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Original Article

Beneficial effects of coconut oil *(Cocos nucifera)* on hematobiochemicl and histopathological markers in CCL4-intoxicated rabbits

Efeitos benéficos do óleo de coco (*Cocos nucifera*) em marcadores hematobioquímicos e histopatológicos em coelhos intoxicados com CCL4

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Abstract

The study was designed to investigate the effect of Coconut Oil on the levels of some liver and hematological parameters in carbon tetrachloride intoxicated rabbits. Also the antioxidant capacity of Coconut Oil for various concentrations was assessed on the basis of percent scavenging of (DPPH) free radical. Experimental animals were divided into five groups, eight rabbits in each group. These were: group A (Normal control), group B (Toxic control), group C (Standard control), group D (Treated with Coconut Oil 50 mL/kg body weight after CCl4 intoxication), group E (Treated with Coconut Oil 200 mL/kg body weight after CCl4 intoxication). The effects observed were compared with a standard hepatoprotective drug silymarine (50 mL/kg body weight). The Coconut Oil (200 mL/kg body weight) significantly (P<0.05) reduced the elevated serum levels of alanine transaminase (ALT), aspartate transaminase (AST) and alkaline phosphatase (ALP) when compared to a toxic control rabbits. The results of extract treated rabbits were similar to silymarine administered rabbits group. Treatment with Coconut Oil root and silymarine caused no significant changes in RBC, Platelets, (Hb), (MCH) concentration and (HCT) values. However, significant (P<0.05) increase was observed in the total WBC count. The present study suggested that Coconut Oil can be used as an herbal alternative (need further exploration i.e to detect its bioactive compound and its efficacy) for hepatoprotective activity.

Keywords: coconut oil, hepatoprotective, alternative of silymarine, antioxidant, rabbits.

Resumo

O estudo foi desenhado para investigar o efeito do óleo de coco nos níveis de alguns parâmetros hepáticos e hematológicos em coelhos intoxicados com tetracloreto de carbono. Também a capacidade antioxidante do óleo de coco para várias concentrações foi avaliada com base na porcentagem de eliminação de radicais livres (DPPH). Os animais experimentais foram divididos em cinco grupos, oito coelhos em cada grupo. Estes foram: grupo A (controle normal), grupo B (controle tóxico), grupo C (controle padrão), grupo D (tratado com óleo de coco 50 mL/kg de peso corporal após intoxicação por CCl4), grupo E (tratado com óleo de coco 200 mL/kg de peso corporal após intoxicação por CCl4). Os efeitos observados foram comparados com um fármaco hepatoprotetor padrão silimarina (50 mL/kg de peso corporal). O óleo de coco (200 mL/kg de peso corporal) reduziu significativamente (P<0,05) os níveis séricos elevados de alanina transaminase (ALT), aspartato transaminase (AST) e fosfatase alcalina (ALP), quando comparado a um coelho controle tóxico. Os resultados dos coelhos tratados com extrato foram semelhantes aos do grupo de coelhos administrados com silimarina. O tratamento com raiz de óleo de coco e silimarina não causou alterações significativas nos valores de RBC, Plaquetas, (Hb), (MCH) e (HCT). No entanto, observou-se aumento significativo (P<0,05) na contagem total de leucócitos. O presente estudo sugeriu que o óleo de coco pode ser usado como uma alternativa fitoterápica (precisa de mais exploração, ou seja, para detectar seu composto bioativo e sua eficácia) para atividade hepatoprotetora.

Palavras-chave: óleo de coco, hepatoprotetor, alternativa de silimarina, antioxidante, coelhos.

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1. Introduction

Liver in the human body are located in the upper right part of the abdomen and is reddish- brown in color (Ozougwu, 2017). The liver has two lobes left and right and performs various vital metabolic functions (Zaefarian et al., 2019). Liver perform a key role in the clearance and transformation of chemicals and thus it is exposed to toxic injury (Gracia-Sancho et al., 2021). Liver injury is related always with cellular necrosis; decrease in tissue lipid peroxidation. More over serum levels of many biochemical markers like alanine amino transaminase (ALT), aspartate amino transaminase (AST), serum glutamic oxalo acetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), total cholesterol, and total bilirubin (TB) are evaluated (Wang et al., 2019). Different aspects of liver injury during hepatotoxicity may include hepatitis (Neuman, 2020), granuloma and vascular lesions (Saukkonen et al., 2006). The major effect of hepatotoxicity is jaundice which is caused due to bilirubin accumulation in the extra cellular fluid, causing weakness, severe fatigue, dark urine and light colored stool (Bleibel et al., 2007). Toxicity in animals may be either through the production of secondary metabolites or by the other organisms, i.e., microbes, plants or other animals hosted by them (Zaynab et al., 2018). More than thousand drugs of the current pharmaceutical era have been shown to cause hepatotoxicity with diverse clinical appearances (Lao, 2020). Several pharmaceutical drugs are used in medical therapy which are hepatotoxic, causes lipid membrane peroxidation resulting hepatocytes injury (Radhika et al., 2014). Several anesthetic drugs like halothane, chloroform, isoflurane, desflurane and nitrous oxide cause direct liver injury, by affecting bilirubin metabolism (El-Shorbagi et al., 2021). Numbers of studies haveA number of chemical toxicants have also been reported for toxicity in animals and humans. Carbon tetrachloride (CCl_4) is one of the toxic agent (Unsal et al., 2020). The level of tissue injury is related to the amount of dose and period of exposure to this toxic agent. Its mechanism of toxicity is based on lipid membrane peroxidation and generation of trichlomethyl radical (•CCl3), causing severe cell injury (Safhi, 2018). Many investigators have given the reports about plants, that having phenolic compounds such as tannins, flavonoids, procyanidins, anthocyanins and phenolic acids have liver, heart and nephroprotective activities (Oliboni et al., 2011). Several studies have revealed that the plant extracts possessing antioxidant activity that defend CCl4 induced hepatotoxicity by preventing peroxidation of lipid and increasing antioxidant Enzyme activity (Shahjahan et al., 2004). Invitro anti-oxidant activates are carried out by using different free radicals including DPPH, Hydrogen peroxide, super oxide, Nitric oxide, trichloromethyl (CCl_3) (Saha et al., 2011). DPPH (2, 2-diphenyl-1- picrylhydrazyl) is a familiar "scavenger") for other radicals (Utkina and Pokhilo, 2012). Herbal remedies that are derived from the extracts of plants are extensively used for the treatment of many illnesses including hepatic ailments (Mohamed Saleem et al., 2010). Hepatoprotective plants consist of variety of chemical components namely phenols, steroids, lignins, monoterpines, caroteniods, lipids, organic acids, essential oil, alkaloids, xanthenes and flavonoids (Madrigal-Santillán et al., 2014). The searches of herbal therapy is a usual practice in developing countries and are commonly used in various illnesses they act as typical healers and these herbal medicines give only health care to most of the people in a protective approach rather than therapeutic (Chang et al., 2007). As a direct curative agent plants are more appealing as compared to advanced medicines because of its easy accessibility and economy (Atanasov et al., 2015).

During the present study the Coconut Oil was selected. The plant belonged to the family Arecaceae –Palm family including of 27 genera and nearly 600 species (Perera et al., 2009; CABI, 2017). Commonly found in Pakistan, Africa and tropical America). It has also been found that some species have antiulcer genic activity (Brito et al., 2018). Therefore in the present research work the mentioned plant i.e. (Coconut Oil was taken for their hepatoprotective activity as the plant has being used traditionally for various disorders.

2. Materials and Methods

2.1. Plant collection

The Coconut Oil was procured from market for further process and where a voucher specimen was deposited in Herbarium of the Department.

2.2. Oil extract

The oil (1500 mL) from coconut fruits was obtained after processing (Ojewumi et al., 2018).

2.3. Animal grouping and experimental regime

Forty (40) adult domestic male rabbits (*Oryctoagus cuniculus*), (700 to 1000 g body weight) were purchased from Rifah Institute of Pharmaceutical Sciences Islamabad. Animal's acclimatization was carried out for 1 week in the laboratory animal house. The animals were provided with standard food as ad libitum fresh water. The animals were kept at room temperature around 22–25 °C with light and dark cycle of about 12 hours. All procedures related to the animals were carried out according to the recommendations of the research animals committee for care and use of (Vasbinder and Locke, 2016). Also approval was taken from the Departmental Animal Ethical Committee of pharmacy university of Malakand.

During the experimentation, a total of forty adult male rabbits were divided into five groups, comprising of eight animals in each group and the experiment was carried out for 21 days.

Group A: (Normal control group) (10 ml/kg body weight of normal saline pre oral each day)

Group B: (Toxic Control group, CCl4, 10 mL) = (administered with CCl_4 at concentration of 10 mL/kg body weight of (50% v/v oral, once a day regularly for 21 days)

Group C: (Standard Control group , Silymarine, 50 mg/kg + CCl4, 10 mL) = (feed oral with silymarin at dose rate 50 mg/kg body weight, dissolved in 10 ml mineral

water once a day regularly for 21 days after intoxication with CCl_4)

Group D: (Coconut Oil 50 mL + CCl4, 10 mL = (treated via oral with Coconut Oil at dose 50 mL/kg body weight, once a day regularly for 21 days after intoxication with CCl4)

Group E: (Coconut Oil 200 mL +CCl4, 10 mL) = (received Coconut Oil at dose rate 200 mL/kg body weight oral, once a day regularly for 21 days after intoxication with CCL4).

2.4. Acute toxicity test

The acute toxicity study was carried out for the *Coconut Oil* at different doses i.e.500, 1000, 1500 and 2000 in mL per kg body weight. Acute toxicity was determined according to the method of (Litchfield Junior and Wilcoxon, 1949) and according to the Organization for economic co-operation and development (OECD) guideline number, 420. During this test, the extract was assigned safe up to highest dose (2000 mL/kg body weight) as no mortality was caused (Chen et al., 2017).

2.5. Animal's dissection

At the completion of experiment all the animals were carried sequentially to the laboratory for the process of dissection and blood collection. All animals were anesthetized via 35 mL/kg pentobar-bital sodium and euthanized by cervical decapitation using procedure according (Cande et al., 2018; Bădărău, 2013).

2.6. Ethical statement

Study was conducted as per approval protocols (notification no: Pharm/EC-Cn/07-02/ 2020-34), in accordance with the animals byelaws 2008, Scientific Procedures Issue-I.

2.7. Blood collection for hematological and biochemical study

Blood collection was carried out via cardiac puncture for studying the biochemical parameters. The blood samples were centrifuged for serum separation at 3500 rpm (Centurion scientific Pvt., Ltd. UK) for 10 min. The serum was analyzed through spectrophotometer (Perkin Elmer; Germany) for investigation of biochemical parameters like serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT) and serum alkaline phosphatase (ALP) via (Human kit;Germany) using UV-Spectrophotometer (Al-Bulushi et al., 2017).

2.8. Histological studies

For histopathological analysis, livers were excised from corresponding group animals and immediately stored in a solution having 10% formalin and 0.9% NaCl. The tissues were then embedded in paraffin, thinly sectioned using a microtome, stained with haematoxylin and eosin (H&E) for conventional morphological assessment, then observed under light microscope (BX50; Olympus, Tokyo). The images were achieved by a digital camera system (Pixcera Co., Osaka, Japan) attached to the microscope. A minimum of 5 fields for each liver and kidney slide were studied and recorded semi quantitatively for severity of changes (Mostafavi, 2019).

2.9. DPPH radical scavenging activity

The *in vitro* antioxidant activities of *Coconut Oil* was evaluated on the basis of 2, 2-diphenyl-1-picryl-hydrazyl (DPPH) scavenging assay. The concentration of solutions prepared for the activity were expressed as parts per million (ppm), equal to mg/L. A 25 ml stock solution of 500 ppm of extract was prepared in methanol. From the stock solution; a 5ml solution each of 20, 40, 80,100 and 200 ppm was prepared in separate test tubes. Each concentration was taken in triplicate. The same procedure was repeated for ascorbic acid which was used as standard using 1700 Shimadzu Japan) at 517 nm for absorbance. The following formula was used: Percent radical scavenging activity = (Ac – As / Ac) × 100, where Ac is the absorbance of control, As is the absorbance of sample (Sarker and Oba, 2020).

2.10. Statistical analysis

The data (expressed as mean ± SE) were analyzed by one way ANOVA and "Tukey test" using SPSS software. Values of $p < 0.05$ were considered to be statistically significant.

3. Result

3.1. Biochemical parameters

The effect of Coconut Oil on the values of liver enzymes such as ALT, AST and ALP are presented in (Table 1). The results indicate that CCl_4 administration significantly ($P < 0.05$) increased the serum levels of ALT, AST and ALP as compare with control group A. After treatment with Coconut Oil at 200 mL/kg body weight, significantly ($P < 0.05$) lowered the concentration of ALT, AST and ALP (group D and group E) and caused a consequent normalization. The results of coconut oil treated groups were comparable with that of silymarin (50 mg/ kg body weight) administered rabbits (group C) (See Table 1) revealed that treatment is dose dependent i.e. high dose (200 mL) oil seems to be better in the recovery of hepatic injury when compared with the results of toxic control rabbits (group B) that received CCl_4 alone confirmed the healing effects of *Coconut Oil*.

3.2. Hematological parameters

The blood hematology study included Red blood cells (RBC), White blood cells (WBC), Platelets, hemoglobin (Hb), hematocrit (HCT) and mean corpuscular hemoglobin concentration (MCHC). The mean values of RBC WBC, Platelets and hemoglobin (Hb) of rabbits of (group A), that received normal saline only was in the normal range. A significant ($P < 0.05$) reduction in the activities of RBC, WBC, Platelets and hemoglobin (Hb) were observed in rabbits that received CCl_4 alone (group B), when compared to normal control rabbits. (Table 2) showed no-significant value ($p>0.05$) difference in the concentration of hemoglobin, RBC and Platelets of groups D and groups E rabbits treated with graded doses of 50mL/kg and 200mL/kg body weights *coconut oil* when compared with group A (normal control) rabbits. Also group C (standard control) rabbits that received a

Table 1. Effects of *Coconut oil* on liver function enzymes in CCl4- intoxicated rabbits at (P < 0.05) of variance.

Liver parameters	Group A	Group B	Group C	Group D	Group E
	(n=8)	(n=8)	(n=8)	(n=8)	(n=8)
Serum ALT (IU/L	27.2 ± 3.7	167.5 ± 3.42	63.7 ± 3.7	53.7 ± 7.0	67.5 ± 3.27
Serum AST (IU/L)	42.5 ± 5.8	281.5± 2.6	85.5 ± 4.9	59.8 ± 7.6	51.2 ± 0.85
Serum ALP (IU/L)	45.8 ± 3.56	143 ± 3.7	47.2 ± 2.23	41.2 ±2.65	91.5 ± 3.06

Group A= normal control (10 mL/kg saline water), Group B= CCl4 = 50 mL/kg, Group C= Coconut Oil (50 mL/kg) (CCL4= 50 mL/kg, Group D= Coconut Oil (200 mL/kg) + (CCL4 = 50 mL/kg, Group E= (silymarine =100 mL/kg + CCL4 50 mL/kg.

Table 2. Effects of *Coconut oil* on hematological parameters in CCl4- intoxicated rabbits (P < 0.05) of variance.

Hematological indices	Group A	Group B	Group C	Group D	Group E
	(n=8)	(n=8)	(n=8)	(n=8)	(n=8)
RBCs levels x103/µL	4.9 ± 0.09	4.2 ± 0.09	5.3 ± 0.13	5.4 ± 0.04	5.6± 0.04
WBC levels x103/µL	5.8 ± 0.34	3.97 ± 0.08	6.00 ± 0.10	5.65 ± 0.06	5.90 ± 0.25
Platelets levels G/dL	2.39 ± 0.64	1.41 ±1.49	1.37 ± 0.85	1.28 ±0.85	1.38 ± 1.19
Hemoglobin levels G/dL	11.47 ± 0.45	10.87 ± 0.63	10.00 ± 3.27	12.15 ± 0.41	11.20 ± 0.51
Hematocrit values %	37 ± 2.0	24.20 ± 0.31	27± 2.3	36.11± 0.21	35 ± 1.41
(MCH) values G/dL	31.60 ± 0.79	20.50 ± 0.84	21.12± 1.05	23.54± 0.85	26 ± 0.74

Group A= normal control (10 mL/kg saline water), Group B= CCl4 = 50 mL/kg, Group C= Coconut Oil (50 mL/kg) (CCL4= 50 mL/kg, Group D= Coconut Oil (200 mL/kg) + (CCL4 = 50 mL/kg, Group E= (silymarine =100 mL/kg + CCL4 50 mL/kg.

Table 3. Showing acute toxicity test results .

Groups	500 mL/kg body weight	1000 mL/kg body weight	1500 mL/kg body weight	2000 mL/kg body weight
Group A	Healthy	Healthy	Disease/live	Death
Group B	Healthy	Healthy	Disease / death	Death
Group C	Healthy	Diseases/live	Disease/live	Death
Group D	Healthy	Healthy	Healthy	Disease/live
Group E	Healthy	Healthy	Healthy	Disease/live

standard anti-oxidant drug, silymarine showed no-significant changes in the values of hemoglobin, RBC and Platelets were compared with group B (toxic control group). However, the concentration of WBC was raised with the treatment of *coconut oil* at dose 50 mL/kg body weight and 200 mL/kg body weight. The results of treated rabbits (group D and E) were somewhat near to the results of silymarine although silymarine is slightly better (Table 2). In addition (Table 2) revealed that the administration of *coconut oil* at the doses of 50 mL/kg and 200 mL/kg have no significant curative effect on HCT and MCHC values of rabbits intoxicated with CCl4 before the administration of the oil. No significant changes were observed in hematological parameters with the administration of coconut oil and Silymarine except WBC, which was improved with extract administration (Table 2). The acute toxicity showed that the coconut oil is sfafe up to 1500 mL/ kg body weight respectively. The results of acute toxicity test were shown in (Table 3).

3.3. DPPH activity

The results for antioxidant activity against DPPH of *coconut oil* various concentrations are shown in (Table 4).

Table 4. The antioxidant activity of *Coconut oil* concentrations on the basis of Percent inhibition of DPPH free radical (P < 0.05) of variance.

Coconut oil Conc	% Inhibition	Mean ± SEM
10 ppm	35.66%	32.70 ± 1.14
30 ppm	41.96%	40.70 ± 2.23
50 ppm	51.71%	52.70 ± 2.11
150 ppm	62.70%	60.76± 1.1
250 ppm	71.55%	70 ± 0.79

The percent inhibition values were: 35.66%, 41.96, 51.71%, 62.70%, and 71.55%. At lowest concentration (10 ppm) percent inhibition was 35.66%, followed by 30ppm, 50ppm, 150 ppm and 250 ppm respectively. The increased in extract concentration caused an increase in percent inhibition. This increase in percent inhibition represents the antioxidant potential of *coconut oil*.

The average initial and final weight rabbits of various experimental groups were given in (Table 5). Results

indicates that the weight was significantly restored at high dose of coconut oil administration to the animals of group D showed the improve impacts on the health of experimental animals.

3.4. Histological examinations

Photomicrographs taken from liver sections of all experimental animals (Figure 1 (A1) to (A5) respectively). Liver sections from control rabbit showed normal hepatic cells with well-preserved cytoplasm, prominent nucleus, nucleolus, central vein and compact arrangement of hepatocytes (Figure 1 (A1). In contrast to this, CCl4 caused hydropic alteration and necrosis in centrilobular hepatocytes (Figure 1 (A2). Sinusoids and central vein Congestion clarified acute inflammatory cells infiltrating. In animals treated with silymarin, show histoarchitecture with mild infiltration of inflamed cells (Figure 1 (A3) than the CCl4 group. The rabbit groups treated with *coconut oil* at the doses of 50 mL/kg body weight and 200 mL/kg body weight, tissue damage and necrosis were of less extent and no disorder was observed at hepatocyte cords (Figure 1 (A4) and (Figure 1 (A5). The scoring of histological damage is displayed in (Table 6).

The present study was aimed to determine the effects of *Coconut Oil* on kidney related serum biochemical parameters such as (URE), uric acid (UA), creatinine (CR) and albumin (ALB), blood urea nitrogen (BUN) and glucose (Table 7). Also the concentration of serum sodium, potassium and magnesium ions in control rabbits were found in normal range. All these serum electrolytes were found to have lower limits in the paracetamol ingested rabbits showing a significant decrease ($P < 0.05$). The treatment of *Coconut Oil* at dose 50 ml/kg body weight and 200 ml/kg body weight showed no significant changes in the serum electrolytes concentration during the three weeks of treatment. Similarly the silymarine was also found to have no significant effects on the all serum electrolytes concentration during the first two weeks of experiment, however a significant curative effects was observed on the serum electrolytes (Table 7).

Table 5. Average initial and final weight rabbits of various experimental groups.

Dosage mL/kg body weight	Initial body weight (g/kg body weight) (n=8)	Final body weight (g/kg body weight) (n=8)
Control animal, Group-A	328.66 ± 0.43	332.40 ± 2.14
CCl4 Control, Group-B	331.96 ±0.12	222.60 ± 3.12
CCl4+ Coconut oil, Group-C	332.71 ±1.23	317.30 ± 1.12
CCl4+ Coconut oil, Group-D	329.70 ±2.12	324.32± 3.4
CCl4+ Silymarine, Group-E	331.22 ±1.12	329 ± 2.32

4. Discussion

The current study aimed to reveals the hepatoprotective, curative and antioxidant effects of *Coconut Oil* against CCl4-

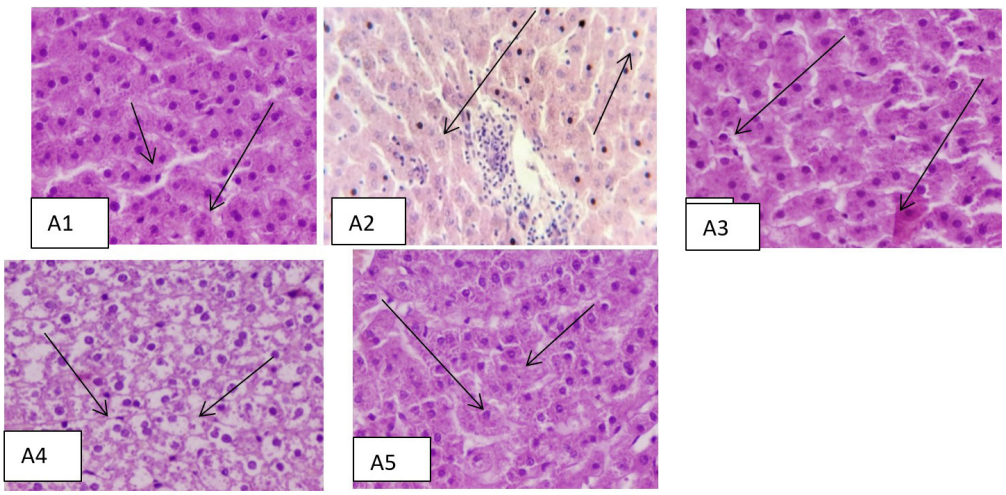

Figure 1 (A1, A2, A3, A4 and A5) shows liver histological micrographs of all experimental rabbit groups, intoxicated with CCl4 and treated with Coconut oil at various doses. Explanation: Figure 1 (A1), (Group A): Histological structure of rabbit liver sections from a control group the pointed arrows suggests normal histoarchitecture and normal mitochondria and cellular nuclei. Figure 1 (A2), (Group B): Histological structure of rabbit liver section from toxic control group (received CCl4 only). The mentioned arrows showing necrosis and infiltration of hepatocytes arrows show damage area. Figure 1 (A3), (Group C): Section of rabbit liver treated with *Coconut oil* 50 ml/kg BW. The pointed arrows depicted mild infiltration of inflammatory cells along with mild necrotic areas. Figure 1 (A4), (Group D): Histological structure of rabbit liver treated with *Coconut oil* of 200 ml/kg BW. The pointed arrow suggests improvement in the liver tissue and regulation of hepatocytes morphology. Figure 1 (A5), (Group E): Histological structure of rabbit liver section from silymarine control (100 mg/kg BW) group. The arrows marked mild hepatocellular architecture normalization and regulation of necrosis and normal cellular histoarchitecture.

Table 6. Semiquantitive score of histopathological findings.

Groups	Hydropic degeneration	Liver steatosis	Inflammatory cell infiltration	Necrosis
Group A	0	0	0	0
Group B	+++	++	+++	+++
Group C	+	0	0	+
Group D	++	0	+	+
Group E	+	0	0	+

Damage grade are as follow: 0 = no abnormality, + = mild injury, ++ = moderate injury and +++ = severe injury.

Table 7. Effects of *Coconut oil* on kidney function tests in CCl4- intoxicated rabbits ($P < 0.05$) of variance.

Parameters (mg/dl)	Group A	Group B	Group C	Group D	Group E
Creatinine	0.25 ± 0.05a	0.86 ± 0.41b	0.39 ± 0.8a	0.61 ± 0.05c	0.34 ± 0.00a
Urea	26.03 ± 0.1a	60.42 ± 1.3b	28.61 ± 1.43a	42.43 ± 4.1c	33.77 ± 1.6a
Uric acid	0.35 ± 0.03a	0.68 ± 0.02b	0.50 ± 0.03a	0.77 ± 0.00c	0.30 ± 0.00ac
BUN	13.00 ± 0.41a	30.01 ± 0.00b	17.45 ± 1.75a	21.967 ± 0.0c	16.03 ± 0.43a
Albumin	3.00 ± 0.00a	4.98 ± 0.11b	3.03 ± 0.62a	5.11 ± 0.15c	3.00 ± 0.11a
Glucose	89 ± 5.40a	140 ± 2.13b	102 ± 2.63a	114 ± 2.33c	101 ± 3.00ac
Ca (mmol/L)	5.00 ± 0.12a	8.99 ± 1.76b	5.00 ± 0.01a	5.10 ± 0.23c	4.12 ± 0.20ac
Mg (mmol/L)	0.52 ± 0.2a	0.90 ±0.9b	0.48 ± 0.11a	0.35 ± 0.11c	0.38 ± 0.01a
Cl (mmol/L)	88.3 ± 4.21a	126.3 ± 0.1b	103.3 ± 3.3a	102.9 ± 4.3c	101.1 ± 1.2ac
P (mmol/L)	1.63 ± 0.08a	2.00 ± 0.00b	2.00 ± 0.01a	1.21 ± 0.00c	1.22 ± 0.00a
K (mmol/L)	3.99 ± 0.31a	8.12 ± 0.80b	6.23 ± 0.0a	5.33 ± 0.21c	5.23 ± 0.34a
Na (mmol/L)	140.4 ± 0.4a	149.8 ± 4.2b	132.1 ± 0.21a	155.1 ± 1.06c	132.5 ± 0.11a

group A= normal control (10 mL/kg saline water), Group B= CCl4 = 50 mL/kg, Group C= Coconut Oil (50 mL/kg) (CCL4= 50 mL/kg, Group D= Coconut Oil (200 mL/kg) + (CCL4 = 50 mL/kg, Group E= (silymarine =100 mL/kg + CCL4 50 mL/kg.

induced liver injury. For the screening of hepatoprotective drugs, frequently CCl4 is used as hepatotoxic agent. CCl4 is break down to the trimethyl radical (CCl3) and a proxy trichlomethyl radical (OOCCl3) by cytochrome P 450 2EI enzyme (Ritesh et al., 2015). These radicals bind covalently to the macromolecules and probably caused lipid peroxidation by attacking membrane polyunsaturated fatty acids, there by disturbing membrane integrity and caused hepatic damage associated with oxidative stress (Horton et al., 1987; Vona et al., 2021). Liver damage is evaluated by assessing the concentration of discharged transaminases including ALT, AST and ALP in blood (Gu et al., 2020). Results of the present study revealed increased in serum ALT, AST and ALP levels in carbon tetra chloride (CCl_4) administered rabbits when compared with normal control rabbits ($P<0.05$).

Abrupt increase in serum levels of ALT, AST and ALP is believed to be a significant indicator of CCl4 induced severe liver injury (Chand et al., 2021). The statement was confirmed by necrosis and infiltration of inflammatory cells during histopathological examination of microphotographs of liver sections, it is reported that several pharmaceutical drugs like rifampicin, isoniazid, paracetamol, etc. are used in medical therapy are hepatotoxic, producing free-radical that causes lipid membrane peroxidation resulting hepatocytes injury (Foster et al., 2020). During the present research the rabbits intoxicated with carbon tetrachloride (CCl_4) were treated with a standard antioxidant drug, Silymarine and *Coconut Oil, of* graded doses. Silymarine is a standard antioxidant drug and frequently used as a hepatoprotective medicine, derived from a plant, *Silybum marianum* (Frățilă et al., 2020; Samudram et al., 2008). In this study administration of Coconut oil, at the doses of 50 mL/kg and 200 mL/kg to CCl4 intoxicated rabbits reduced CCl4 induced elevation toward normal or only slightly elevated of serum ALT, AST and ALP indicating protection against liver damage. This means coconut oil exhibited significant hepatoprotective potential against CCl4, which is nearly equivalent to the standard drug, Silymarine. The current study is in agreement with that of (Assadi et al., 2021). Who investigated the liver protective activity of the alcohol extract of *Capparis sepiaria* stem against CCl4 intoxicated Albino rats. The liver sections of rabbits treated with *coconut oil* after CCl_4 intoxication are revealed to have amended cellular membrane architecture or less damage to the hepatic cells as compared to rabbits treated with CCl4. The improved histoarchitecture further verify the liver preventive potential of the *coconut oil*

and support the results of biochemical parameters. The preventive consequence of the oil is attributed through antioxidant or free radical scavenging potential and healing effects were possible due to alkaloids, flavonoid and saponins constituents of *coconut oil*. This study claims that future investigation should be required on *coconut oil* because its 200 mL/kg body weight concentration showed a significant hepatoprotective potential against CCl4, which is nearly equivalent to the standard silymarine medicine which used 50 mL/kg while oil may contain few milligrams of bioactive compound. Previously the fruit, stem and leaves of coconut plant have been explored for antioxidant potential and phenolic contents (Naz et al., 2021; Pradhan et al.; 2021). During the present study the coconut oil was subjected for their in-vitro antioxidant activity against DPPH free radical scavenging activity and increase in the percent inhibition was observed with increase in extracts concentration further conformed that the coconut oil has antioxidant scavenging activity.

5. Conclusion

From the result of the present study it was concluded that the Coconut Oil is most effective against toxicity in general and specifically it is hepatoprotective and can be used as a remedy for various illness. These oil is l for most of hematological and lipid profile ers. Histopathological results further confirmed the effectiveness of *Coconut Oil* indicating almost similar results like silymarine.

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