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EFFECT OF THE ETHANOL EXTRACT OF *PORIA COCOS* ON LIVER INJURY INDUCED BY CARBON TETRACHLORIDE IN ADULT MALE RATS

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ABSTRACT

Poria cocos (*P.cocos*) has long been utilized in alternative medicine due to various desired effects on body health. This study aimed to evaluate the hepatoprotective potential of ethanol extract of *P.cocos* (EEPC) against carbon tetrachloride (CCl_4) induced liver injury in rats. A total of 40 male rats were randomly divided into four groups (n=10). 1) The control group administered olive oil (0.5 ml/kg/day, orally), 2) the CCl_4-induced liver damage rats received CCl_4 dissolved in olive oil (0.5 ml/kg orally), twice weekly for one month, 3) the EEPC-treated group (250 mg/kg/day, orally) for one month and 4) CCl_4 + EEPC-treated group co-administered CCl_4 (0.5 ml/kg) + EEPC (250 mg/kg/day) for one month. The biochemical analysis revealed that CCl_4 exposure significantly elevated liver enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transpeptidase (GGT) activities. Furthermore, an increase in the inflammatory markers, such as tumor necrosis factor-alpha (TNF-α), Interleukin -6 (IL-6) and heat shock protein70 (Hsp70) and the lipid profile, along with the oxidative stress (MDA), as well. Moreover, antioxidants such as glutathione, superoxide dismutase, and catalase (CAT) were depleted in tissues. Treatment with EEPC resulted in a significant correction of the disrupted values caused by CCl_4 exposure. A significant reduction was detected in liver enzyme activity (ALT, AST, GGT), inflammatory cytokines (TNF-α, IL 6, Hsp70), lipid peroxidation (MDA) and blood lipids, total cholesterol (TC) and triglycerides (TG). In addition, tissue antioxidants (GSH, SOD, CAT) were restored to the normal levels. In conclusion, ethanol extract of *P. cocos* effectively mitigates CCl_4-induced liver injury in rats by adjusting liver enzymes, suppressing inflammation and enhancing antioxidant capacity. These highlight the potential of *P. cocos* as a promising natural therapeutic for acute liver diseases and related consequences.

Keywords: *Poria cocos*, Carbon tetrachloride, Oxidative stress, Liver enzymes, Antioxidants

INTRODUCTION

Traditional Chinese medicine has long held the medicinal fungus *Poria cocos* (Schw.) Wolf, a member of the Polyporaceae family, is highly esteemed. It is known as Fu Ling in Chinese. This fungus is primarily harvested from the sclerotia of the fungus, which grows on the roots of pine trees. Historically, *P. cocos* has been prescribed for its diuretic, sedative, and tonic properties, and was often used to treat ailments such as edema, insomnia, and spleen deficiencies (Wang *et al.*, 2004; Ríos, 2011). Modern pharmacological research has established a lot of its regular

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applications by determining its bioactive components responsible for its therapeutic effects. Key components include triterpenoids (e.g., pachymic acid, poricoic acid), polysaccharides and β-glucans, as mediators of its therapeutic benefits (Zhou *et al.,* 2008). These compounds possess anti-inflammatory, antioxidant, antitumor and immuno-modulatory functions, making *P. cocos* a flexible choice for the management of chronic diseases associated with oxidative stress and inflammation (Li *et al.*, 2019; Fu *et al.*, 2021).

The hepatoprotective activities of *P. cocos* are extensively examined in recent years. For instance, polysaccharides isolated from *P. cocos* can mitigate induced liver fibrosis through inhibiting hepatic stellate cell activation and reduce collagen deposition in rats (Wang *et al.*, 2018). Triterpenoid-abundant extracts, likewise, showed a powerful antioxidant activity against acetaminophen-induced liver injury, by restoring glutathione (GSH), and super-oxide dismutase (SOD) activity (Adetuyi *et al.*, 2022).

These findings align with the growing trend toward using natural products as alternatives to synthetic pharmaceuticals with unwanted side effects. The emerging role of *P. cocos* in modern pharmaco-therapy, particularly for liver disorders, reflects a relationship between empirical tradition and evidence-based scientific approaches.

CCl_4 remains among the most common chemicals to experimentally induce hepatotoxicity. Its mechanism of action is well characterized and considered a gold standard in liver injury research for evaluating of potential therapeutic drugs. When administered, CCl_4 engages in cytochrome P450 2E1 (CYP2E1) mediated metabolic process in the liver to create a highly reactive trichloromethyl (' CCl_3) along with trichloromethyl peroxy (' $OOCCl_3$) radicals (Weber *et al*., 2003). These radicals trigger a cascade of oxidative damage by attacking poly-unsaturated essential fatty acids in cellular membrane leading to lipid peroxidation. The resulting malondialdehyde (MDA) is a byproduct of lipid peroxidation and considered a key marker of oxidative damage (Recknagel *et al*., 1989; Ibrahim & Abd EL-Maksoud, 2025). Additionally, CCl_4-induced liver injury triggers an inflammatory response, characterized by elevated levels of pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF-α), Interleukin -6 (IL-6) which aggravate tissue damage (Kisseleva &Brenner, 2021).

Notably, depletion of endogenous antioxidants (GSH, SOD, and CAT) is a crucial event of CCl_4 toxicity. GSH is a tripeptide thiol crucial for the neutralization of free radicals and redox balance. Its decrease upon CCl_4 exposure exposes hepatocytes to oxidative assault, and reduced CAT and SOD activities hinder the cell's detoxification of superoxide anions and hydrogen peroxide (H_2O_2) (Szymonik-Lesiuk *et al*., 2003). These biochemical alterations mirror the pathophysiology of human liver disorders including non-alcoholic steatohepatitis, alcoholic liver disease along with drug induced liver injury and validate CCl_4 as a translational model for clinical trials.

Despite the strong evidence of *P. cocos* hepatoprotective activities, its mechanisms in the context of CCl_4-induced injury are not yet fully understood. Almost all previous scientific studies were performed with isolated compounds (e.g., triterpenoids or polysaccharides) and the synergistic effects of the entire extract were not explored. In addition, oxidative stress, inflammation and metabolic dysregulation in CCl_4 toxicity call for an alternative evaluation.

This research was conducted to investigate the ethanol extract of *P.cocos* as a multimodal therapeutic agent against CCl_4-induced liver injury such as its effects on

liver function enzymes (ALT, AST, GGT), inflammatory cytokines (TNF-α, IL-6, Hsp70), and oxidative stress markers (MDA, GSH, SOD, CAT). Furthermore, its influence on metabolic parameters (glucose, total cholesterol, triglycerides) to elucidate systemic benefits. By addressing these objectives, this work seeks to advance the translational potential of *P. cocos* in managing physiological and metabolic liver injuries.

MATERIALS AND METHODS

1 Animals

Forty adult male *Albino* rats were used in this study, aged 2-3 months with body weight of (200-230 g). All animals were obtained from (AL-Nahrain University/ Biotechnology Research Center). Animals were kept in standard polypropylene cages (20×25×20) cm with wood chip bedding. For one week, animals were subjected to an environment with a temperature of 25 ± 3ºC, a humidity of 55 ± 5%, and a light/dark cycle of 12:12 hours. All animals were given an unlimited supply of food and water.

2. Chemical Reagent

CCl_4 was obtained from Sigma Aldrich Chemical Co., (St. Louis, MO, USA), and used to induce hepatic damage type centrolobular damage (zone3) with steatosis.

3. Preparation of the EEPC

The raw *P. cocos* was purchased from the local market, washed and allowed to dry air and ground into fine powder before being extracted. We used 100 g of the plant for the extraction, which was performed in ambient conditions for a duration of four hours using 75% ethanol. The crude extract was then concentrated and condensed into powder using the freez dryer technique (Kim *et al.,* 2022). The obtained powder was then dissolved into deionized water at an optimum concentration to match the required dose.

The percentage of the extracted powder was calculated as follows:

Extracted powder%= weight of raw plant / weight of extract *100

=16.84g/100g*100

=16.84%

4. Experimental Design

Forty adult male rats were randomly divided into 4 groups (ten /group) received the following treatments:

1. ***Group1:*** (Control group) rats in this group received olive oil (0.5 ml/kg. B.W.) for one month by gavage needle
2. ***Group 2:*** Liver injury-induced group (CCl_4 group), liver injury was induced in rats according to (Mansour et al. 2022), where rats received 0.5 ml/kg B.W. CCI_4 dissolved in olive oil in ratio 1:3 v/v twice a week for one month by oral gavage.
3. ***Group 3***: (EEPC) Rats in this group received EEPC (250 mg/kg. B.W.), by gavage needle daily for one month
4. ***Group 4***: (CCl_4+ EEPC): Rats in this group received EEPC (250 mg/kg. B.W.), by gavage needle daily and CCl_4 twice a week at a dose (0.5 ml/kg. B.W.) CCl4 dissolved in olive oil in ratio 1:3 for one month at the same time as EEPC administration.

5. Sample collection

5.1. Blood sample

After the trial was over and at least 24 hours had passed after the last dosage, rats were anesthetized using diethyl-ether and blood samples were taken from a retro-orbital vein using heparinized capillary tubes. Prior to analysis, serum samples were preserved at -20 °C following centrifugation at 3000 rpm for 10 minutes.

Figure 1. Experimental Workflow for Evaluating Hepatoprotective Effects of EEPC

5.2. Liver sample

After collecting blood samples, all rats were sacrificed by cervical dislocation. The liver was prepared by removing any blood, washed with phosphate buffer saline, homogenized, and then centrifuged. The hepatic cells oxidative stress parameter was measured using the supernatant recovered after centrifugation.

6. Biochemical Serum Analysis

The Serum amounts of (ALT, AST, GGT, GLU, TC and TG) have been calculated based on the manufacturer's process as mentioned by the kit (Randox, Northern Ireland). The serum levels of TNF-α, IL-6 and Hsp70 were determined by ELISA (Sunlong Biotech Co. Ltd, China).

7.Hepatic cells oxidative stress parameter

Oxidative stress was assessed on the supernatant of liver homogenate. The lipid peroxidation (MDA) amount in the liver has been based on the technique discussed in (Gilbert *et al*., 1984). The technique of (Moron *et al*., 1979) was utilized to figure out glutathione and also the techniques of (Marklund & Marklund,1974) and (Aebi 1983) have been applied to analyze liver (SOD) and (CAT) activity, respectively.

8. Statistical Analysis

All data of the study were exposed to statistical analysis using IBM SPSS Statistics 23rd edition. One- way (ANOVA test) was performed followed by Duncan Multiple test to find significance among means ($P \leq 0.05$).

9. Ethical approval

The experimental study was approved by the ethical committee of Dentistry College, Al-Iraqia University, with number (ESA & HER-05-15-05-5-24).

RESULTS

1. Effect of EEPC on liver enzymes in rats with induced liver damage by CCl_4

ALT, AST and GGT activities are outlined in Table (1). The treatment with EEPC alone did not affect the activity of liver enzymes examined in this study, compared with the control. The activity of the 3 enzymes examined showed the same significant ($P \leq 0.05$) elevation as a result of the administration of CCl_4. The treatment with EEPC reduced the elevated levels of these enzymes in the fourth group. However, the values still statistically ($P \leq 0.05$) higher than the control.

Table 1: Effect of EEPC on liver enzymes in rats exposed to liver damage induced by CCl_4.

Parameters / Treatments	ALT (U/L)	AST(U/L)	GGT(U/L)
Control	44.78 ± 3.27 c	73.36 ±4.54 c	26.75 ± 2.74 c
CCl_4(0.5ml/kg.b.w.)	141.81±7.02 a	137.20 ± 5.56 a	66.40 ± 3.32 a
EEPC (250mg/kg.b.w.)	43.35 ± 2.30 c	73.87 ± 4.74 c	32.18 ± 4.53 c
CCl_4 (0.5ml/kg.b.w.) + **EEPC** (250mg/kg.b.w.)	114.31±7.66 b	103.94 ±6.91 b	44.08 ±3.73 b

Values are presented as Mean±**SE** (Standard error) n = 10 in each group,
Different letters in the same column refer to significant differences ($P \leq 0.05$)
CCl_4 (carbon tetrachloride), **EEPC** (ethanol extract of P.cocos), **ALT** (alanine aminotransferase), **AST** (aspartate aminotransferase), **GGT** (gamma-glutamyl transpeptidase)

2. Effect of EEPC on some inflammatory markers in rats exposed to liver damage, induced by CCl_4.

In the CCl_4 treated group, the level of TNF-α was noticeably higher ($P \leq 0.05$), (Table 2). The treatment with EEPC alone did not change TNF-α significantly than the control level. The administration of EEPC along with CCl_4 lowered the TNF-α, however no significance compared to the CCl_4 group on one side and the normal control group on the other side.

Table 2: Effect of EEPC on some inflammatory markers in rats exposed to liver damage, induced by CCl_4.

Parameters / Treatments	TNF-α (pg/ml)	Hsp 70 (ng/ml)	IL-6 (pg/ml)
Control	10.46 ± 1.6 b	22.43 ± 1.07 c	5.68 ± 0.42 c
CCl_4 (0.5ml/kg.b.w.)	24.8± 2.2 a	48.26± 3.19 a	19.90 ± 0.61 a
EEPC (250mg/kg.b.w.)	15.6±1.3 b	25.44±2.12 c	6.12 ± 0.38 c
CCl_4(0.5ml/kg.b.w.)+**EEPC**(250mg/kg.b.w.)	19.6± 2.3 ab	36.13± 2.5 b	10.36 ± 0.91 b

Values are presented as Mean± **SE** (Standard error) n = 10 in each group
Different letters in the same column refer to significance ($P \leq 0.05$)
CCl_4 (carbon tetrachloride), **EEPC** (ethanol extract of *P.cocos*), **TNF-α** (tumor necrosis factor-alpha), **Hsp70** (heat shock protein70), **IL-6** (Interleukin -6).

On the other hand, the Hsp70 concentration elevated dramatically ($P \leq 0.05$) with the treatment with CCl_4, compared to the control. The treatment with CCl_4 along with EEPC significantly reduced the Hsp-70 level compared to the CCl_4-treated. However, was statistically ($P \leq 0.05$) higher than the control. Worth mentioning, the

value of Hsp-70 observed in Table (2) in the treatment with the EEPC extract only, mimicking the control group.
Furthermore, we found an elevation in the concentration of IL-6 due to the treatment with CCl_4, which recovered upon treatment with EEPC.

3. Effect of EEPC on some metabolites in rats exposed to liver damage, induced by CCl_4.

To further investigate the effect of EEPC on liver function tests, additional markers in this context were determined. The concentration of GLU, TC and TG was investigated (Table 3). Regarding the GLU and TC, the only significant ($P \leq 0.05$) increase was observed in the CCl_4-treated group, whereas the other groups were statistically ($P \leq 0.05$) similar to each other indicating an ameliorative effect of EEPC to the liver damage caused by CCl_4. CCl_4-treated male rats significantly elevated the concentration of TG in the blood decreased significantly ($P \leq 0.05$) (Table 3). After liver damage was treated with EEPC, the TG concentration was restored to a level close to the control level.

Table 3: Effect of EEPC on some metabolites in rats exposed to liver damage, induced by CCl_4.

Parameters / Treatments	GLU (mg/100 ml)	TC (mg/100 ml)	TG (mg/100 ml)
Control	73.5400±3.80 b	64.23±2.51 b	80.54±3.708 bc
CCl_4(0.5ml/kg.b.w.)	85.3220±4.66 a	85.47±2.85 a	113.60±4.79 a
EEPC(250mg/kg.b.w.)	71.2576±2.07 b	67.01±3.67 b	76.45±3.55 c
CCl_4(0.5ml/kg.b.w.)+ **EEPC**(250mg/kg.b.w.)	68.3520±3.38 b	66.39±4.06 b	90.70±5.25 b

Values are presented as Mean± **SE** (Standard error) n = 10 in each group
Different letters in the same column refer to significance ($p \leq 0.05$)
CCl_4(carbon tetrachloride), **EEPC** (ethanol extract of *P.cocos*), **GLU**(glucose),**TC**(triglyceride),**TG** (total cholesterol)

4. Effect of EEPC on oxidative stress markers in CCl_4 induced liver damage

Given that EEPC may possess antioxidant properties to counteract the oxidative injury induced by CCl_4, we extended our laboratory investigation toward the oxidative markers in the liver tissue. The MDA and GSH concentrations in addition to the activities of both SOD and CAT enzymes confirm our above speculations in terms of the EEPC efficiency to restore the liver function (Table 4).

MDA concentration in liver cells was elevated after treating rats with CCl_4, compared to the control. Administration of EEPC (group 4) considerably decreased this action ($P \leq 0.05$). The treatment with only EEPC did not significantly change the MDA concentration in the liver, which suggests that EEPC is more efficient under stress conditions.

The observations of GSH concentration in the liver confirm that of MDA, since GSH serves as an endogenous antioxidant, which is believed to be depleted during stress conditions. The group exposed to CCl_4 revealed a significant ($P \leq 0.05$) decrease, compared to the other three groups.

The activity of CAT and SOD (Table 4), which are the main antioxidant enzymes in the cell, is consistent with each other. A significant ($P \leq 0.05$) reduction in their activity was observed after the treatment with CCl_4, compared to the control group. On the other hand, the treatment with EEPC with CCl_4 resulted in a recovery of the enzymes activity in liver cells to be at the same statistical level as the normal control group.

To summarize our results, treating induced liver injury using EEPC successfully amended most liver functions, inflammatory, and oxidation markers in the rats' blood and cells.

Table 4: Effect of EEPC on some oxidative stress markers in rats exposed to liver damage, induced by CCl_4.

Parameters / Treatments	MDA (nmol/mg. tissue)	GSH (µmol/g. tissue)	SOD (U/mg.tissue)	CAT (U/mg.tissue)
Control	3.7 ± 0.23 c	4.77 ± 0.32 a	6.26 ± 0.32 a	101.80 ±3.81 a
CCl_4(0.5ml/kg.b.w.)	9.65 ± 0.64 a	3.13 ± 0.29 b	4.23 ± 0.26 b	77.68 ±2.26 b
EEPC(250mg/kg.b.w)	4.80 ± 0.42 c	4.11 ± 0.23 a	6.67 ± 0.29 a	99.44 ±5.18 a
CCl_4(0.5ml/kg.b.w.)+ **EEPC**(250mg/kg.b.w)	7.11 ± 0.23 b	4.86 ± 0. 30 a	5.80 ± 0.39 a	94.08 ±2.88 a

Values are presented as Mean± **SE** (Standard error) n = 10 in each group,
Different letters in the same column refer to significance ($p \leq 0.05$)
CCl_4(carbon tetrachloride), **EEPC** (ethanol extract of P.cocos), **MDA**(malondialdehyde),**GSH** (Glutathione), **SOD** (superoxide dismutase),**CAT** (catalase)

DISCUSSION

The hepatoprotective consequences of EEPC against CCl_4 induced liver damage were examined in rats. The results revealed remarkable changes in liver enzyme activities, inflammatory markers, oxidative stress parameters and metabolic profiles showing the therapeutic value of EEPC in liver disease. The ALT, AST and GGT levels were elevated upon CCl_4 administration and reflect oxidative stress and inflammation. Liver damage was induced because CCl_4 produces highly reactive trichloromethyl radicals that attack liver cells leading to cellular damage and enzyme leakage into the blood (Recknagel *et al*., 1989; Weber *et al*., 2003). Previous studies reported similar liver enzyme elevations in CCl_4-induced hepatotoxicity models (Manibusan *et al*., 2007; Almatroodi *et al*., 2020; Shaaban *et al.*, 2023).

Therapy with EEPC in combination with CCl_4 decreased the elevated enzyme levels and brought them back close to control values. This indicates that EEPC has a therapeutic impact on liver damage mitigation and cellular repair processes. This decrease in enzyme levels could be the result of the antioxidant and anti-inflammatory activities of *P.cocos* as it could restore liver function through downregulation of oxidative stress and inflammatory pathways (Zhou *et al*., 2008).

In terms of inflammatory markers, CCl_4 treatment significantly increased the levels of TNF-α IL-6 and Hsp70. This aligns with studies indicating that CCl_4 induced liver injury triggers an inflammatory cascade, exacerbating tissue damage (Kisseleva & Brenner, 2021). EEPC treatment significantly reduced these markers, indicating its ability to suppress the inflammatory response and protect against liver injury (He *et al*., 2022; Jiang *et al*., 2022).

Studies investigated the effects of the active ingredient, such as pachymic acid contained in poria extract, align with the findings of the current study in terms of modulating inflammatory markers, such as TNF-α, IL-6, through controlling macrophage activation, thereby reducing the release of the inflammatory cytokines (Cai *et al.*, 2017; Li *et al.*, 2017).
Additionally, *Poria cocos* extract administrations may inhibit nitric oxide (NO) release in mice tissue culture through modulating the production of NO from macrophages via suppressing the transcription of iNOS gene, consequently

enhancing cellular anti-inflammatory activity (Lee and Jeon, 2003). Another important mechanism is the inhibition of the gene expression of COX-2 enzyme, hence a reduction in prostaglandin PGE2 (Lee *et al.*, 2017).

The mechanism of CCl_4 induced lipid peroxidation is depleting endogenous antioxidants and enhances free radical generation (Almatroodi *et al*., 2020). The results of the oxidative stress markers confirm the hepatoprotective properties of EEPC by reducing MDA levels and also restoring the activity of endogenous antioxidant enzyme including GSH, SOD and CAT, indicating its antioxidant capability. The antioxidative action of *P. cocos* denotes the ability of the extract to down-regulate the gene expression of such pro-apoptotic and/or caspase 3 gene family leading to reduce apoptosis triggered by oxidative stress (Park *et al.*, 2009). More recent evidence also highlight that *P.cocos* polysaccharides likewise promote antioxidant defenses and attenuating oxidative stress (Tang *et al.*, 2021; Zhou, *et al.*, 2024).

It has been illustrated that pachymic acid is able to activate the enzymatic system, AMP-activated protein kinase (AMPK), especially in the adipose tissue and liver cells of rodents. AMPK plays a vital role in mitochondrial bioenergetics, improving ATP-AMP cycle, hence a reduction in ROS (Liu, Ding *et al.*, 2024).

Concerning the metabolic indicators, GLU, TC and TG, were disrupted by CCl_4, which also impaired lipid and carbohydrate metabolism (Taamalli *et al*., 2020; Unsal *et al*., 2021). EEPC therapy has efficiently reduced the lipid profile, normalizing the metabolic status. This underscores the therapeutic effect of *P.cocos* on liver diseases, through restoring metabolic homeostasis (He *et al*., 2022; Jiang *et al*., 2022).

CONCLUSION

In summary, EEPC demonstrated robust hepatoprotective activity through lowering liver enzymes, liver inflammation, and mitigating oxidative damage with lowering inflammatory markers and also correcting metabolic imbalance. These results reinforce earlier results that *P.cocos* could be utilized as a natural therapeutic agent for the prevention and treatment of liver diseases. Future research should focus on clinical trials to validate these effects on humans and further elucidate the molecular mechanism behind EEPC benefits.

LIMITATIONS OF STUDY

Further studies are needed to elucidate the exact mechanisms and long-term effects of EEPC treatment. Additionally , histological examinations are required to provide further insights into the protective effects of *P.cocos* on liver tissue and determined whether *P.cocos* possesses synergistic effects when combined with other hepatoprotective drugs.

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تأثير المستخلص الكحولي لبوريا كوكس على الأذى الكبدي المستحدث بواسطة رابع كلوريد الكربون في ذكور الجرذان البالغة

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يعرف البوريا كوكس من ضمن النباتات المستخدمة في الطب البديل لما له من خصائص طبية متنوعة على صحة الجسم. تضمنت الدراسة الحالية الكشف عن امكانية التاثير الوقائي للمستخلص الكحولي لبوريا كوكس على كبد الجرذان المعاملة برابع كلوريد الكربون. استخدمت في هذه الدراسة أربع مجاميع من ذكور الجرذان البالغة (١٠ جرذان/ مجموعة) وشملت: المجموعة الضابطة وأعطيت زيت الزيتون (٠،٥ مل/ كغم/يوم)، المجموعة الثانية (مجموعة أحداث الاذى الكبدي) تلقت هذه المجموعة (٠،٥ مل/ كغم من وزن الجسم) من رابع كلوريد الكربون المذاب في زيت الزيتون بنسبة 1:3 مرتين اسبوعيا و لمدة شهر عن طريق الفم، المجموعة الثالثة (مجموعة المستخلص الكحولي للبوريا كوكس) عوملت ب(٢٥٠ ملغم/ كغم) وأخيرا المجموعة الرابعة المعاملة بكل من رابع كلوريد الكربون والمستخلص الكحولي للبوريا كوكس (٠،٥ ملغم/ كغم و ٢٥٠ ملغم /كغم على التوالي). اشارت النتائج أن المعاملة برابع كلوريد الكربون قد أحدثت ارتفاع معنوي في فعالية إنزيمات الكبد (الالانين ناقل الامين،الاسبرتيت ناقل الامين وناقل ببتيد كاما كلوتاميل)، مؤشرات الالتهاب (عامل نخر الورم –الفا ، الانترلوكين -6 وبروتين الصدمة الحرارية 70) ، دهون الدم مؤشر الإجهاد التاكسدي (المالونديالديهايد) وانخفاض معنوي في مضادات الأكسدة (الكلوتاثيون، سوبراوكساید ديسميوتيز والكتاليز) في نسيج الكبد. أدت المعاملة بالمستخلص الكحولي للبوريا كوكس الى تصحيح معنوي للاضطراب الحاصل في القيم جراء المعاملة برابع كلوريد الكربون اذ تبين حصول انخفاض معنوي في فعالية إنزيمات الكبد ومؤشرات الالتهاب من السايتوكينات فضلا عن انخفاض مؤشر تزنخ الدهون (المالونديالديهايد) ودهون الدم (الكوليسترول الكلي والكليسيريدات الثلاثية) وارتفاع معايير مضادات الأكسدة في الأنسجة. يستنتج من الدراسة الحالية أن المستخلص الكحولي للبوريا كوكس له تاثير ايجابي فاعل في تصحيح وظائف الكبد، المعايير الالتهابية والقابلية المضادة للأكسدة في الجرذان المعاملة برابع كلوريد الكاربون، مما يسلط الضوء على البوريا كوكس ممكن استخدامها كعلاج واعد لأمراض الكبد الحادة ومضاعفاتها.

الكلمات المفتاحية: بوريا كوكوس ، رباعي كلوريد الكربون ، الإجهاد التأكسدي، إنزيمات الكبد، خصائص مضادة للأكسدة