

Original Research Article

Effect of Basrah bee honey and n-acetylcysteine against citalopram hepatotoxicity

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Abstract

Purpose: To investigate the effects of Basra bee honey and N-acetylcysteine as monotherapies and in combination against citalopram hepatotoxicity.

Methods: A total of 30 healthy female albino Wistar rats were equally and randomly divided into five groups (n = 6). Group 1 received distilled water (control group), group 2 received citalopram (10 mg/kg/day), group 3 received Basra bee honey (150 mg/kg/day) and citalopram (10 mg/kg/day), group 4 received N-acetylcysteine (200 mg/kg/day) and citalopram (10 mg/kg/day), group 5 received Basra bee honey (150 mg/kg/day), N-acetylcysteine (200 mg/kg/day) and citalopram (10 mg/kg/day) via oral route. After 30 days, the rats were sacrificed under diethyl ether anaesthesia. Liver tissue homogenate and serum were taken, and tumour necrosis factor alpha (TNF- α), interleukin-1 beta (IL-1 β), interleukin-10 (IL-10), and glutathione (GSH) and malondialdehyde (MDA) were determined by enzyme-linked immunosorbent assay (ELISA).

Results: Group 2 demonstrated significantly higher MDA, TNF- α , and IL-1 β , and lower GSH and IL-10 compared to control ($p < 0.05$). Furthermore, group 5 showed significantly lower MDA, serum and tissue TNF- α , and IL-1 β ($p < 0.05$) compared to control. Furthermore, GSH, serum and tissue IL-10 were significantly higher in group 5 compared to control ($p < 0.05$).

Conclusion: Combination of Basra bee honey and N-acetylcysteine significantly reverses citalopram-induced hepatotoxicity in rats.

Keywords: Basra bee honey, Citalopram, Inflammatory cytokines, N-acetylcysteine, Oxidative stress

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INTRODUCTION

Citalopram, a selective serotonin reuptake inhibitor (SSRI), is a major class of antidepressants used in the treatment of major anxiety disorders, depressive disorders, obsessive-compulsive disorder and social phobia [1]. Also, citalopram is effective in treating alcohol dependence, premenstrual dysphoria and behavioral disturbances of dementia [2]. Citalopram has also been used as an off-label

medication for treating chronic neuropathic pain [3]. Toxicity of SSRIs like citalopram is associated with adverse effects such as drowsiness, nausea, dyspepsia, increased sweating, headache, and sexual dysfunction. Severe adverse events include suicidal ideation and behavior, serotonin syndrome, prolongation of the QTc interval, acute glaucoma and acute mania [4]. Citalopram-induced liver toxicity mainly occurs through mechanisms involving oxidative stress and inflammatory responses [5].

Treatment with citalopram increases pro-inflammatory cytokines, particularly tumour necrosis factor- α (TNF- α), which play a major role in the development of liver injury [6]. Also, hepatotoxicity is associated with impaired antioxidant defense systems, such as reduced levels of glutathione (GSH). These alterations reflect oxidative damage to hepatic tissue and disruption of redox homeostasis [7]. Basra honey is primarily made up of water and natural sugars, supplemented with various bioactive compounds, minerals, and vitamins contributing to its distinct flavour and health benefits. The exact composition varies based on the specific floral from which it was obtained, such as Ziziphus (Sidr), Medicago (Barsim), and Eucalyptus (Calbtose) [8]. Hepatoprotective effect of honey is directly related to the phenolic content and antioxidant activity [9]. Bee honey is effective in managing liver toxicity triggered by chlorpyrifos [10]. Also, honey contains vital compounds capable of ameliorating oxidative stress damage [11]. N-acetylcysteine (NAC) is a glutathione (GSH) precursor that elevates GSH levels within cells with excellent antioxidant and anti-inflammatory activity [12]. A previous study showed that NAC is effective against carbon tetrachloride and trichloroethylene-induced hepatotoxicity [13]. Prolonged citalopram therapy for depression has been associated with hepatic injury. Therefore, this study investigated the protective effects of Basra honey and NAC on citalopram-induced liver toxicity by examining levels of inflammatory cytokines and oxidative stress markers in female albino Wistar rats.

METHODS

Preparation of samples

Basra bee honey was obtained from local apiaries in Basra province, Iraq. The honey was freshly collected, filtered to remove impurities, and stored at room temperature in a dark container. Citalopram (Accord, UK), N-acetylcysteine capsules (AMS, USA), and Basra bee honey were completely dissolved in distilled water (Pioneer, Iraq) before administration. Additive substances that were used in this study include formaldehyde solution (S.D.F.C. Limited, India) and normal saline (Pioneer, Iraq).

Animals

A total of 30 healthy female rats (180 – 250 g) from the Veterinary Medicine College at Basra University were randomly and equally assigned into 5 groups. The animals were kept in polypropylene cages in the animal house of the Department of Pharmacology under standard

conditions (21 ± 4 °C, 12 h light / dark cycle) to acclimatize for 2 weeks. During this time, the animals were given unrestricted access to nutrition-balanced diet and fresh water. Ethical approval was obtained from the Ethical Committee of Basra College of Pharmacy and complied with the internationally accepted guide for the care and use of laboratory animals [14].

Design

The animals were randomly and equally assigned to five groups ($n = 6$). Group 1 received 1 mL distilled water, group 2 (positive control) received citalopram (10 mg/kg/day) [15], group 3 received Basra bee honey (150 mg/kg/day), followed by citalopram (10 mg/kg/day) [16], group 4 received N-acetylcysteine (200 mg/kg/day), followed by citalopram (10 mg/kg/day) [17], and group 5 received Basra bee honey (150 mg/kg/day), N-acetylcysteine (200 mg/kg/day), and citalopram (10 mg/kg/day) orally for 30 days with dosing interval of 60 min. The rats were not fed for 24 h after administration.

Evaluation of parameters/indices

Serum tumor necrosis factor- α (TNF- α), Interleukin-1 β (IL-1 β), Interleukin-10 (IL-10)

Venous blood (5 mL) was obtained through cardiac puncture and left to stand at room temperature for 30 min. The blood was centrifuged (Genex, FL, USA) for 15 min and serum TNF- α , IL-1 β and IL-10 were measured.

Tissue homogenate

Tissue levels of malondialdehyde (MDA), reduced glutathione (GSH), TNF- α , IL-1 β and IL-10 were measured using enzyme-linked immunosorbent assay (ELISA) kits following manufacturer's instructions.

Statistical analysis

Data were analysed using Statistical Packages of Social Sciences (SPSS, version 25.0; SPSS Inc., Chicago, Illinois, USA). Continuous data were presented as mean $\pm$ standard error of mean (SEM), and compared using one-way analysis of variance (ANOVA). $P < 0.05$ was considered statistically significant.

RESULTS

Levels of MDA

Group 2 showed significantly higher MDA compared to group 1 ($p < 0.05$). Also, groups 3

to 5 showed significant reduction in MDA levels ($p < 0.05$). Furthermore, group 4 showed significantly higher MDA levels compared to group 2 ($p < 0.05$). Among the groups, MDA level was lowest in group 5 ($p < 0.05$; Figure 1).

Levels of glutathione (GSH)

Group 2 showed significantly lower GSH levels compared to group 1 ($p < 0.05$). Also, group 5 showed significantly higher GSH levels compared to group 2 ($p < 0.05$). Among the groups, GSH level was significantly higher in group 5 ($p < 0.05$; Figure 2).

Serum and tissue TNF- α

Serum (Figure 3 A) and tissue TNF- α (Figure 3 B) were significantly higher in group 2 compared to group 1 ($p < 0.05$). Furthermore, serum and

tissue levels of TNF- α were significantly lower in groups 3, 4, and 5 compared to group 2 ($p < 0.05$).

Serum and tissue IL-1 β

Group 2 showed significantly higher serum and tissue IL-1 β levels compared to group 1 ($p < 0.05$). Furthermore, groups 3, 4, and 5 showed significantly lower serum and tissue IL-1 β levels compared to group 2 ($p < 0.05$; Figure 4 A, B).

Serum and tissue IL-10

Group 2 showed significantly lower serum and tissue IL-10 levels compared to group 1 ($p < 0.05$). Groups 3, 4, and 5 showed significantly higher serum and tissue levels of IL-10 compared to group 2 ($p < 0.05$; Figure 5).

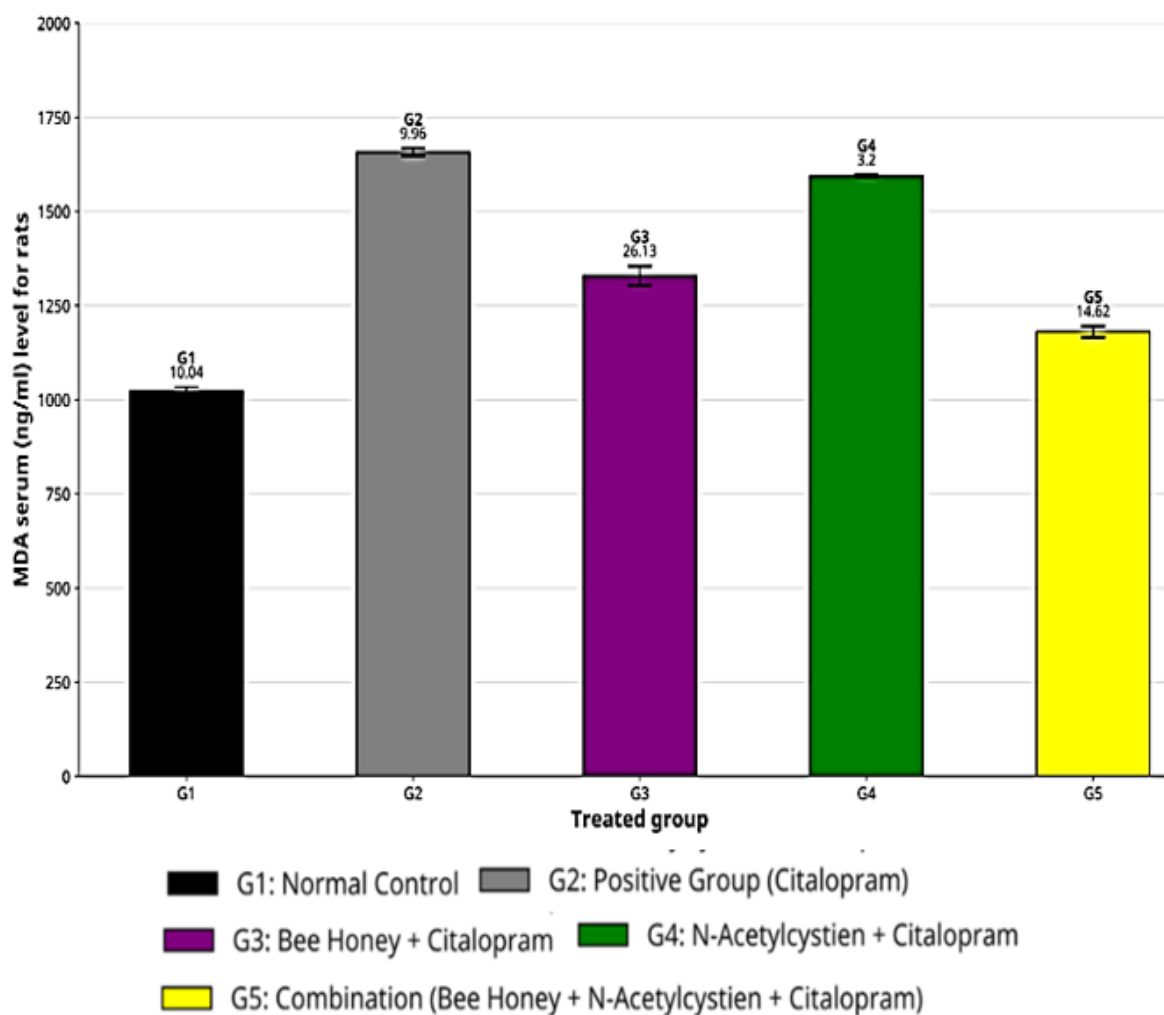


Figure 1: Serum MDA concentrations. Group 1 (control group), group 2 (10 mg/kg/day citalopram), group 3 (150 mg/kg/day Basra bee honey + 10 mg/kg/day citalopram), group 4 (200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram), group 5 (150 mg/kg/day Basra bee honey + 200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram)

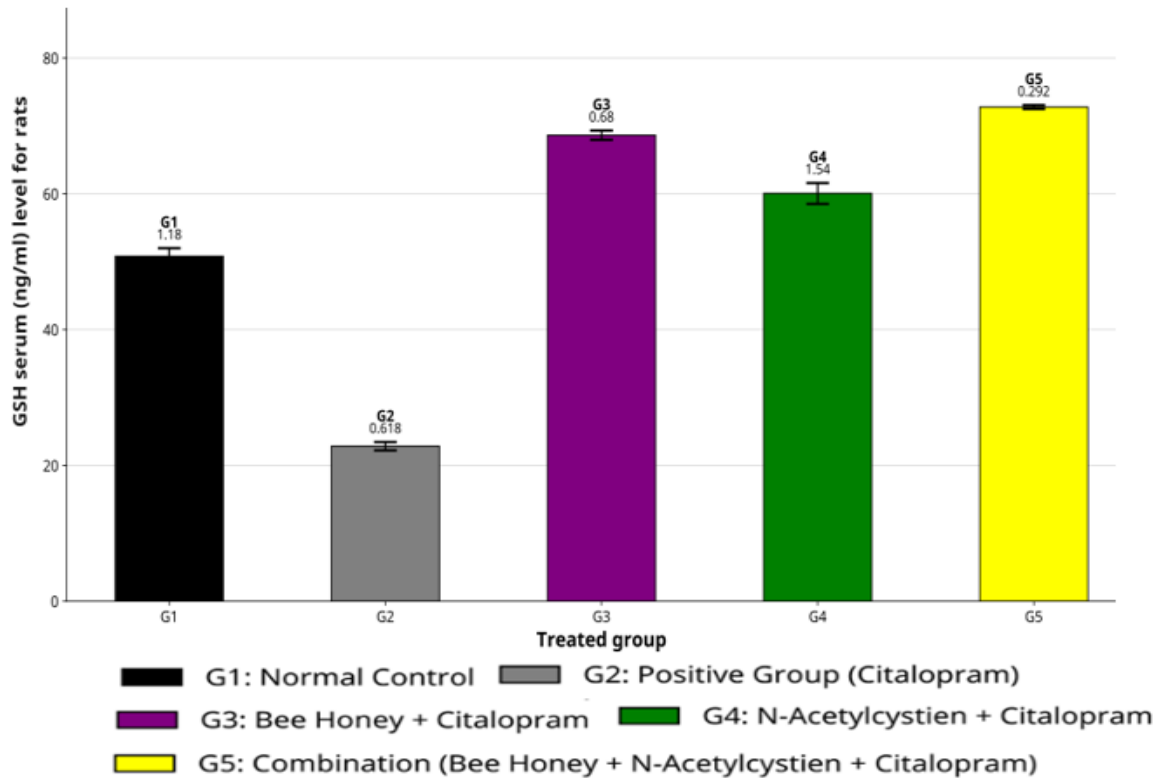


Figure 2: Serum GSH level. Group 1 (control group), group 2 (10 mg/kg/day citalopram), group 3 (150 mg/kg/day Basra bee honey + 10 mg/kg/day citalopram), group 4 (200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram), group 5 (150 mg/kg/day Basra bee honey + 200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram)

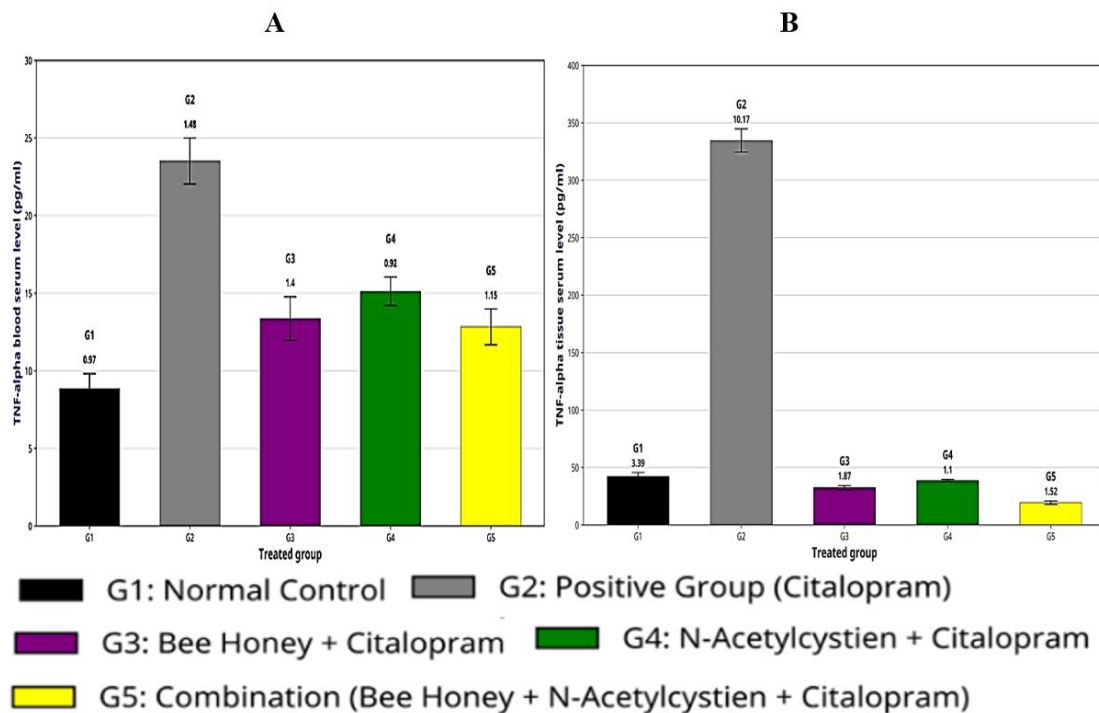


Figure 3: (A) Serum TNF-α level; (B) Tissue TNF-α level. Group 1 (control group), group 2 (10 mg/kg/day citalopram), group 3 (150 mg/kg/day Basra bee honey + 10 mg/kg/day citalopram), group 4 (200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram), group 5 (150 mg/kg/day Basra bee honey + 200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram)

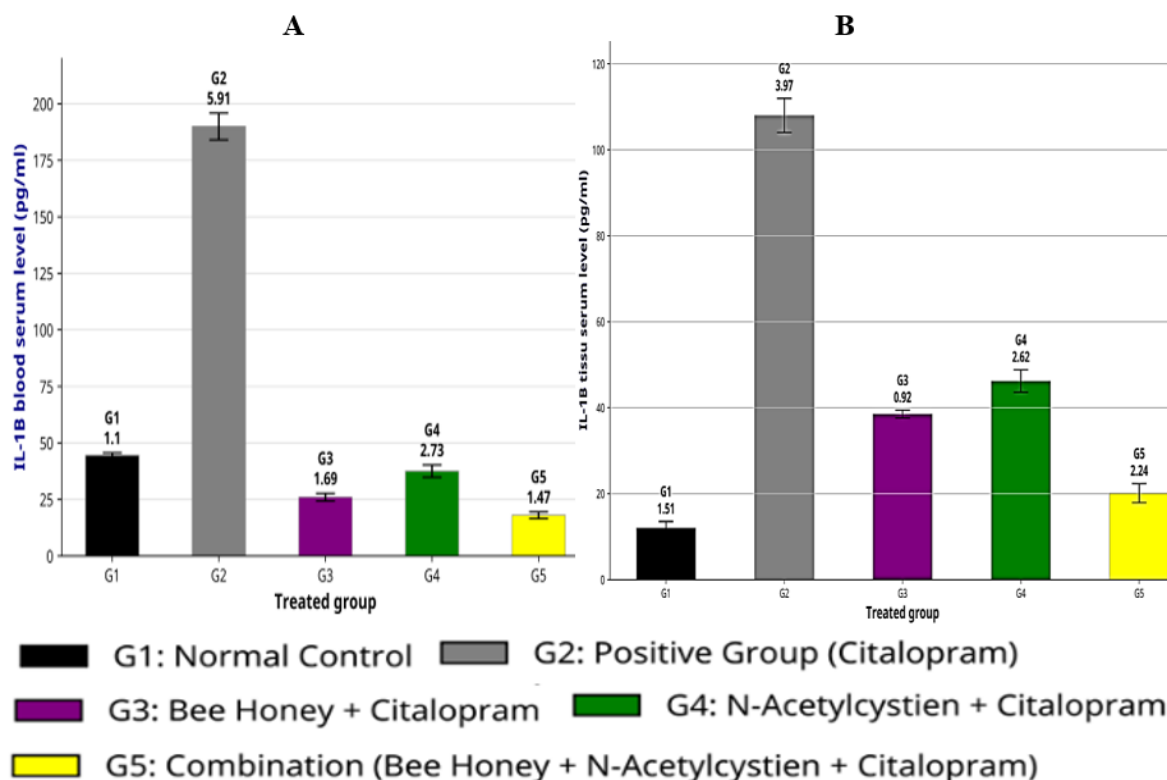


Figure 4: (A) Serum IL-1B levels; **(B)** Tissue IL-1B levels. Group 1 (control group), group 2 (10 mg/kg/day citalopram), group 3 (150 mg/kg/day Basra bee honey + 10 mg/kg/day citalopram), group 4 (200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram), group 5 (150 mg/kg/day Basra bee honey + 200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram)

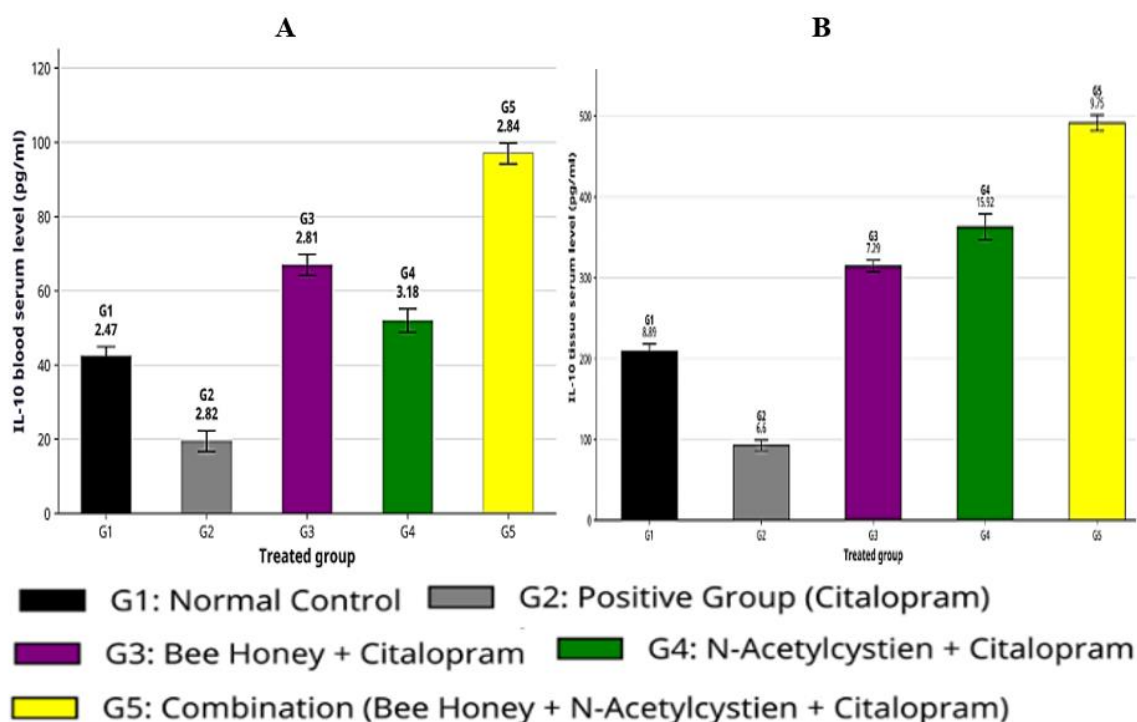


Figure 5: (A) Serum IL-10 levels; **(B)** Tissue IL-10 levels. Tissue IL-1B levels. Group 1 (control group), group 2 (10 mg/kg/day citalopram), group 3 (150 mg/kg/day Basra bee honey + 10 mg/kg/day citalopram), group 4 (200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram), group 5 (150 mg/kg/day Basra bee honey + 200 mg/kg/day N-acetylcysteine + 10 mg/kg/day citalopram)

DISCUSSION

Citalopram, a selective serotonin reuptake inhibitor (SSRI), is mostly used for anxiety disorders and depression with potential hepatotoxicity following prolonged use [15]. Serum MDA and GSH, which are important markers of liver injury, were measured in all groups. Serum MDA indicates the accumulation of lipid peroxidation in the liver, while decreased GSH levels indicate impaired or reduced reactive oxidative species as well as increased oxidative stress [18]. Administration of citalopram (10 mg/kg per oral) significantly increased MDA compared to control, indicating the presence of lipid peroxidation and reactive oxygen species, which must have been generated during hepatic metabolism [5]. An increase in MDA results in lipid peroxidation, acting as a major mechanism in citalopram-induced liver toxicity [5]. Furthermore, group 2 also showed significant reduction in hepatic GSH levels compared to control group, indicating a deteriorating redox balance.

A previous study has shown that citalopram diminishes endogenous antioxidants and aggravates liver damage [5], which is in tandem with this current study. These findings prove that the observed GSH depletion serves as a sensitive marker of oxidative damage, impaired antioxidant defense mechanisms, and elevated lipid peroxidation [1]. These findings align with previous studies, which revealed that antidepressant-induced hepatotoxicity is primarily driven by oxidative stress and mitochondrial dysfunction [5]. This redox imbalance is further characterized by a significant increase in proinflammatory cytokines, specifically TNF- α and IL-1 β , in both serum and liver tissues, acting via paracrine signaling to drive hepatocyte necrosis and local immune cell recruitment [19]. On the other hand, IL-10 directly inhibits transcription and secretion of key proinflammatory cytokines such as TNF- α and IL-1 β by macrophages and dendritic cells [20]. Therefore, evaluating IL-10 dynamics is essential for understanding the resolution phase of inflammation and the delicate balance between necessary immune activation and tissue preservation [20]. Tumor necrosis factor- α (TNF- α) is a key mediator in the development of liver injury, often triggering further ROS production and apoptosis in hepatocytes [19]. An increase in IL-1 β suggests the activation of the inflammasome pathway during drug-induced stress [20]. Similarly, citalopram-induced toxicity was associated with enhanced local inflammation, characterized by higher TNF- α and IL-1 β concentrations in liver tissues compared to

serum levels. Systemic and tissue levels of IL-10 were significantly lower in group 2 compared to group 1. Depletion of IL-10 is a reflection of the failure of compensatory mechanisms intended to minimize hepatic inflammation, which is consistent with findings from previous study [21].

Bee honey possesses potent antioxidant and hepatoprotective properties due to its rich content of flavonoids (quercetin and chrysin) and phenolic acids. These compounds block the activity of NF- κ B, a protein complex that triggers inflammation [22], leading to higher GSH levels. This protects the cell membrane, keeping the liver tissues contra-oxidative to injury, scavenging free radicals and improving endogenous antioxidant defenses [8]. N-acetylcysteine (NAC) is a well-established antioxidant and a precursor to GSH. It is an antioxidant, anti-inflammatory, and vasodilator, enhancing tissue perfusion and reducing mitochondrial dysfunction. As a result, it is frequently used to inhibit toxin-induced hepatic injury [23]. Findings from this study revealed that groups 3, 4, and 5 demonstrated significant reductions in serum MDA levels compared to group 2, suggesting a potential protection against citalopram induced hepatotoxicity consistent with earlier findings [10]. Also, in citalopram induced rats, NAC supplementation effectively reduces hepatic MDA, consistent with previous study [13]. Synergistic effect was observed when Basra bee honey was combined with NAC, resulting in the most significant reduction in MDA levels in group 5 compared to group 2. This current study also revealed that the group treated with Bee honey following citalopram induction had elevated GSH levels compared to citalopram alone. Phenolic compounds in bee honey improve antioxidant defenses by increasing GSH content and restoring redox balance [9]. Furthermore, in rats exposed to cadmium, administration of NAC significantly raises hepatic GSH [24]. Based on the results of this study, NAC therapy also restored GSH levels, as demonstrated. However, in groups 3 and 4, the treatment effectively enhanced IL-10 levels. This suggests that Basra honey showed a potent immunomodulatory effect, shifting the cytokine balance toward a protective, anti-inflammatory state [16]. While NAC primarily acts as a GSH precursor to reduce triggers of inflammation, the significant elevation of IL-10 in group 4 indicated a more direct influence on the immune response.

The triple regimen of bee honey, NAC, and citalopram (G5) likely increased MDA and reduced GSH levels compared to double regimens (as in groups 3 and 4) due to reduced lipid peroxidation, less consumption of GSH from

elevated antioxidant turnover and enhanced detoxification. This rapid elevation of MDA suggests that the combination therapy was a more effective antioxidant, stimulating GSH-dependent repair and conjugation reactions to manage oxidative stress. In group 5, reduced levels of TNF- α and IL-1 β were observed compared to double regimens (as seen in groups 3 and 4) due to lower cellular activation of inflammatory signals, recall of immune cells, and enhanced detoxification. These results, encompassing oxidative and inflammatory markers, confirmed the potent protective efficacy of Basra bee honey and NAC against citalopram-induced hepatotoxicity through the dual regulation of antioxidant defenses and cytokine signaling.

CONCLUSION

Basra bee honey and NAC effectively prevented citalopram-induced hepatotoxicity, normalized oxidative stress markers, and reduced systemic (serum) and local (liver tissue) inflammatory cytokines. Also, the combination of Basra bee honey and NAC improved antioxidant defense system and decreased the harmful effects of citalopram on systemic and hepatic inflammatory responses. As a single regimen, Basra bee honey showed stronger defense against citalopram-induced oxidative damage and cytokine elevation.

DECLARATIONS

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Ethical approval

It is stated in the method.

Use of Artificial Intelligence/Large language models

The authors confirm that AI-based tools were used solely for language editing and manuscript polishing purposes (e.g., grammar, phrasing, and clarity). No AI tools were used to generate content, interpret data, or influence study design, results, or conclusions. All intellectual contributions, study analyses, and interpretations

reported in the manuscript are the original work of the authors.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

No conflict of interest is associated with this work.

Contribution of authors

We declare that this work was done by the author(s) named in this article, and all liabilities pertaining to claims relating to the content of this article will be borne by the authors. Hassan Ali Lafta developed the concept and proposal, and prepared the manuscript. Asst. Prof. Dr. Manal Abdul Khaliad conducted the literature search and organized the data obtained. Asst. Prof. Dr. Manal. N. Alhayder edited, reviewed the manuscript and carried out statistical analysis.

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