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The Protective Effect of Omeg-3 Aganist Carbimazole Induced Hepatotoxicity in Rats

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ABSTRACT

Background: The majority of medicines can predispose liver injuries when given in overdoses or with long time such as Carbimazole which is used for treatment of thyrotoxicosis. These adverse effects could be minimized by co-administer an antioxidant agents such as Omega-3 fatty acids which can reduce hepatic cell damages and improve patient outcomes.
Objective: The aim of this study is to evaluate the protective effect of Omega-3 in reducing hepatic damage by chemicals and drugs.
Method: This study was conducted in thirty constitutive days, the animals were divided in equally numbered four groups. Control group, Carbimazole group, Omega-3 group, and Carbimazole and Omega-3 group. After the final treatment, blood was withdrawn from each animal for biochemical tests and then killed and the livers were isolated for histological studies.
Result: The result of our study indicate that Omega-3 possess potential antioxidant effect that protect body organs from oxidative stress injuries. Red blood cells return to normal value hepatic enzyme ALP was reduced significantly in the protective group (p=0.007), other enzymes were not clearly reduced by Omega-3 supplement. Histological study show that Carbimazole group show a great hepatic deterioration such as distortion of liver architecture and necrosis area, while animal group treated with Carbimazole and Omega-3 represent a hepatic tissue with an appearance like a normal tissue.
Conclusion: The findings of this study suggest that the basic mechanisms of the hepatoprotective effects of fish oil involve inhibition of oxidative stress, enhancement of cellular antioxidant defenses, and stabilization of cell membranes.

Keywords- Carbimazole, Hepatotoxicity, Hepatoprotective effect, Liver enzymes, and antioxidants.

I. INTRODUCTION

Carbimazole is a drug used for the treatment of thyrotoxicosis. Carbimazole is considered the main thionamide agents which is used as anti-thyroid drug. It is administered as a prodrug which is converted to methimazole inside the body and concentrated in the gland inhibiting the production of its hormones(1). Carbimazole (C7H10N202S) is present as solid crystalline with a white needle-shaped appearance that are sparingly soluble in water (2). In addition, it is soluble in water in small quantities and has sites that accept hydrogen bonding(3). Carbimazole exerts its action by inhibiting T3 and T4 formation because it acts as a substrate for the enzyme thyroid peroxidase and prevents the iodination of tyrosine in the thyroglobulin. Thyroid peroxidase is predominantly present in the follicles of the thyroid glands which is vital in the iodination and oxidative couple processes that affect thyroid hormones production (4). In addition to its anti-thyroid effect, Carbimazole also has antioxidant activity by acting as a scavenger of the free radicals like hydroxyl radicals, these types of particles are result of oxidation reaction inside the body and can lead to multi-organs injuries (5, 6). Carbimazole therapy is

not free of adverse effects, these are divided into minor such as nausea, bone pain, gastrointestinal disturbances, taste and smell disturbances and major serious adverse effects like agranulocytosis and hepatic injuries in addition to its serious toxic effect on fetuses and newborn thyroid gland during pregnancy or by breast feeding (7). These effects are studied and observed by a scientists and documented in a scientific research (8).

Animal studies stated that Carbimazole therapy is associated with renal system damage such as glomerulonephritis and tubular necrosis. It is estimated that major adverse effects of anti-thyroid agents are age and dose related. These effects could be minimized by co-administration of anti-oxidant agents such as Omega-3 fatty acids.

Omega-3 fatty acids are unsaturated fatty acid that include 3 primary types which are eicosapentoic acid, docosapentaenoic acid, docosahexaenoic acid. In mammals, Omega-3 must be supplied with daily food because it isn't synthesized endogenously(9). The advantages of Omega-3 consumption from their natural source such as fish oil, breads, and eggs were examined and proved for a number of human disease such as cardiovascular disease and tumors(10). After consumption, Omega-3 fatty acids integrated into the cell membrane and induced secretion of anti-inflammatory types of prostaglandin and less effective leukotriene which resulted in reducing inflammation and repair the damaged tissues. This mechanism is the main recorded effect of these fatty acids. Similarly, the cardio protective effect was examined and it is related to the ability to reduce triglyceride level and minimizing atherosclerotic plagues formation resulted in reducing systolic and diastolic blood pressure (11).

Drug induced liver damages account for a considerable number of liver disease and a common cause of about 50% of hepatic disease with a mortality rate of about 10%(12). Large number of scientific researches and projects were performed to testing the hepatotoxicity of a diverse types of medicines (13). The mechanism of liver injuries by medicines were examined to clarify the exact damaged pathways since it is vital complication and should be assisted especially in patient who have liver impairment because it can result in multi-organ damage and the oxidative stress and oxidation reactions are the main cause of hepatocyte injuries (14). It is important to evaluate hepatic toxicity of all drugs because it is complicated challenges and can negatively affect both patients and drugs since a lot of medicines were withdrawn from the market because of its drawback effects on hepatic and renal tissue (15, 16). The main idea of this research is to examine the antioxidant effect of Omega-3 fatty acids supplement using rat model with hepatic toxicity induced by Carbimazole to engage its use since it is available and could prevent serious damage easily.

II. ANIMALS AND STUDY DESIGN

The present research was conducted from October to November 2024 at the College of Pharmacy, University of Kerbala, in accordance with the ethical guidelines established by the institutions Animal Ethics Committee. The experiment was carried out over a period of thirty constitutive days.

24-female albino rats, each weighted approximately 250-300 g were obtained from the animal house of the College of Pharmacy. The animals were housed under standard laboratory conditions with a 12 hours light \dark cycles and were provided a standard diet with an free access to water and food to ensure uniform growth and physiological performance. The animals were randomly assigned to four groups (n=6 per group). Group I served as the control and received only standard food. Group II received Carbimazole at a dose of 6 mg/kg via oral gavage. Group III was administered an Omega-3 supplement at a dose of 1200 mg/kg orally. Group IV received both Carbimazole and Omega-3 orally with a dose of 6mg/kg and 1200mg/kg respectively, once a daily. 24 hours following the final treatment, the animals were anesthetized using chloroform. Blood samples (5 ml) were collected via cardiac puncture. The samples were centrifuged at 3000 rpm for ten minutes, which was subsequently analyzed for biochemical markers, including Aspartate-aminotransferase (AST), Alanine-aminotransferase (ALT), total bilirubin, and direct bilirubin using an automated analyzer (Fujifilm Corporations, Japan). Following blood collection, the animals were euthanized by cervical dislocation. Hepatic tissues were excised and fixed in formalin for histopathological processing and microscopic examination.

Materials

Carbimazole tablets (NeoMercazole® , AMIDAPHARMA, Ireland. Omega-3 soft capsile (MeraOmega3®, Mera Pharma GmbH, Switzerland), Normal saline (pioneer, Iraq). all drugs were purchased from commercial pharmacy.

Ethical Approval

The research design and methodology was agree with the requirements of the Scientific and Ethics Committee of the Pharmacy College/ University of Kerbala with a special research code (2024An. 16) that was APPROVED on July 18, 2024.

Statistical study

The collected data of this study were entered from the data analysis sheets and analyzed statistically by package for the social science (SPSS version 26). The data were expressed as frequencies and percentages in appropriate tables. Pairwise values were obtain to compare between groups of animals. Statistical association was considered significant when p value equal or less than 0.05 (p value ≤ 0.05).

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III. RESULT

The results of the tested biomarkers were analyzed statistically to obtain the minimum, maximum, mean, and stander deviation values. All these information are explained in table 1 below.

Table1: Statistical characteristics of the tested biochemical parameter

Biomarker	Mean± SD	Reference	High/low
WBCX10^9/L	13.300±2.599	14.1667	Low
RBCX10^{12}/L	6.945±0.588	7.2733	Low
Hb.g/dl	12.930±1.097	13.650	Low
HCT%	38.496±3.338	40.983	Low
T.bill.mg/dl	0.1±0.250	0.0600	High
D-bill.mg/dl	0.0926±0.244	0.0717	High
ALTU/L	51.765±9.727	58.817	Low
ASTU/L	159.348±28.881	187.550	Low
ALPU/L	505.543±228.900	586.150	Low
AST/ALT	3.157±0.717	3.200	Low

WBC: White blood cells, RBC: Red blood cells, Hb: Hemoglobin, HCT: Hematocrit, T-Bill: Total bilirubin, D-Bill: Direct bilirubin, ALT: Alanine transaminase, AST: Aspartate transaminase, ALP: Alkaline phosphatase.

The examined markers were tested statistically to test the significant of each parameter. Also pairwise comparison was done to compare the data of different groups under study. All these information are shown in table 2 below.

Table 2: Comparison of biomarkers results between animal groups

Parameter	Groups				Pairwise comparison	p-value (p≤0.05)
WBCX10^9/L	Control N=6	Carb. N=6	Omega3 N=5	Carb.+Omega3 N= 6		
RBCX10^12/L	14.1667	13.8467	11.512	13.38		0.366
Hb.g/dl	7.2733	6.9133	7.162	6.4700	G1< G4 G3< G4	0.017* 0.045*
HCT%	13.650	12.917	13.16	12.033	G1< G4	0.01*
T.Bili.mg/dl	40.983	38.650	39.04	35.400	G1< G4 G3< G4	0.003* 0.045*
D.Bili.mg/dl	0.0600	0.0783	0.2700	0.02		0.398
ALTU/L	0.0717	0.0433	0.268	0.0167		0.345
ASTU/L	58.817	54.783	50.300	42.917	G1< G4 G2< G4	0.003* 0.02*
ALPU/L	187.55	145.933	146.00	155.683	G1>G2 G1>G3	0.029*
AST/ALT	586.15	292.6	506.84	636.800	G1< G2 G4< G2	0.018* 0.0078
WBCX10^9/L	3.200	2.733	2.92	3.733	G4< G2	0.015*

WBC: White blood cells, RBC: Red blood cells, Hb: Hemoglobin, HCT: Hematocrit, T-Bili: Total bilirubin, D-Bili: Direct bilirubin, ALT: Alanine transaminase, AST: Aspartate transaminase, ALP: Alkaline phosphatase.

Histological processing was also carried out to the hepatic tissue after animal killing to examine the morphological changes of liver tissue and the results are explained in the below figures

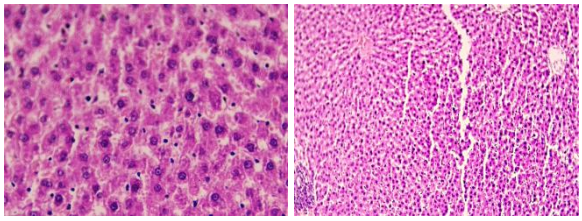

Fig.1: A photomicrograph of liver section of control group with normal hepatocyte and portal veins

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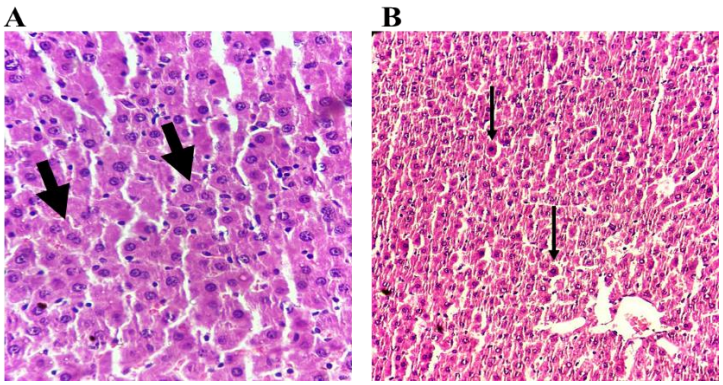

Fig. 2: A photomicrograph of liver sections of CBZ treated group stained with hematoxylin–eosin. A) Distortion of the liver architecture and swollen hepatocytes with severe vacuolated cytoplasm (thick arrow). B) Pyknotic nuclei (thin arrow), and mononuclear leukocytic infiltration (IF) were also seen. Scale bare = 50 µm.

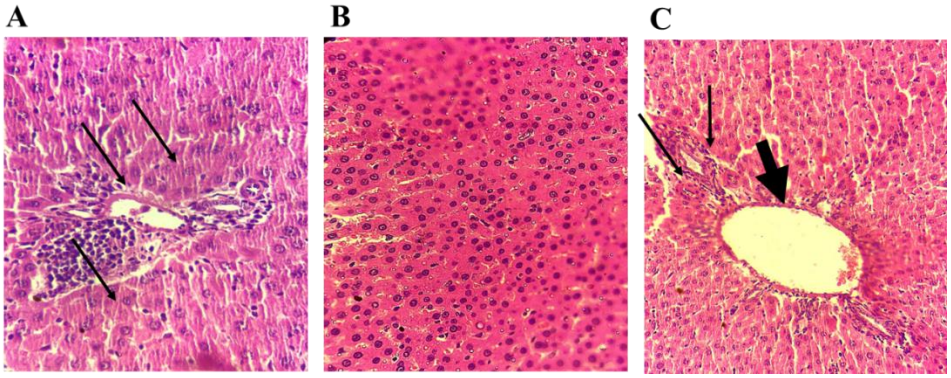

Fig. 3: A photomicrograph of liver sections of CBZ group treated with omega3 stained with hematoxylin–eosin. A) Normal CV and blood sinusoid lined by Kupffer cells (thin arrows). B) Demonstrating the nearly normal structure of hepatocytes with vesicular nuclei . Scale bare = 50 µm. C) A photomicrograph of a section of the liver of rat treated with Omega3 showing normal hepatocytes structures with clear CV (Thick arrow) and blood sinusoid lined with Kupffer cells (Thin arrows). Scale bare = 50 µm.

IV. DISCUSSION

The liver is considered one of the most vital organs in the human body due to its central role in metabolizing variance substances primarily owing to the abundance of metabolic enzymes(17). Drugs and xenobiotic enter hepatic tissue directly from the gastrointestinal tract at high concentration which makes the liver particularly susceptible to injury by active metabolites (18). Acute or chronic liver injuries may result from reactive species generated during metabolic reactions such as free radicals and unstable chemical metabolites. These species can interact with a macromolecules, causing lipid peroxidation, protein dysfunction, and damage to the DNA genetic materials(19). Commenly used serum biomarkers for diagnosis hepatic injuries include alanine aminotransferase (ALT),aspartate aminotransferase (AST), gamma-glutamyl transferease (GGT), alkaline phosphatase (ALP), and total bilirubin. In addition, histopathological evaluation is a crucial diagnostic tool as various changes could be observed and assessed including inflammation, necrosis, regeneration could be seen to determine the extent of liver toxicity (20).

Certain drugs such as Carbimazole, when used over prolonged periods, have been associated with toxicity to vital organs such as liver or kidneys. These toxic effects could be evaluated by assessing biomarkers such as hepatic enzymes (AST, ALT,..), bilirubin levels, and hematological parameters. Toxicities and adverse effect could be minimized and prevented through administration of protective agents such as vitamins, antioxidants and free radical scavengers,. These agents function by reducing oxidative stress, scavenging, or modulating alternative protective pathway. Omega-3 fatty acids have been investigated in numerous studies and recognized for their antioxidant effect (21, 22). It is widely available, cost-effective and palatable across different age groups. In the present study, the protective effect of Omega-3 was evaluated against Carbimazole toxicity by using rat model.

Table 1 represent the mean -/+ stander deviation values of each tested parameter to illustrate the differences between experimental groups and control. Table 3 details the data for each group and compared it with others. firstly, white blood cells in the control group is greater than Carbimazole group and this may be related to Carbimazole toxicity cause

inhibition to the immune cells and reduce the number of immune cell generation and development. Although, it was anticipated that WBC in Carbimazole group is greater than control group because it is given to the animal in toxic dose, this observation was not align with other previous studies reporting Carbimazole overdose usually resulted in agranulocytosis (23).In the Carbimazole and Omega-3 group WBC level didn't differ significantly from those in the Carbimazole group indicating that Omega-3 not have significant effect in this dose on inflammatory cells. These findings are consistent with a study conducted in India by John et al. (2021) which demonstrated that long-term used of carbimazole (month or longer) may lead to neutropenia and general reduction in white blood cells , P- value between groups was greater than 0.05 which mean no significant differences in WBC counting between different study groups(24).The values of red blood cells and hemoglobin was also examined in the four study groups (Table 2). Values in the control and Omega 3 groups were significantly higher than in Carbimazole and Carbimazole with Omega-3 groups ($p=0.017$, $p=0.045$ respectively). This reduction may be attributed to the toxic effect of Carbimazole since it cause renal deterioration and reduce erythropoietin synthesis which is the stimulus hormone for RBC synthesis secreted by the kidney cells(25).Contrary to expectation, Omega-3 supplementation did not improve RBC, Hb, and HCT parameter.

This may be related to mechanism of Omega-3 effect since it may require longer time to exert its protective effects and facilitate renal tissue damage. Total and direct bilirubin values did not differ significantly across all groups. bilirubin metabolism occur in the liver where it is conjugated with glucuronic acid producing inactive metabolites that are excreted into the bile duct. The conjugation process may not substantially affected by Carbimazole toxicity since it depends on the storage glucuronic acid. However, a reduction in in bilirubin level in (carbimazole + omega-3 group)compared to Carbimazole group suggest a potential protective effect of omega-3 in reducing hepatotoxicity. Hepatic enzymes such as ALT, AST, and ALP levels are commonly elevated in response to hepatotoxic agents which cause oxidative stress and irreversibly inhibition of peroxidases involved in reactive oxygen species (ROS) scavenging(26).

The liver , due to its high metabolic activity generates high levels of (ROS) contributing to oxidative stress. In most cases, elevated enzymes levels indicate hepatocyte degeneration, necrosis, and cellular lysis, releasing intracellular enzymes into the blood stream. The findings of this study show that Carbimazole toxicity cause slightly reduction in levels of hepatic enzymes than control group, which may reflect inhibition of specific metabolic pathways or insufficient time for damage to manifest fully. These observations contrast with previous study from Kerbala university of Kerbala, since they found that induction hepatic toxicity produce elevated level of enzymes(25). Notably, ALT and AST levels in Carbimazole and Omega-3 group is lower than that of Carbimazole (toxicity) group indicating the protective effect of Omega-3. While ALP increased significantly (p value= 0.018) this may related to inhibition of specific intracellular pathway or Omga-3 have lower protective effect related to this enzyme.

The general environment of the experiment may affect the result of the study, for example, stress, type and amount of food since starvation can cause stress leading to inaccurate results.

Histological study was performed to confirm the biochemical findings and evaluate the morphological effect of the toxin and the protective agent on the liver tissue. Liver section of Carbimazole-treated group showed sever tissue damage including extensive hepatic architecture distortion, cytoplasmic vacuolation of hepatocyte, infiltration of leucocytes, inflammatory cells was also found. Moreover, pyknotic nuclei, and dilated congested sinusoids were also detected. Figure1 A-B

In contrast, the liver sections of animals receiving Carbimazole and omega3 exhibited near-normal hepatic structure including vesicular nuclei, intact central veins and Kupffer cells-lined sinusoids. Figure2 A-B

Rats treated with Omega3 alone showed normal hepatic morphology without evidence of toxicity of distortion, clear hepatocytes, blood sinusoids lined with proliferating Kupffer cells (Figure2 C). Several studies have reported the antioxidant and anti-inflammatory properties of omega-3(27) . Omega-3 polusaturated fatty acids (Omega-3 PUFAs) exert beneficial effect by suppressing inflammatory response and oxidative stress as well as modulating cell proliferation(28). The mechanisms underlying the hepatoprotective effects of Omega-3 PUFAs includes its ability to increase GSH along with its capability to scavenge free radicals and consequently inhibit lipid peroxidation(29). Meanwhile; Maksymchuk, 2014, previously demonstrated that there was more than two-fold increase in the content of cytochrome P450 2El (CYP2E1) in the liver of rats receiving Omega-3 PUFAs for 4 weeks in the standard daily diet. Similarly, Calder (2010) reported a threefold increase in CYP2E1 levels following dietary Omega-3 (30).

V. CONCLUSION

The observations of the study suggest that the basic mechanisms of the hepatoprotective effects of Omega3 and its components involve inhibition of oxidative stress, enhancement of cellular antioxidant defenses, and stabilization of cell membranes, suggesting that fish oil may be a dietary supplement for hepatoprotection and antioxidants, especially in patients undergoing anti-thyroid therapy who are at risk for hepatotoxicity.

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Conflict of interest:

The authors declare no conflict of interest.

Source of supply

Personally funding study

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