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Hepatoprotective effect of *Prunus Persica* leaves extract against carbon tetrachloride induced hepatic injury in rats

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ABSTRACT

The aim of the present study was to evaluate the hepatoprotective effect of *Prunus persica* leaves extract against carbon tetrachloride induced hepatic injury in rats. Animals in Group I served as normal control (distilled water) group, Group II served as toxic control (CCl_4 treated) group, Group III served as standard (Silymarin) group, and Group IV, V served as (200 & 400 mg/kg bw, p.o) *Prunus persica* leaves extract treated groups respectively. The levels of the serum biomarkers such as serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), bilirubin, albumin & total protein were significantly increased in CCl_4 treated rats when compared with the normal control group but the rats treated with *Prunus persica* leaves extract (200 mg/kg) showed decrease in SGOT (250.5 ± 4.43), SGPT (176.4 ± 5.20), ALP (242.43 ± 6.30), bilirubin (2.20 ± 0.05) level and increase in serum albumin (3.20 ± 0.05) and total protein (3.81 ± 0.07) level as compared to that of toxic control group. The rats pre administered with ethanolic extract, 400 mg/kg (Group V) showed the more decrease in levels of SGOT (208.4 ± 5.52), SGPT (95.63 ± 5.30), ALP (204.41 ± 5.03), bilirubin (1.76 ± 0.02) level and increase in serum albumin (3.99 ± 0.06) and total protein (4.69 ± 0.08) level when compared to that of toxic control group. On the basis of results obtained, it can be concluded that the ethanolic extract of *Prunus persica* leaves seems to have hepatoprotective activity which may be due to the presence of flavonoids.

Keywords: *Prunus persica*, Hepatoprotective Activity, Serum Enzymes, Silymarin, CCl_4 .

INTRODUCTION

Liver is the vital organ of metabolism and excretion. Hepatotoxicity is a damage or injury to liver which is caused by various drugs, chemicals and other agents. Extent of liver damage or injury depends on degree of exposure, mild liver damage cause dysfunction but severe liver damage result in liver failure [1]. Damaged liver is unable to perform all these functions properly and it may not lead to secrete bile acid which is the primary way that liver dispose of waste product [2]. Plant drugs are known to play a vital role in the management of liver diseases. There are numerous plants and polyherbal formulations claimed to have hepatoprotective activities [3]. *Prunus persica* which belongs to family Rosaceae is a plant growing in temperate region used as laxative, sedative, anti-cancer and also consist of glycosides, flavonoids, anthocyanins, vitamins etc [4]. It also possess hepatoprotective property. To prove the activity scientifically the ethanolic extract of *Prunus persica* was studied against CCl_4 induced Hepatotoxicity in albino rats.

MATERIALS AND METHODS

2.1 Chemicals

All chemicals used were of analytical grade. The kits for the estimation of serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), bilirubin and total protein were purchased from Span Diagnostics Ltd. India.

2.2 Plant Collection, Authentication and Extraction

The leaves of *Prunus persica* were collected from the garden of S.G (P.G) College, Sarurpur Khurd, Meerut in the month of September and then authenticated by Dr. H.B. Singh, Chief Scientist & Head, Raw Materials Herbarium & Museum (RHMD), National Institute of Science Communication & Information Resources (NISCAIR), New Delhi vide voucher no. (NISCAIR/RHMD/Consult/-2012-13/2111/118).

The leaves of *Prunus persica* were dried under shade and powdered with a mechanical grinder and passed through sieve no.40. The sieved powder was stored in airtight container and kept at room temperature. Coarsely powdered leaves (350 g) were extracted with soxhlet apparatus using petroleum ether for about 24 hrs. After defatting, the marc was dried in hot air oven at 50°C, packed in soxhlet apparatus, and further extracted with 95% ethanol. The solvent were removed from the extracts under reduced pressure by using rotary vacuum evaporator [5].

2.3 Animals

Wistar albino rats (180-200 g) were used for all other studies, and they were housed at a temperature of 23±2 °C and humidity (50-55 %) with 12 hrs light and dark cycles. They were caged with a maximum of three animals in each polypropylene cage and were fed with standard diet and water *adlibitum*.

2.4 Hepatoprotective activity

Animals were randomly divided into five groups of six animals each. Group I served as normal control and received distilled water (5 ml/kg), Group II served as toxic control and received distilled water (5 ml/kg) for 9 days and 1:1(v/v) mixture of CCl₄ in liquid paraffin (2 ml/ kg i.p.) on 9th day, Group III served as standard group and received silymarin (100 mg/kg) for 9 days and 1:1(v/v) mixture of CCl₄ in liquid paraffin (2 ml/ kg i.p.) on 9th day, while Group IV and V were treated with ethanolic extract of *Prunus persica* at the dose of 200 and 400 mg/kg/day, p.o. for nine days, respectively [6].

2.5 Analysis of liver function enzymes

After 24 hours of CCl₄ administration (Day 10th) blood samples were collected by retro-orbital plexus puncture under mild ether anesthesia. The collected blood was allowed to clot at room temperature and serum was separated by centrifugation at 2500 rpm for 15 min. Then serum was used for the estimation of biochemical parameters such as serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), total bilirubin (TB) and total protein (TP). The biochemical parameters were estimated as per the standard procedure prescribed by manufacturer's instruction manual provided in the standard kit using autoanalyser [7].

2.6 Statistical Analysis

All the data are expressed as Mean ± S.E.M. One way analysis of variance (ANOVA) was used for the statistical analysis of data. Dunnett's multiple comparison tests was used for determining the significance. A value of p<0.05 was considered as significant [8].

RESULTS

The result of the present study showed that, the levels of SGOT [(313.43±6.63) IU/L], SGPT [(240.03±6.55) IU/L], ALP [(407.53±6.12) IU/L] and bilirubin [(3.17±0.05) mg/dl] were significantly increased in toxic control group (Group II) when compared with normal control group but the levels of albumin [(1.87±0.04) g/dl] & total protein [(3.08±0.32) g/dl] were significantly decreased. Rats pre-treated with *Prunus persica* leaves extract (200 mg/kg) showed significant reduction in the levels of SGOT, SGPT, ALP & bilirubin when compared with toxic control group. But the maximum reduction of SGOT [(208.4±5.52) IU/L], SGPT [(95.63±5.30) IU/L], ALP [(204.41±5.03) IU/L] & bilirubin [(1.76±0.02) mg/dl] were observed in the high dose group (400 mg/kg). Moreover, the level of

albumin [(3.99±0.06) g/dl] & total protein [(4.69±0.08) g/dl] were significantly increased in the high dose group.

Parameters	Normal Control	Toxic Control	Silymarin (100 mg/kg)	Extract (200 mg/kg)	Extract (400 mg/kg)
SGOT (IU/L)	87.36±2.82	313.43±6.63 ^{##}	185.73±5.63*	250.5±4.43**	208.4±5.52**
SGPT (IU/L)	61.4±2.73	240.03±6.55 ^{##}	86.3±3.54*	176.4±5.20**	95.63±5.30**
SALP (IU/L)	142.15±4.60	407.53±6.12 ^{##}	181.55±0.52**	242.43±6.30**	204.41±5.03**
Sr. Bilirubin (mg/dl)	0.65±0.02	3.17±0.05 ^{##}	1.14±0.17*	2.20±0.05**	1.76±0.02**
Sr. Albumin (g/dl)	4.10±0.15	1.87±0.04 ^{##}	3.11±0.07**	3.20±0.05**	3.99±0.06*
Sr. Total Protein (g/dl)	6.18±0.10	3.08±0.32 ^{##}	5.23±0.08**	3.81±0.07**	4.69±0.08**

All values represent Mean ± S.E.M. of n=6/group; ^{##} p<0.01 when compared with normal control & *p<0.05, **p<0.01 as compared with toxic control group.

DISCUSSION

The present study involves the evaluation of hepatoprotective activity of ethanolic extract of *Prunus persica* leaves against CCl₄ induced liver damage in rat liver. Carbon tetrachloride has been used as a tool to induce hepatotoxicity in experimental animals. Carbon tetrachloride has been used as a tool to induce hepatotoxicity in experimental animals. CCl₄ induces hepatotoxicity by metabolic activation; therefore it selectively causes toxicity in liver cells maintaining semi normal metabolic function [9]. CCl₄ is biotransformed by cytochrome P450 system in the endoplasmic reticulum to produce trichloromethyl free radical (CCl₃). CYP2E1 is the major isozyme involved in bioactivation of CCl₄ and subsequent production of free radicals [10]. Trichloromethyl free radical when combined with cellular lipids and proteins in the presence of oxygen, form trichloromethylperoxyl radical (CCl₃O[•]), which may attack lipids on the membrane of endoplasmic reticulum and causes a chain reaction; faster than trichloromethyl free radical [11]. Thus, trichloromethylperoxyl free radical leads to elicit lipid peroxidation; destruction of Ca²⁺ homeostasis and finally results in cell death [12]. The present study showed that ethanolic extract of *Prunus persica* leaves possess hepatoprotective activity, as evidenced by the significant inhibition in the elevated levels of serum enzymes activities induced by CCl₄.

There was an increase in SGOT (↑258.7%), SGPT (↑290.6%), ALP (↑186.6%) and bilirubin (↑387.6%) level of CCl₄ + distilled water treated group when compared to that of normal control group where as there was decrease in serum albumin (↓54.3%) and total protein (↓50.1%) level. Further, rats pretreated with standard drug silymarin (100 mg/kg) exhibited decrease in SGOT (↓40.7%), SGPT (↓64.0%), ALP (↓55.4%), bilirubin (↓64.0%) level and increase in serum albumin (↑66.3%) & total protein (↑69.8%) level as compared to that of toxic control group. Also, the rats pretreated with ethanolic extract of *Prunus persica* (200 mg/kg) showed decrease in SGOT (↓20.0%), SGPT (↓26.5%), ALP (↓40.5%), bilirubin (↓30.5%) level and increase in serum albumin (↑71.1%) and total protein (↑23.7%) level as compared to that of toxic control group. But rats pre administered with ethanolic extract, 400 mg/kg (Group V) for one week; showed the more decrease in percentage of SGOT (↓33.5%), SGPT (↓60.2%), ALP (↓76.1%), bilirubin (↓41.7%) level and increase in serum albumin (↑113.3%) and total protein (↑52.2%) level when compared to that of toxic control group.

The ethanolic extract of *Prunus persica* leaves definitely possess hepatoprotective properties in the dose dependant manner, against CCl₄ intoxication in rats, after one week pretreatment; at the dose level 200mg/kg and 400 mg/kg.

CONCLUSION

The present study reports the hepatoprotective activity of *Prunus persica* leaves extract on CCl₄ induced hepatotoxicity in rats. Phytochemical screening revealed the presence of glycosides, flavonoids, alkaloids, phytosterols, saponins and phenolic compounds in the extract. Several investigators have shown that plant extract containing flavonoids are responsible for hepatoprotective potential in various experimental animal models. Thus, it can be interpreted that the hepatoprotective effect may be due to the presence of flavonoids [13].

On the basis of results obtained, it can be concluded that the ethanolic extract of *Prunus persica* leaves seems to have hepatoprotective activity. The further studies are needed to evaluate potential usefulness of ethanolic extract in clinical condition associated with liver damage.

REFERENCES

- [1]. T.S. Mohamed Saleem, M. Chetty, S. Ramkanth, V.S.T. Rajan, K. Mahesh, K. Gauthaman, *Int. J. Res. Pharm. Sci.* **2010**, Vol-1, Issue-1, 1-5.
- [2]. N.P. Yadav and V.K. Dixit, *Journal of Ethnopharmacology*, **2003**, Vol. 86, pp. 197-202.
- [3]. P.V. Habbu, R.A. Shastry, K.M. Mahadevan, J. Hanumanthachar, S.K. Das, *Afr J Tradit Complement Altern med*, **2008**, Vol. 5, Issue 2, pp. 158-164.
- [4]. L. deb, B. Pandey, D. bhowmik, A.S. Dutta, *Scholars Research Library Der Pharmacia Lettre*, **2010**, 2(5): 379-386.
- [5]. M.G. Hogade, K.S. Patil, G.H. Wadkar, S.S. Mathapati, and P.B. Dhumal, *African Journal of Pharmacy and Pharmacology*, **2010**, Vol. 4, Issue 10, pp. 731-734.
- [6]. R. Sundaram and G. Murugesan, *Asian Pacific Journal of Tropical Biomedicine*, **2011** pp. 348-352.
- [7]. V. Priyanka and V. Arpit, *International Journal of Current Biomedical and Pharmaceutical Research*, **2012**, Vol. 2, Issue 1, pp. 222-225.
- [8]. J. Reddy, D. Gnanasekaran, D. Vijay and T.V. Ranganathan, *International Journal of Pharmaceuticals Analysis*, **2010**, Vol. 2, Issue 2, pp. 05-16.
- [9]. M.E. Blazka, J.L. Wilmer, S.D. Holladay, R.E. Wilson, M.I. Luster, *Toxicol Appl Pharmacol* **1995**; 133:43-52.
- [10]. T. Lynch, A. Price, *Amer Fam Physi* **2007**; 76:391-396.
- [11]. N. Verma, C.V. Rao, *International Journal of Pharmacology*, **2010**, Vol. 3, Issue. 2, pp. 140-145.
- [12]. J.D. Castrol, G.C. Ferrya, C.R. Castro, H. Sasama, O.M. Fenos and J.R. Gillete, *Biochemical pharmacology*, **1974**, Vol. 23, pp. 295-300.
- [13]. K.A. Nema, A. Agarwal, *International Journal of Pharmacology*, **2011**, Vol. 3, Issue. 2, pp. 102-105.
- [14]. S.S. Handa, *Text book of pharmacognosy*, **1986**, 3rd edition, pp. 96-99.
- [15]. C.K. Kokate, A.P. Purohit and S.B. Gokhale, *Text book of pharmacognosy*, **2005**, IVth edition, pp.108-114.
- [16]. K.R. Kirtikar, B.D. Basu, *Indian medicinal plants*, Published by international book distributors, **2005**, Vol. 3, pp. 340-345.
- [17]. P. Mohan, T. Mohan, *Text book of pathology*, published by Jaypee Brothers Medical Publishers (P) Ltd., **2005**, 5th edition, pp. 621-622.
- [18]. Robbins, L. Stanley, *Basic pathology*, published by a division of Reed Elsevier India Pvt Ltd, **2005**, 7th edition, pp 456-459.
- [19]. Ross and Wilson, *Anatomy and physiology in health and illness*, **2004**, 9th edition, pp. 307-310.
- [20]. K.D. Tripathi, *Essentials of medical pharmacology*, Jaypee brothers medical publishers (p) ltd, **2008**, 6th edition, pp. 771-772.