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Original article

Protective effect of curcumin on chloroform as by-product of water chlorination induced cardiotoxicity

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ABSTRACT

Chloroform ($CHCl_3$) is one of the volatile organic compounds detected most frequently in both ground and surface water. This study aimed to evaluate the efficacy of curcumin (CMN) to attenuate $CHCl_3$ toxicity and cellular dysfunction in cardiac tissue of female albino rats. Fifty rats were divided into 5 groups, 1st group was control; 2nd group rats were intoxicated with 150 mg $CHCl_3$/kg BW; 3rd group rats were treated with 50 mg CMN/kg BW; 4th group rats were treated with 50 mg CMN/kg BW for 30 days then intoxicated with 150 mg $CHCl_3$/kg BW for 60 days and 5th group rats were intoxicated with 150 mg $CHCl_3$/kg BW plus 50 mg CMN/kg BW, respectively. Treatment was continued for 90 days. The levels of lipid peroxidation, myeloperoxidase (MPO) and xanthine oxidase (XO) were increased and the activities of antioxidant enzymes, protein content and endogenous antioxidants were decreased in cardiac tissues in rats treated with $CHCl_3$ in comparison with control group. Serum cholesterol, triglycerides and LDL-C levels were increased while high HDL-C was decreased in rats treated with $CHCl_3$ in comparison with control group. Treatment with CMN helps in improving the adverse effect of $CHCl_3$ toxicity; also our histological results confirm this finding. The present study could be concluded that CMN has protective and ameliorative effects against $CHCl_3$ induced oxidative stress.

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1. Introduction

Chloroform ($CHCl_3$) is the most prevalent by-product of water disinfection with chlorine-based chemicals [1] and is also formed in large quantities as a by-product of chlorination of cooling water in power plant and in the process of bleaching paper [2]. Therefore, a large part of the human population may be chronically exposed to chloroform from different sources. Also, drinking water has been considered the main one. This aliphatic organic compound is representative of a large group of organic compound having a widespread use in industry as dispersant, solubilizer and diluent. It is one of the top 20 priority environmental toxicant [3] and is classified as a group 2B carcinogen [4]. The acute toxicity demonstrated by $CHCl_3$ is due to its biotransformation to nucleophilic bi-functional metabolite phosgene that reacts with glutathione to fom diglutathionyl dithiocarbonate or is directly metabolized to carbon dioxide and chloride free radicals [5]. Cytochrome P450-2E1 is the enzyme that catalyses the conversion process. Phosgene, formed in the biotransformation process of $CHCl_3$, bind covalently to tissue macromolecules resulting in impairment of cellular vital functions. $CHCl_3$ like most other liver damaging agents, such as acetaminophen and alcohol or viral hepatitis release reactive oxygen species (ROS) at the site of injury [6]. The acute toxic effects of $CHCl_3$ in animals are similar to those observed in humans and the main target organs are the liver, kidney and the central nervous system [7].

$CHCl_3$ toxicity is basically mediated by free radical; therefore a free radical scavenger such as curcumin has been in use in $CHCl_3$ insults. Curcumin, an important constituent of turmeric (*Curcuma longa L.*), has been widely used for centuries as an indigenous medicine [8]. Curcumin exhibits a wide range of pharmacological effects such as antioxidant, antitumor, anti-inflammatory and hepatoprotective effects of curcumin against 1,2-dimethylhydrazine-induced colon cancer and alcohol as well as carbon tetrachloride induced hepatotoxicity [9]. The assay of lipid peroxidation and antioxidant enzymes in cardiac tissue of chloroform treated animals has evolved as a reliable method for screening protective effect of curcumin during chloroform induced toxicity in female rats.

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2. Materials and methods

2.1. Chemicals

Chloroform ($CHCl_3$), curcumin (*Curcuma longa L.*) and other fine chemicals were obtained from sigma chemical company St Louis, U.S.A. All other chemicals and reagents used were of analytical grade. The doses of chloroform ($CHCl_3$) and curcumin were 150 mg/kg BW [10] and 50 mg/kg BW [11].

2.2. Animals

Fifty adult female albino rats (*Rattus norvigicus*) weighting about 110 ± 10 g were obtained from the breeding unit of Egyptian organization for biological products and vaccines, Helwan, Egypt. The rats were kept in the laboratory for one week before the experimental work and maintained on a standard rodent diet (20% casein, 15% corn oil, 55% corn starch, 5% salt mixture, and 5% vitaminized starch; Egyptian Company of Oils and Soap Kafr-Elzayat, Egypt) and water available ad libitum. Light was on a 12:12 hr light to dark cycle. The experimental protocol was approved by Local Ethics Committee and Animals Research.

2.3. Experimental protocol

The rats were randomly and equally divided into 5 groups (10 animals each).

G1: rats were given the vehicle (corn oil) only at a dose volume of 2.0 ml/kg BW/day by oral gavage for 90 successive days (control group).

G2: rats were intoxicated with chloroform by gavage at a dose of 150 mg/kg BW/day dissolved in corn oil for 90 successive days.

G3: rats were treated with curcumin by gavage at dose of 50 mg/kg BW/day for 90 successive days.

G4: rats were treated with curcumin at a dose 50 mg/kg BW/day by gavage for 30 days then given chloroform at a dose 150 mg/kg BW/days by gavage for 60 successive days.

G5: rats were co-treated with curcumin at a dose50 mg/kg BW/day by gavage plus chloroform at a dose 150 mg/kg BW/days by gavage for 90 successive days.

At the end of experiment, blood samples were individually collected from the inferior vena cava of each rat in heparinized and non-heparinized glass tubes. Blood serum was separated by centrifugation at 3000 rpm for 15 minutes. The collected serum was stored at $-18\,^{\circ}$C. The concentration of cholesterol, triglyceride, high-density lipoprotein-cholesterol (HDL-C) and low density lipoprotein-cholesterol (LDL-C) were determined with Kits from ELLTECH.

Heart was immediately removed; washed using chilled saline solution then weighted. Tissues were minced and homogenized (10% W/V), separately in ice-cold sodium phosphate buffer (0.01 M, pH 7.4) containing 1.15% KCl in a potter El Vehjem type homogenizer. The homogenate was centrifuged at $10.000 \times g$ for 15 min at $4\,^{\circ}$C and the resultant supernatant was used for determination of biochemical parameter.

The activity of MPO was determined by using O diansidine and hydrogen peroxide according to Buchman and Lindenstrom [12]. XO activity was determined according to the method described by Waud and Rajagopalan [13]. CAT activity was measured spectrophotometrically at 240 nm by calculating the rate of regarding to H_2O_2 the substrate of the enzyme [14]. The glutathione peroxidase activity (GSH-Px) was measured according to the method of Gross et al. [15]. According to the method of Mesbash et al. [16], the extent of lipid peroxidation was measured in the term of thiobarbituric acid reactive substance (TBARS) formation was measured. Tissue total antioxidants capacity were measured by the method of described by Benzie and Strain [17]. Total thiol content and non protein thiol (GSH) were performed according to Sedlak and Lindsay [18]. The protein content of tissues was determined by the method described by Lowry et al. [19].

Three rats from each group were anesthetized with Thiopental. The thorax was opened with surgical incision on the sternum and the perfusion was done from left ventricle and right atrium. A rinsing solution was perfused before the fixation solution (10% neutral buffer formalin). To make rinsing solution, 9.0 g NaCl, 25 g polyvinyl pyrrolidone, 0.25 g heparin, and 5.0 g procain-HCL were dissolved in one liter of water by thorough stirring. The pH was adjusted to 7.35 with 1 N NaOH and twice filtered through Millipore filters of 3.0 μm or less pore size. The perfusion of both solutions was performed by using a scalp vein attached to a 50cc syringe. Hearts were immediately removed taking care to handle specimens gently and a portion of left ventricular was fixed in 10% neutral buffered formalin for standard histological processing paraffin sections. Sections were used for haematoxylin and eosin stains as a routine method after Bancroft and Stevens [20] for general morphological evaluation.

2.4. Statistical analysis

One-way analysis of variance (ANOVA) was used to assess significant difference among treatment groups. For each significant effect of treatment, the Dunnett lest was used for comparisons. The criterion for statistical significance was net at $P \leq 0.05$ or $P \leq 0.01$ [21].

3. Results

The present study was intended to characterize the role of oxidative stress in the mechanism of $CHCl_3$ toxicity by studying the change in same parameters after sub chronic exposure. Table 1 shows significant decrease in body weight gain in group treated with $CHCl_3$ and group treated with CMN then $CHCl_3$ while a significant increase in relative organ weight of heart in both groups II and IV as compared with the control. Table 2 shows the changes in lipid profile in different groups under study; serum cholesterol, LDL-C and TG were significantly higher in group II ($P < 0.01$) and group IV ($P < 0.05$) as compared with the control. Mean serum HDL were significantly lower ($P < 0.01$) in groups II and IV as compared with the control. On the contrary, we showed that curcumin alleviated the effect of CHCl3 in group treated with curcumin plus $CHCl_3$. Also, curcumin depleted significantly the level of cholesterol, TG, LDL-C in group treated with CMN. Table 3 shows significant increase of MDA and reduction of protein content in ($P < 0.01$) in both groups II, IV as compared with the control. In contrast, we showed significant decrease ($P < 0.01$) in MDA concentration of group treated with CMN and significant as compared with the control group. On the other hand, we showed insignificant change in protein content of group III as compared with the control. Total thiol content, GSH content and TAC were significant decrease in both groups II, IV in comparison to its corresponding of group I, in the contrast, we showed significant increase ($P < 0.01$) in group treated with CMN as compared with curcumin (Table 3).

The activities of GSH-Px and CAT in heart of all studied groups were shown in Table 3, a significant decrease ($P < 0.01$) of both groups II, IV in correspondence with the control. GSH-Px and CAT enzymes activities account significant increase in group treated with CMN as compared with the control. The present results showed that $CHCl_3$ significantly elevated ($P < 0.01$) the activities of xanthine oxidase and myeloperoxidase in heart of rats treated with CHCl3 (Table 3). Although the current study reflected that the

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