Natural Product Sciences
4(4) : 226 ~ 229 (1998)

Antihepatotoxic Activity of *Cassia tora* Leaf Extract

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Abstract – Methanolic extract of *Cassia tora* leaves was evaluated for its hepatoprotective activity in rats by inducing hepatotoxicity with paracetamol (acute model). The extract at a dose of 400 mg/kg orally exhibited significant protective effect by lowering the serum levels of transaminase (SGOT and SGPT), bilirubin, and alkaline phosphatase (ALP). The effects produced were comparable to that of a standard hepatoprotective agent.

Key words – *Cassia tora*, methanolic extract, SGOT, SGPT, ALP, bilirubin, paracetamol, hepatic damage.

Introduction

Cassia tora Linn. (Family Leguminosae) is a well known plant, widely distributed in India and other tropical Asian countries. It is an annual undershrub and grows well in wasteland. It is commonly known as sicklepod. Various parts of the plant are reputed for their medicinal value (Nadkarni *et al.*, 1954). The seeds of *Cassia tora* have been used in Chinese medicine as aperient, antiasthenic, diuretic agents, to improve the visual activity and constitute a valuable remedy in skin diseases, chiefly for ringworm and itch (Kirtikar and Basu, 1975; Chatterjee and Pakrashi, 1992). The leaves are useful against eczema (Asolkar *et al.*, 1992) and also have been found to possess significant antifungal potential (Mukherjee *et al.*, 1996). The leaves of *Cassia tora* contain several anthraquinone glycosides which are well known for their medicinal value. The extracts of *Cassia tora* leaves showed purgative action (Pal *et al.*, 1984). A significant hepatoprotective activity was observed in paracetamol induced rats, when the drug was administered orally at a dose of 400 mg/kg body weight.

Experimental

Plant material – The fresh leaves of *Cassia tora* were collected in the month of August - September. It was identified by Botanical Survey of India, Shibpur, Howrah, India. A voucher specimen (C-02) has been kept in our laboratory for future references. The leaves were air dried, powdered and passed through 40 mesh sieve and kept in a well closed container for further extraction.

Preparation of extract – The powdered drug (500 G) was percolated with 90% methanol (1500 ml). The extract was concentrated under vacuum. Yield of the extract was 25 G which is brownish black in colour. The concentrated extract was used further to study the hepatoprotective activity.

On phytochemical screening the extract showed the presence of an anthraquinone glycoside, characterization of which is under

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Table 1. Effect of methanol extract of *Cassia tora* leaves and liver tonic on serum biochemical parameters during paracetamol induced acute liver damage in rats (n=10)

Groups	SGOT	SGPT	ALP	Bilirubin
Group-I (Control)	50.95±4.253	31.91±3.96	51.98±5.546	1.51±0.322
Group-II (Paracetamol control 750 mg/kg)	189.84±7.766[a]	103.70±6.110[a]	149.64±6.710[a]	6.62±1.125[a]
Group-III, Extract (400 mg/kg)+ Paracetamol (750 mg/kg)	101.98±5.556[b]	50.64±3.924[b]	77.68±5.271[b]	3.59±0.556[c]
Group-IV, Liver tonic (0.5 ml/kg)+ Paracetamol (750 mg/kg)	83.51±4.661[b]	40.80±3.437[b]	68.00±4.240[b]	2.74±0.379[b]

Statistically significantly different from control: [a]$p<0.001$. Statistically significantly different from paracetamol control: [b]$p<0.001$ and [c]$p<0.01$, by student's t-test.

process.

Paracetamol induced hepatotoxicity (acute model) – 40 albino rats of either sex weighing in between 130-150 g were devided into four groups of 10 animals each. Rats of Group I & II were fed orally with 0.1 ml/kg/day of normal saline and Group III & IV were fed orally with 400 mg/kg, and 0.5 ml/kg of *C. tora* leaf extract and standard liver tonic (Neutrosec) respectively for 7 days. On the 7th day paracetamol suspension was administered at a dose of 750 mg/kg, i.p. to rats of Group II, III & IV except Group-I which served as normal saline control (Hiroshini *et al.*, 1987).

Assay of serum GOT and GPT activities – All rats were killed under light ether anaesthesia after 36 h of paracetamol administration and blood was withdrawn from the carotid artery, was centrifuged at 300 rpm for minutes (Chung-Ching *et al.*, 1995) to separate the serum. Serum transaminase activity were measured according to the method of Reitman and Frankel (1957).

Assay of serum bilirubin and serum alkaline phosphatase – Serum bilirubin was estimated following Malloy and Evelyn method (1937). Serum alkaline phosphatase was estimated following Kind and Kings method (1971).

Results

The results of paracetamol induced hepatotoxicity has been represented in Table 1. In rats treated with paracetamol alone (Gr. II) there was significant rise in SGOT, SGPT, Alkaline phosphatase (ALP) and bilirubin values (Table 1). Pretreatment with *C. tora* leaf extract and standard liver tonic (Neutrosec) resulted in significant $p<0.001$, $p<0.01$, $p<0.05$ protection against the increase of SGOT, SGPT, ALP and bilirubin in rats of group II & IV as compared to paracetamol control group II (Table 1, Figs. 2, 3 and 4). However, the liver tonic exhibited more significant protection. Histologically paracetamol treated animals showed central or submassive necrosis (Fig. 2) whereas in the *C. tora* extract and liver tonic treated groups (Figs. 3 and 4) necrotic lesions were absent and were comparable with the control (Fig. 1).

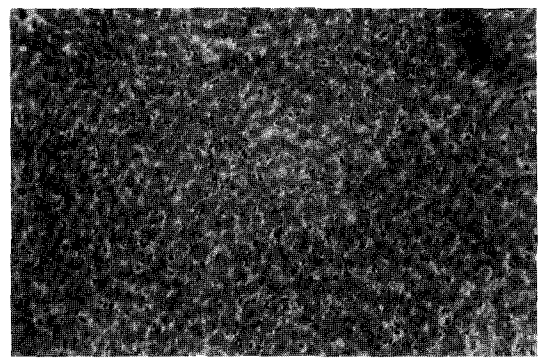

Fig. 1. Normal Control rat. Section of liver showing normal hepatic cells with broughtout nuclei, cytoplasm, central vein and portal triad (H & E×65).

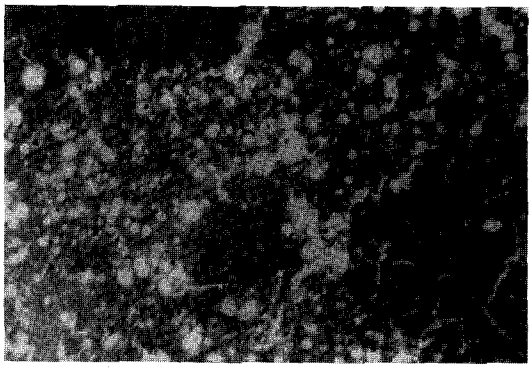

Fig. 2. Paracetamol treated rat. Section of liver showing central or submassive necrosis (H & E×65).

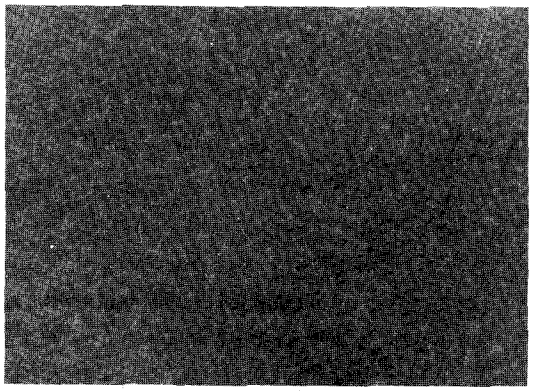

Fig. 3. Representative photomicrograph of liver. Section of rat liver showing absence of necrotic lesions (H & E×65).

Discussion

Paracetamol (acetaminophen), a widely used antipyretic analgesic drug produces acute liver damage if accidental overdosage (which may occur in alcoholics and elderly) are consumed. The co-valent binding of N-acetyl-p-benzoquinoneimine, an oxidation product of paracetamol to sulphydryl groups of protein resulting in cell necrosis and lipid peroxidation induced by decrease in glutathione in the liver as the cause of hepatotoxicity induced by paracetamol have been reported earlier (Jollow *et al.*, 1978, Wendel *et al.*, 1987).

Our findings confirmed the hepatotoxicity of paracetamol (in toxic doses) and free radical mechanism suggested for the toxic effects of this chemical. The paracetamol induced lipid peroxidation was inhibited significantly in *C. tora* leaf extract and neutrosec (liver tonic) treated groups.

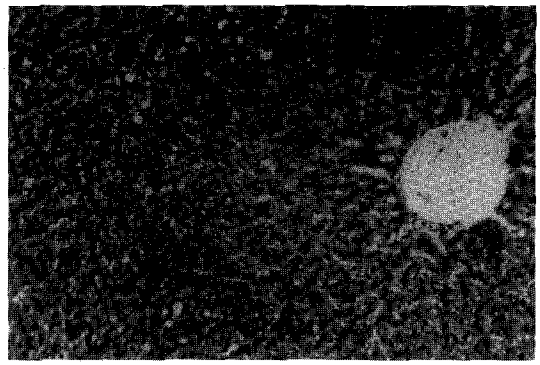

Fig. 4. Paracetamol and liver tonic (Neutosec) treated rats. Section of liver showing normaly of hepatic cells and central vein (H & E×65).

Thus the present study confirm the liver protective action of the methanol extract of *C. tora* against experimentally induced liver damage in rats, which was comparable to that of a standard hepatoprotective drug Neutrosec. SGOT, SGPT, ALP and serum bilirubin are the most sensitive tests employed in the diagnosis of hepatic disease (Herper, 1961). The elevated levels of these parameters were significantly reduced by the treatment of *C. tora* leaf extract as well as liver tonic. This indicates that the extract may be used as an effective hepatoprotective agent.

Acknowledgement

The authors are thankful to the authority of Jadavpur University for providing all facilities.

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(Accepted August 13, 1998)