pm .546$	$11.65^{b} \pm 0.97$
G(5): 0.75% w/w adenine+150mg curcumin	$157.80^{bc} \pm 12.96$	$12.10^{b} \pm 0.51$	$12.22^{b} \pm 0.79$
G(6): 0.75% w/w adenine+ low protein 12.5%+100mg curcumin	$164.40^{b} \pm 6.10$	$12.86^{b} \pm 0.63$	$12.49^{b} \pm 1.15$
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	$164.00^{b} \pm 3.80$	$13.20^{a} \pm 0.76$	$11.85^{b} \pm .237$

*Values are expressed as means ±SE.

*Values at the same column with different letters are significant at $P<0.05$.

Table (2): Effect of curcumin on Relative Organs Weight of Rats with chronic kidney disease.

Parameters / Groups	Liver (%)	Kidney (%)
G(1):Control (-Ve)	17.28 ^{a} ± 0.57	7.32 ^{a} ± 0.46
G(2): Control (+Ve)	15.18 ^{c} ± 0.58	5.88^{c} ± 0.44
G(3): 0.75% w/w adenine+ low protein 12.5%	15.52^{bc} ± 0.58	6.10 ^{b}± 0.43
G(4):0.75% w/w adenine+100mg curcumin	16.10 ^{b} ± 0.29	6.80 ^{b}± 0.40
G(5): 0.75% w/w adenine+150mg curcumin	16.00 ^{b} ± 0.58	6.34 ^{b}± 0.30
G(6): 0.75% w/w adenine+ low protein 12.5%+100mg curcumin	16.40 ^{b} ± 0.73	6.74 ^{b}± 0.47
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	16.12 ^{b} ± 0.46	6.68 ^{b}± 0.42

Table (3): Effect of Curcumin on Serum Liver Functions of Rats with Chronic Kidney Disease.

Parameters / Groups	ALT	AST	ALP
	(µ/L)		
G(1):Control (-Ve)	23.00^{d} ± 2.8	36.60^{c} ±2.07	825.20^{b} ± 41.82
G(2): Control (+Ve)	68.60^{a} ± 0.45	126.0^{a} ±1.00	868.00^{a} ±1.00
G(3): 0.75% w/w adenine+ low protein 12.5%	56.40^{b} ±1.81	105.0^{b} ± 6.55	840.80^{b} ±3.83
G(4):0.75% w/w adenine+100mg curcumin	32.80^{c} ±2.86	42.40^{c} ± 5.50	837.40^{b} ± 2.07
G(5): 0.75% w/w adenine+150mg curcumin	28.80^{c} ± 2.86	41.80^{c} ± 4.91	838.60^{b} ± 2.70
G(6): 0.75% w/w adenine+ low	30.60^{c} ±	39.00^{c} ±	835.20^{a}

protein 12.5%+100mg curcumin	2.70	2.91	± 3.56
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	26.80^{c} ±1.30	37.20^{c} ± 2.49	828.60^{b} ± 3.78

Table (4): Effect of Curcumin on Kidney Functions of Rats with Chronic Kidney Disease.

Parameters / Groups	Urea	Creatinine	Uric acid
	mg/dl		
G(1):Control (-Ve)	28.40^{c} ±1.81	0.70^{c} ±.022	3.18^{b} ± 0.08
G(2): Control (+Ve)	40.80^{a} ±1.05	1.86^{a} ± 0.55	4.95^{a} ± 0.23
G(3): 0.75% w/w adenine+ low protein 12.5%	35.80^{b} ±1.92	1.02^{b} ± 0.13	4.46^{ab} ± 0.19
G(4):0.75% w/w adenine+100mg curcumin	31.00^{c} ±4.18	0.66 ± 0.04^{c}	3.38^{b} ± 0.31
G(5): 0.75% w/w adenine+150mg curcumin	28.80^{c} ±0.83	0.71^{c} ± 0.01	3.60^{b} ± 0.41
G(6): 0.75% w/w adenine+ low protein 12.5%+100mg curcumin	30.00^{c} ±2.34	0.66^{c} ± 0.058	3.44^{b} ± 0.20
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	26.80^{c} ±0.83	0.65^{c} ± 0.05	3.30 ± 0.15^{b}

Table (5): Effect of Curcumin on Serum Lipid Profile Rats with Chronic Kidney Disease.

arameters / Groups	TC (mg/dl)	TG (mg/dl)	HDL-c (mg/dl)	LDL-c (mg/dl)	VLDL-c (mg/dl)
G(1):Control (-Ve)	129.60 c ± 8.67	116.40c ± 6.10	39.60 b ± 1.51	66.72b ± 8.07	23.28c ±1.22
G(2): Control (+Ve)	242.20a ± 7.85	200.00a ± 5.09	49.20 a ± 1.92	153.00a ±8.11	40.00a ±1.02
G(3): 0.75% w/w adenine+ low protein 12.5%	140.60 b ± 4.50	154.60b ± 6.34	44.80ab ± 2.28	64.88b ±4.71	30.92b ±1.27
G(4):0.75% w/w adenine+100mg curcumin	142.20 b ± 1.64	143.20 bc ±5.63	47.20 a ±2.38	66.36 b ±2.33	28.64b ±1.12
G(5): 0.75% w/w adenine+150mg curcumin	141.40 b ± 2.88	141.40 bc ±6.69	44.60 a ±1.81	68.52b ± 5.04	28.28 b ±1.33

Groups \ Parameters	Malondialdehyde (nmol/min/mg protein)			Glutathione U/mg protein	
G(6): 0.75% w/w adenine+ low protein 12.5%+100mg curcumin	136.80 b ±4.43	122.20c ±1.48	46.40a ±1.81	65.96b ± 4.77	24.44 b ± 0.29
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	137.52 b ±3.43	123.34c ±1.67	47.10a ±3.25	66.57b ±3.53	25.03b ± 0.19

Table (6): Effect of Curcumin on Serum Malondialdehyde and Glutathione of Rats with Chronic Kidney Disease.

G(1):Control (-Ve)	70.20 ^{b} ±6.05	4.10 ^{a}±0.00
G(2): Control (+Ve)	150.00 ^{a} ± 6.44	2.56 ^{c} ±0.54
G(3): 0.75% w/w adenine+ low protein 12.5%	146.60 ^{a} ± 1.14	4.94 ^{a} ±0.41
G(4):0.75% w/w adenine+100mg curcumin	82.20 ^{b} ± 2.58	3.94 ^{ab}±0.47
G(5): 0.75% w/w adenine+150mg curcumin	78.60 ^{b} ± 2.07	4.92 ^{a}±0.46
G(6): 0.75% w/w adenine+ low protein 12.5%+100mg curcumin	76.80 ^{b} ± 4.60	3.54 ^{b}±0.55
G(17): 0.75% w/w adenine+ low protein 12.5%+150mg curcumin	75.20 ^{b} ± 3.42	3.94 ^{ab}±0.82

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تأثير الكركمين على امراض الكلى المزمنة للفئران

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الملخص العربى

الهدف من هذه الدراسة هو دراسة تأثير الكركمين على الفئران المصابة بأمراض الكلى المزمنة. تم استخدام اثنين وأربعين من ذكور الجرذان البيضاء في هذه الدراسة اوزنها ١٦٠ ± ٥ جم. تم تقسيم ذكور الجرذان (عددهم = ٤٢) إلى ٧ مجموعات لمدة ٦ أسابيع: المجموعة الأولى وهي المجموعة الضابطة السالبة وتتغذي علي الغذاء الاساسي فقط ، المجموعة الثانية (عددها = ٦) تغذت على الغذاء الاساسى بالإضافة إلى (٠,٧٥٪ وزن / وزن مكمل غذائي الأدينين) لاحداث أمراض الكلى المزمنة للفئران ، مجموعة ٣: (عددهم = ٦ ، ٠,٧٥٪ وزن / وزن أدينين + نظام غذائي مكمل منخفض البروتين ١٢,٥٪) ، المجموعة ٤: (عددهم = ٦ (١٠٠ مجم / كجم / يوم كركمين + ٠,٧٥٪ وزن / وزن نظام غذائي مكمل بالأدينين) ، المجموعة ٥ (عددهم = ٦ (١٥٠ مجم / كجم / يوم كركمين + ٠,٧٥٪ وزن / وزن نظام غذائي مكمل بالأدينين) ، المجموعة ٦: (عددهم٦ = ٦ ، ١٠٠ مجم / كجم / يوم كركمين + ٠,٧٥٪ وزن / وزن الأدينين + نظام غذائي مكمل منخفض البروتين ١٢,٥٪) المجموعة ٧: (عددهم = ٦ ، ١٥٠ مجم / كجم / يوم كركمين + ٠,٧٥٪ وزن / وزن أدينين + بروتين منخفض ١٢,٥٪). أظهرت النتائج أن جميع مجموعات أمراض الكلى المزمنة التي تتلقى ١٠٠ ملجم و ١٥٠ ملجم من الكركمين زيادات متفاوتة في زيادة وزن الجسم ، كما أظهرت النتائج وجود فروق معنوي بين المجموعة الضابطه السالبه ومجموعة الكركمين ١٠٠ ملجم و ١٥٠ ملجم كركمين .اما بالنسبه لأوزان الأعضاء لا يوجد اختلاف بين المجموعة الضابطه السالبه والمجموعة الفئران السليمة. هناك أرتفاع معنوى فى كلا من انزيمات الكبد ,وظائف الكلى, دهون الدم و MDA بالمجموعة الضابطه الموجبه ولكن الجلوتاثيون انخفض معنويا مقارنة بالمجموعه الضابطة السالبة . أضافة لذلك أشارت النتائج إلى أن مجموعات الجرذان المصابة بأمراض الكلى المزمنة والتي عولجت بالكركمين بجرعة ١٠٠ مجم أو ١٥٠ مجم أدت إلى انخفاض معنوي فى كل هذه العناصر باستثناء الليبوبروتين عالى الكثافة مقارنة بالمجموعه الضابطة السالبة. لذلك, أوصت هذه الدراسة لاستخدام الكركمين لأمراض الكلى.