

Protective Effect of Parsley Oil on the Histological Structure of Liver in Adult Mice Induced by Cadmium Chloride

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Abstract

The purpose of the current investigation is to determine whether parsley oil may mitigate the toxicological effects of cadmium chloride on rat liver cells at the histopathological level. Forty numbers of mice separated into 4 groups each of 10 animals formed the experimental groups.. The first group served as a control and received distilled water. Cadmium chloride was administered intraperitoneally to the second group at a dose of 3.5 mg/kg, the third group was injected intraperitoneally with cadmium chloride at a dose of 3.5 mg/kg and parsley oil was dosed at a volume of 0.5 ml/kg, the fourth group dosed with parsley oil at a volume of 0.5 ml/kg. For a month, the therapy was administered once daily. The results of the histopathological assessment of the hepatic tissue in the cadmium chloride treated group revealed generalized blood vessel congestion, diffuse vacuolar degeneration and coagulative necrosis of the hepatocyte and inflammation. The cadmium chloride and parsley oil group showed recovery of hepatocytes to their normal structure, and the central vein was surrounded by hepatocytes similar to the healthy control group This study showed that parsley oil can counteract the toxic effects of cadmium chloride on hepatocytes, which manifest as necrosis and inflammation, and can lessen the toxic consequences of cadmium chloride from a histological perspective.

Key words: cadmium chloride, liver, parsley oil, mice.

Consuming food contaminated with the metal can raise tissue levels of the chemical cadmium chloride (CdCl), which is prevalent in soil and plants (Deveci and Deveci, 2011). These substances are widely utilized in coating processes, stable PVC manufacture, plastic and glass dyeing, cadmium battery production, commercial phosphate fertilizer production, tobacco and chemical industries, and plastic and glass dyeing (Gabr *et al.*, 2019). When exposed to cadmium compounds, especially CdCl, it spreads quickly throughout tissues and has the potential to be hazardous to both humans and animals (Deveci and Deveci, 2011).

CdCl has been linked in numerous studies to exposure to malignancies in both humans and animals, including those of the thyroid, prostate, breast, pancreas, and urinary bladder. Hepatocellular carcinoma induction in rats (at dosages of 4 and 8 mg/kg for 30 days) is of specific concern (Haouem and El Hani, 2013).

After environmental and occupational exposure, prior studies showed that cadmium toxicity causes its accumulation in the liver and kidney (Rapisarda *et al.*, 2018). Some early indicators of liver dysfunction are seen in studies on the hepatotoxicity of cadmium chloride (El-Shall and Badr, 2012). Rats exposed to cadmium chloride 1.1 mg/kg for 15 and 30 days may suffer substantial damage to their liver, lungs, brain and, kidneys Combined with liver biochemical and structural abnormalities (Renugadevi, and Prabu, 2010). Given cadmium chloride at a dose of 0.5 or 2.0 mg/kg,

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intraperitoneally rats developed lipid peroxidation and hepatic cellular degeneration while a dose of (3.5 mg/kg, i.p. for 15) was demonstrated in another investigation resulting in several metabolic and structural changes in the liver, which are demonstrated by a disruption in the activity of a number of plasma enzymes, including AST, ALT, ALP, and lactate dehydrogenase (Alakilli, 2010).

Mice's hepatic histopathological alterations may occur after 4 weeks of exposure to 150 mg/L of cadmium chloride in water (Karimi *et al.*, 2022). In rats and mice, chronic exposure to cadmium chloride can result in tissue necrosis or apoptosis. One study found that aspartate aminotransferase levels significantly increased after exposure to cadmium chloride in rats at a dose of 5 mg/kg orally for 4 weeks, resulting in an imbalance in the level of liver enzymes AST, alkaline phosphatase ALP, alanine transaminase ALT, and in serum (Alakilli, 2010).

Parsley contains antioxidants, which belong to the Umbelliferae family, and is frequently utilized in the nutrition, pharmaceutical, fragrance, and cosmetics industries. It contains flavonoids and carotenoids, ascorbic acid, myristicin, apiole, terpenoids, and coumarins, according to a phytochemical investigation. Apigenin, a physiologically active flavonoid found in the herb parsley, has antioxidant, anti-inflammatory, and anticancer properties. Due to the presence of β -carotene, this herb has antioxidant and free radical scavenging properties, according to Alakilli (2010). The parsley herb has a number of significant active components, including coumarin, flavonoids, and significant amounts of the vitamins A, C, and B, iron, calcium, and potassium. Parsley's phytochemicals, or carotenoids, have been linked to a lower risk of several chronic diseases in people, including age-related macular degeneration and various cancers (El-Shall and Badr, 2012).

Materials and Methods

Animals: In this study, albino mice were used. Forty adult mice, each weighing between 25 and 30 grams and aged between 12 and 13 weeks, were housed in plastic cages with ten mice per container. The animals were raised

in laboratory conditions that were suitable in terms of temperature and light, and they received continuous and adequate access to food and water throughout the study period (17-19). These processes were approved by the University of Mosul.

Chemicals: A German company's cadmium chloride was utilized (Merck). The parsley oil used in the study was purchased from Mosul's local markets.

Dosage planning: The dosages were created in accordance with the prescribed dose value. Cadmium chloride was administered at a dose of 3.5 mg/kg, at a volume of 10 ml/kg. The dose that was administered was then determined based on the animal's weight. The dosage of parsley oil was 0.5 ml/kg of body weight.

Experiment design: In this study, the animals were placed into 4 groups with 10 mice each, and they received daily, once-daily treatments for a month in the following order:

- The first group was used as a control group, receiving only distilled water.
- Cadmium chloride was administered intraperitoneally to the second group for one month at a dose of 3.5 mg/kg.
- Cadmium chloride was administered intraperitoneally to the third group at a dose of 3.5 mg/kg, along with 0.5 ml/kg of parsley oil.
- The fourth group was administered 0.5 ml/kg of parsley oil.

The animals were given ether anaesthesia following the conclusion of the preceding treatment period in order to conduct an anatomical examination on them and sacrifice them. The mice livers were then removed, thoroughly cleaned with water, and put in 10% diluted formalin till the tissue cutting and other histological procedures were carried out. The sections were stained with hematoxylin and eosin dye. The slides were histologically examined under a light microscope, five fields in each slide were selected at random to demonstrate the apparent histopathological changes in the liver tissue of the experiments treated animals.

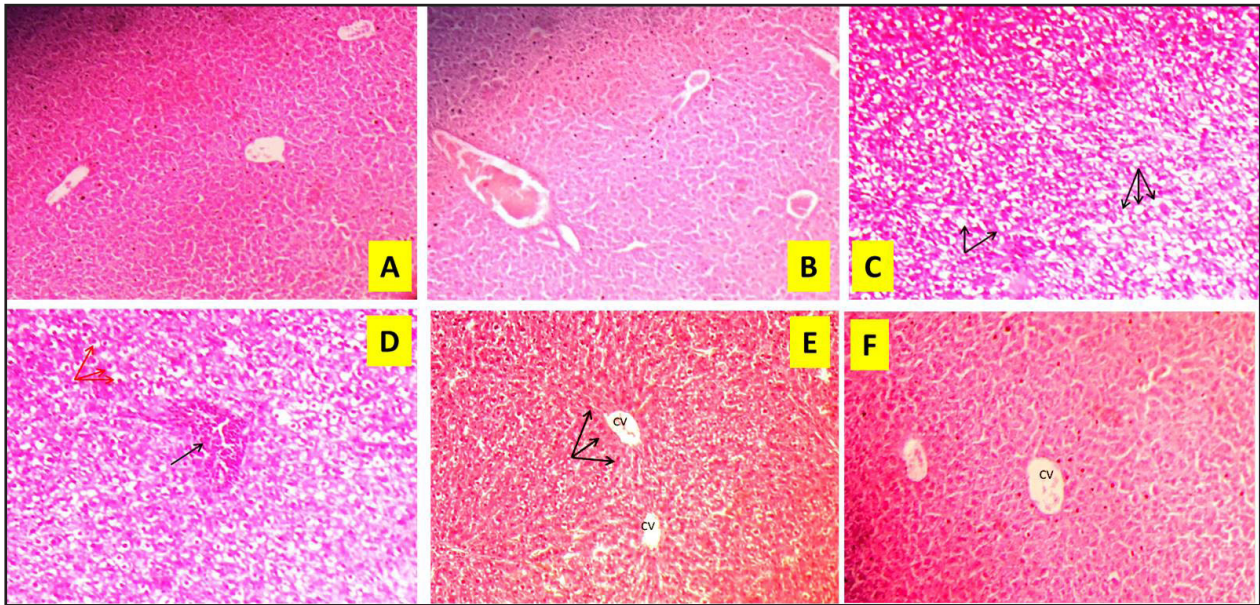


Fig 1. Representative images for potential protective effects of parsley oil against cadmium toxicity. (A) Control group showing central vein (CV) surrounded by the normal architecture of hepatic cords (B) Cadmium chloride-treated group showing generalized blood vessels congestion including veins in the central, veins in the portal area and hepatic artery. (C) Cadmium chloride-treated group showing vacuolar degeneration of the hepatocytes (black arrows). (D) Cadmium chloride-treated group showing infiltration of inflammatory cells (black arrows) with coagulative necrosis of the hepatocytes (red arrows). (E) Cadmium chloride+parsley oil treated group showing partial recovery in the histology of hepatic tissue which appears normal morphology including central vein (CV) surrounded by cords of hepatocytes (black arrows). (F) Parsley oil-treated-only group showing normal appearance of central vein (CV) surround by cords of hepatocytes. H&E stain, 100X.

Results

The control group's histopathological analysis revealed that the liver tissue had a normal architecture, with a central vein surrounded by hepatocytes and a sinusoidal capillary between them (Fig 1A). Histopathologic liver sections of the second group of mice that received cadmium chloride showed generalized blood vessels congestion including central vein, portal vein and hepatic artery (Fig 1B), diffuse vacuolar degeneration of the hepatocytes (Fig 1C) and also exhibited inflammatory cell infiltration and coagulant necrosis of the hepatocytes (Fig 1D).

While the histopathological assessment of the groups treated with cadmium chloride and parsley oil found that the liver tissue's histopathological structure had improved, as evidenced by the presence of a central vein surrounded by cords of hepatocytes that appeared normal (Fig 1E). The hepatic tissue architecture of the animals treated with parsley oil only appeared normal in histopathological sections (Fig 1F).

Discussion

The current investigation documented substantial histological abnormalities in the group that received treatment with cadmium chloride, which was characterized by the presence of hepatocytes coagulative necrosis and vacular degeneration with congestion, inflammation, and the infiltration of inflammatory cells. Concur with research from other authors who found focal liver necrosis with cadmium exposure (Abdulqader *et al.*, 2022).

It has also been reported that vacuolation occurs in the cytoplasm primarily as a result of disturbances in lipid inclusions and metabolism that occur during pathological disorders. Tissue harmful effects are typically characterized by cell degeneration accompanied by the formation of large vacuoles, lipid accumulation, and tissue necrosis (Alabbasi and Alabdaly, 2022).

According to Hamed *et al.*, (2022) acetophylline buildup in tissues or direct effects on

cells could both contribute to liver necrosis, demonstrated that cadmium produces apoptosis and/or necrosis in cells, *in vivo* and *in vitro*. The results of those studies showed fragmentation of the nucleus (Karyorrhexis) and programmed death modifications (Hamed *et al.*, 2022).

According to several reports, cadmium most likely causes apoptosis by attacking mitochondria. According to one study, cadmium chloride causes mitochondrial loss and cellular organelle depletion along with swelling and the existence of fat droplets that fill the cytoplasmic gaps. Hepatotoxicity plays a role in the mechanism of acute poisoning via two different pathways, one of which is primary harm brought on by direct cadmium infection and the other is a secondary infection brought on by inflammation. The initial damage seems to be caused by Cd²⁺'s interaction with sulfhydryl groups and important components in mitochondria. Oxidative stress, a change in the transmission of mitochondrial permeability, and mitochondrial malfunction are caused by the thiol group becoming inactive (Yazihan *et al.*, 2011).

According to what was previously stated, oxidative stress is thought to be a major factor in the pathological damage that occurs in hepatocytes exposed to cadmium chloride. This is because, according to several studies, cadmium chloride increases the concentration of oxidants like malondialdehyde while decreasing the concentration of glutathione and other SOD. The increase in free radicals produced by the metal cadmium is the cause of this, according to a study conducted by Alabdaly *et al.*, (2021).

These findings demonstrated that parsley oil, which has a high concentration of antioxidants glutathione and other antioxidants, can lessen the damaging consequences that cadmium chloride has on hepatocytes. According to multiple studies, cadmium exposure causes an increase in lipid peroxidation in the livers of rats, which is reduced by parsley oil.

Cells are constantly exposed to oxidative stress from inside or outside the body and the genetic regulator in cells can detect these high levels of oxidative stress and activate levels of enzymes that can reduce reactive oxygen

species and repair the oxidative damage caused by free radicals. For example, SOD can convert oxygen into hydrogen peroxides and water while minimizing damage from oxygen superoxide, this depends on the level of antioxidants inside the body, and parsley oil may be one of them (Almukhtar *et al.*, 2022).

The beneficial outcome of the present study induced by parsley oil should be considered in the protection of the liver from the destructive effects of other chronically used drugs if any, viz., statins, proton-pump inhibitors, and CNS drugs. Moreover, the same positive effects obtained from parsley oil could be provided by other harmless OTC drugs, such as vitamins, or minerals, or herbal remedies or xanthine oxidase inhibitors (allopurinol). Nevertheless, claiming potentially positive agents from these drugs need experimental *in vivo* studies on laboratory animals before their clinical application to humans, if any (Abdulrazzaq *et al.*, 2020).

Conclusion

The present study suggests that parsley oil can counteract the toxic effects of cadmium chloride on hepatocytes, which manifest as necrosis and inflammation, and can lessen the toxic consequences of cadmium chloride from a histological perspective.

Conflict of interests

The authors declare no potential conflict of interests.

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The Effect of Aflatoxin on the Number, Diameter and Distribution of Hydatid Cyst in Mice

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Abstract

We carried out this investigation to demonstrate the impact of aflatoxin contamination of diet and its connection to escalating the severity of hydatid cysts in mice. In order to do this, Swiss mice were employed in this investigation, divided into 3 groups of 8 mice each. The first group served as the control group and received no therapy. A single intraperitoneal injection of about 0.2 ml of the medium containing the

protoscoleces, approximately 2000 units, was given to the second and third groups. The second group had a regular diet that was comparable to the diet of the control animals, whereas the third group received a daily addition of 1 ppm of aflatoxin B toxins to its diet. The procedures ran continuously for 30 days. In contrast to the non-aflatoxin-diet group, the aflatoxin-diet group had a significantly higher number of hydatid cysts, and a significantly high percentage of cysts with a diameter of (0.3 mm) was found in comparison to the non-aflatoxin-diet

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