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Hepatoprotective Activity Of *Cyperus Rotundus L.* Against Paracetamol On Wister Albino Rats

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A B S T R A C T
Plants fulfil the requirements of not only human existence but also whole animal kingdom, specifically due to the occurrence of miscellaneous bioactive compounds. *Cyperus rotundus* L. is one of the most noxious perennial weeds in 52 crops in more than 90 tropical and subtropical countries and ranked as world's worst weed. The nomenclature *Cyperus rotundus* originated from the Latin word 'rotundus' meaning circular or round. The growth habit and mode of propagation of the weed pose tremendous problems in its control. *C. rotundus* cultivates in all types of earth (soil) and even it can endure at advanced temperatures and produces a widespread system of subversive corms from & which they can renew. While comparatively minor in height, this quick increasing plant can rapidly form solid gatherings due to its aptitude to yield a widespread scheme of rhizomes and tubers. The plentifully shaped corms form an effective means of dispersion and imitation. As the Ayurveda said, *C. rotundus* rhizomes are used as diaphoretic, diuretic, astringent, analgesic, aromatic, carminative, antispasmodic, antitussive, emmenagogue, litholytic, sedative, stomachic, vermifuge, tonic, antibacterial and stimulant.

Keywords*: Nutsedge, anti-oxidant activity, hepatoprotective activity, anti-inflammatory activity, antitussive*

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INTRODUCTION
The Sanskrit words "Ayur" and "Veda," which stand for "life" and "knowledge," were combined to form the phrase "Ayurveda"[1].

Ayurveda is the "science of life and longevity" or "knowledge of life"[2].

C. rotundus L., often known as java-grass or nutgrass, is a medicinal plant in the Cyperaceae family. It grows from corms that are approximately 0.39 to 1.18 inches long and have skinny, peeling creep shoots that are rounded on the bottom and rise behind them. Through a distinctive fluorescence, the rhizomes continue to be red-white confidential and seemingly black in colour. The greeneries are rectilinear, shaded green, and fluted at the higher surface levels, and the rhizomes grow to a height of about 9.8 inches. Minor flowerings with 2-4 scales per leaf and little amounts of reddish-brown shell are present. The three rectilinear, ovate, and oblong enthusiast are yellowish in hue and turn blackish when placed. C. rotundus is an Indian native, although it currently thrives in humid, subtropical climates[3].

This plant is the most irritating wildflower known to man, dispersing to a global circulation over humid and temperate regions. C. Due of its extensive invasion of more than 50 crops and identification as a wildflower in more than 100 nations, rotundus has also earned the moniker "the world's vilest wildflower." The plant, like other sedges, usually grows in damp, marshy areas, sand beds, forests, meadows, and wildernesses[4].

This plant produces protruding underground enlarged bulbs and grows yearly, reaching heights of between 7.87 and 15.75 inches. These bulb bases remain dormant beyond the growing season and under conflicting circumstances. They are connected to one another in a constraint by sparse measures of thin rhizomes[5].

The plant C. rotundus has rhizomes that may be utilised for a variety of conditions, including biliousness, malaise, and soreness, as well as for reducing pain, relaxing muscles, and many other conditions. The primary active compounds found in Cyperus rotundus are cyperone, selinene, cyperene, cyperotundone, patchoulenone, sugeonol, kobusone, and isokobusone, which may be used to comprehensively explain both its traditional and alternative medical applications.

Java-grass rhizomes have been used in traditional Iranian medicine to treat biliousness, soreness, and convulsions. Research has shown the plant's potent flavonoids and unrestricted deep-seated searcher behaviour[6].

It is also a form of at-home treatment for gastrointestinal disorders including dyspepsia. The rhizomes are stated to treat a variety of illnesses in the oldest transcripts and are employed in herbal medicine[7].

MATERIALS AND METHODS

Collection and Authentication:

Cyperus rotundus was authenticated at Naugarh, Siddhartha Nagar, UP, India. The plant's rhizomes were dried out at room temperature (20–25 °C) in the dark, then ground into a coarse powder using an electric grinder.

Prof. N. K. Dubey was able to identify and certify the plant (FNASC, FNAAS). in Varanasi, UP, India's Banaras Hindu University (BHU), a taxonomy study of plants. where voucher specimen with number CYPERA.2021/5 was sent.

Plant extractions:

Petroleum ether was used to defattify the powdered Cyperus rotundus.*Cyperus rotundus* ethanolic extract preparation-

Cyperus rotundus rhizome powder (700 g) was defatted with petroleum ether, totally extracted with ethanol using the Soxhlet method at 60 °C for 72 hours, and extracted three more times using the same solvent (ethanol). Whatman No. 1 filter paper let through the extract without any problems (sigma,USA). followed by rotatory evaporation at 40 °C under decreased pressure. For further phytochemical screening, the resultant greenish-brown semi-solid extract was suspended in 200ml of distilled water[8].

METHODOLOGY

Chemicals used:

Ethanol, normal saline, paracetamol, silymarin,formalin

Paracetamol induced hepatotoxicity model:

Rats weighing around 250g were placed into five groups of six: normal control, toxic control for hepatotoxin, test (250/500mg), and positive control (usual treatment). The typical control group received oral vehicle handling (saline/water/tragacanth) at 0 h, 24 h, and 48 h (three days continuously). For the first two days, the animals in the hepatotoxin treatment group were given a vehicle. On day three, they were given paracetamol (in normal saline) at a dosage of 1 ml/kg/day 0. The first test groups (250 mg) received three doses of ethanolic extracts over the course of three days: the first dose at 0 hours, the second dose at 24 hours, and the third dose at 48 hours[9].

The second test group received 500 mg of the ethanolic extract over the course of two days in two doses: the first dose was administered at 0 hours, the second dose was administered at 24 hours, and the third dose was administered at 48 hours. A dose of silymarin (100 mg/kg, normal prescription) was administered to the positive control group at 0 hours, followed by another dose at 24 hours, a dose of paracetamol at 24 hours, and a third dose at 48 hours. After 72 hours, blood samples from each group were taken and allowed to coagulate in order to prepare for serum separation. Using techniques that have been validated, the separated serum was utilised to estimate LFT (SGOT, SGPT, ALKP, and TB)using procedures that have been published in other publications[10].

There are five groups of six animals in each, totalling 30 animals.

Groups	**Treatment/Dose**	**Animal Required**
Group-I: Normal	Saline, water, and 1ml/kg/day for three days of 0.5 percent CMC.	6
Group-II: ToxicControl	on day 3, a single dosage of 3g/kg of paracetamol.	6
Group-III: treated with a common medication (Silymarin)	3 days of paracetamol Plus 100 mg/kg/day of silymarin.	6

Group-IV: Given a test medication (Ethanolic extract of rhizome of Cyperus rotundus)	Every day for three days, 3g/kg of paracetamol and 250 mg/kg of ethanol extract were administered orally	6
Group-V: used a test medication to treat (Ethanolic extract of rhizome of Cyperus rotundus)	Three days of paracetamol 3g/kg with ethanol extract 500 mg/kg/day orally	6

Histopathology research:

Specifically, raised biochemical limits from each test, positive control, hepatotoxin, and control groups were used to determine which animal from the treatment groups had the greatest effect. The liver was removed from the killed animals' bellies by cutting them open. Sections of liver slices that were 5 mm thick were sliced into pieces, stained with haematoxylin-eosin dye, and mounted in di phenyl xylene after being fixed in Bouin's solution for 12 hours (a mixture of 75 ml of saturated picric acid, 25 ml of 40% formaldehyde, and 5 ml of glacial acetic acid). After that, the slices were photographed while being studied under a microscope for histological deviations in liver architecture[11].

Statistical analysis:

For each parameter, the mean values and SEM are determined. percent drop in comparison to Hepatotoxin levels in test samples were estimated using the difference in enzyme levels between100% less hepatotoxin was administered to the treated group compared to the control group. In order to decide the substantial inner group difference was examined using a single, independent analysis for each parameter. After doing an analysis of variance (ANOVA), group comparisons were made on an individual basis. Using Dunnet's approach, mean values were calculated[12].

RESULTS AND DISCUSSION

Biochemical parameters and liver function test:

Since the parameters SGOT, SGPT, ALKP, TBL, and TPT are within the boundaries of control, the ethanolic extract of Cyperus rotundus was shown to be non-toxic at the chosen dose (100mg/kg p.o.) in the evaluation of its effects on normal liver function[13]. In normal rats, the toxic effects of paracetamol greatly increased the levels of SGOT, SGPT, ALKP, TBL, and TPT, indicating acute hepatocellular injury and biliary blockage[14].

In comparison to normal rats, paracetamol intoxification (3g/kg p.o.) caused a considerable rise in the SGPT (52.48±2.76 to 209.76±7.16), SGOT (56.29±2.11 to 322.01±6.23), ALKP (34.03±2.60 to 151.18±2.90), TBL (0.23±0.02 to 1.17±0.04), and a drop in TPTN (3.29±0.07 to 1.25±0.06), and this the remaining group of rats treated with EE at doses of 250 and 500 mg/kg p.o. demonstrated a significant decrease ($p<0.05$) in nearly all of the elevated levels of biochemical parameters and a significant increase ($p<0.05$) in the depleted TPTN level, which was comparable to what was observed in the group of rats treated with silymarin[15].

TABLE 1: CYPERUS ROTUNDUS ETHANOLIC EXTRACT'S IMPACT ON NORMAL LIVER FUNCTION

Groups (N)	SGOT (IU/L)	SGPT (IU/L)	ALKP (IU/L)	TBL (mg/dl)	TPT (g/dl)
Normal control	56.29 ± 2.11	52.48 ±2.76	34.03 ±2.60	0.23 ±0.02	3.29 ±0.07
Toxic control	322.01 ±6.23	209.76 ±7.16	151.18 ±2.90	1.17 ±0.04	1.25 ±0.06
Std (Silymarin 100mg/kg)	86.93 ±1.9	78.23 ±1.82	52.18 ±2.30	0.34 ±0.01	2.87 ±0.06
EECR (250mg/kg)	130.52 ±2.79	129.25 ±2.62	96.06 ±2.06	0.68 ±0.03	2.11 ±0.04
EECR (500mg/kg)	90.68 ±2.4	92.30 ±1.51	63.06 ±1.31	0.47 ±0.02	2.67 ±0.03

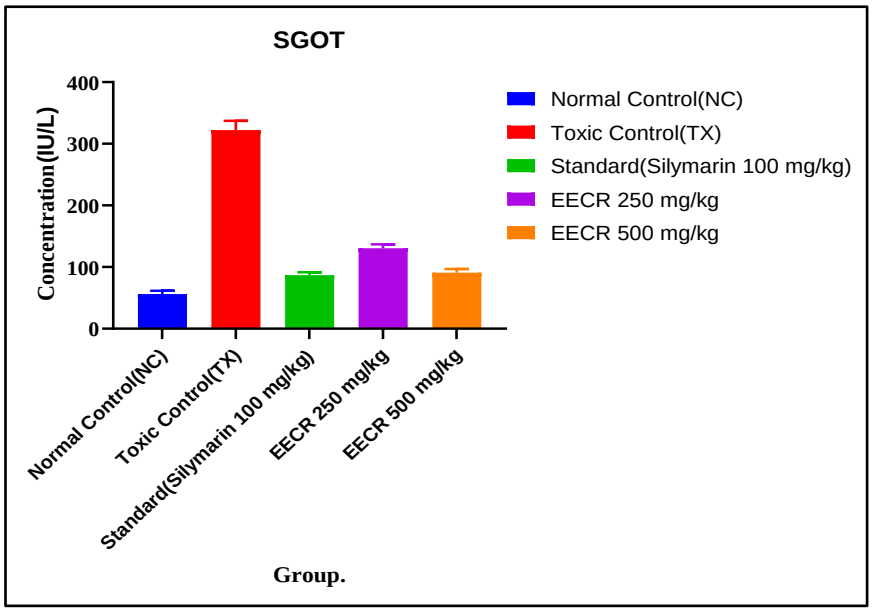

Fig.a. Effect of EECR on serum SGOT (IU/L) against paracetamol induced toxicity in rats.

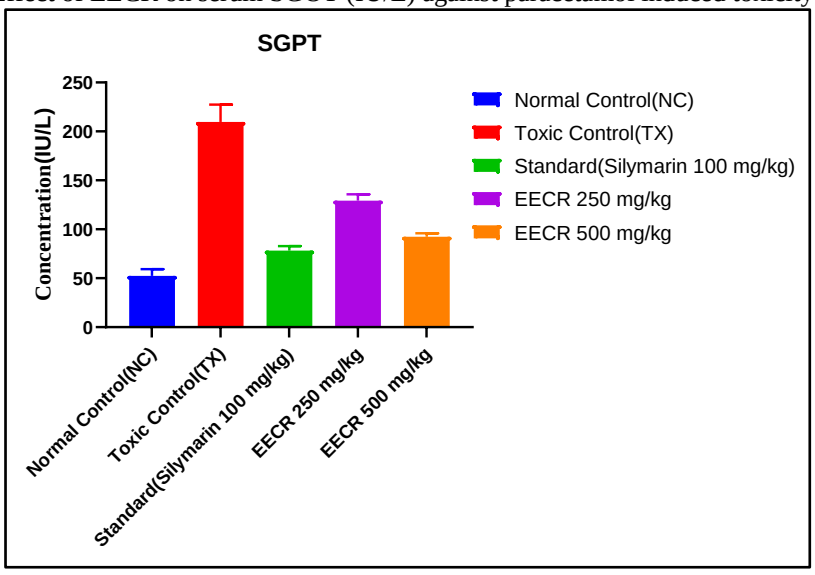

Fig.b. Effect of EECR on serum SGPT (IU/L) against paracetamol induced toxicity in rats.

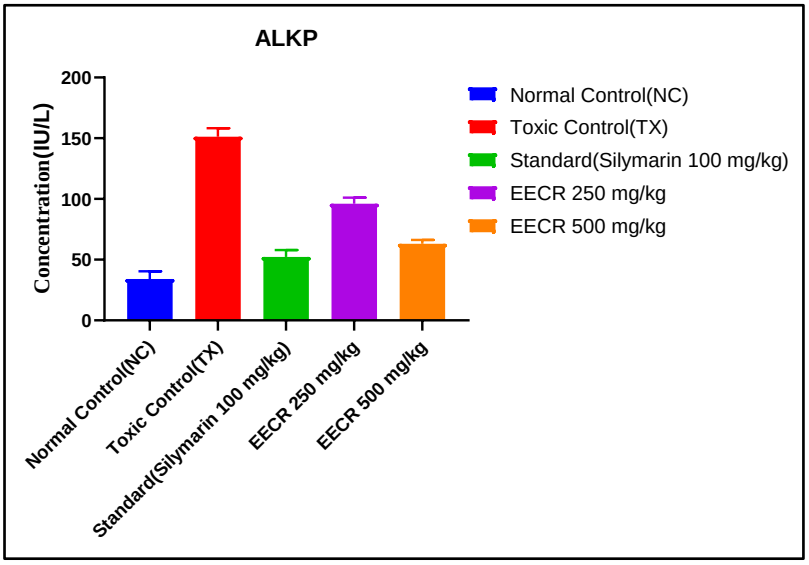

Fig.c. Effect of EECR on serum ALKP (IU/L) against paracetamol induced toxicity in rats.

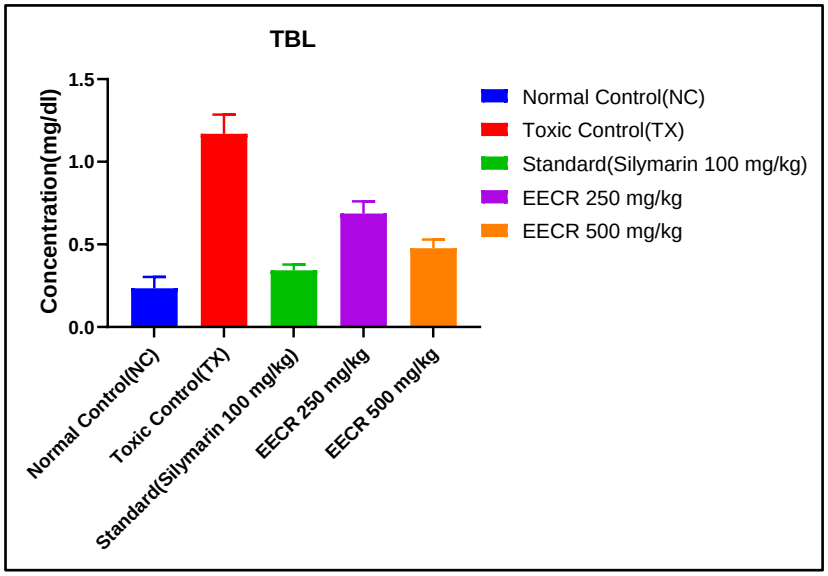

Fig.d. Effect of EECR on serum TBL (mg/dl) against paracetamol induced toxicity in rats.

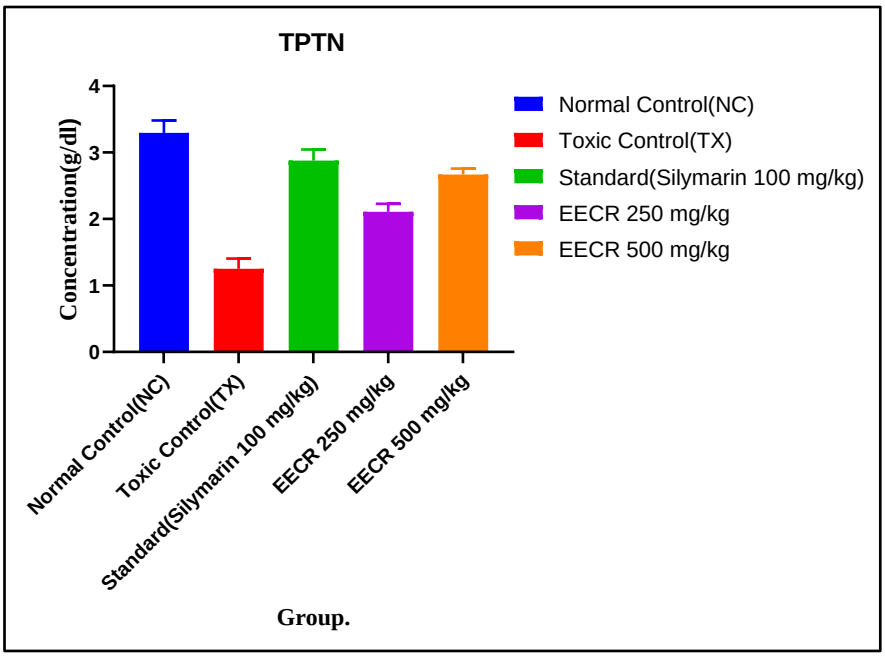

Fig.e. Effect of EECR on serum TPTN(g/dl) against paracetamol induced toxicity in rats.

Histopathological profile:

Rats treated with ethanolic extract's histological profile revealed no discernible alterations, indicating that the extract is safe at the chosen dosing regimen (fig.a)[16]. The liver slices from the control group's histological analysis revealed normal cellular architecture with discrete hepatic cells, sinusoidal gaps, and a central vein (fig.b). In the liver of a paracetamol-toxic individual, abnormal hepatic cells are seen together with severe centrilobular necrosis and vacuolization of the periportal vein (fig.c)[17]. In addition to the preventive effect of the extract, the liver sections of the rats treated with silymarin and drunk with paracetamol (fig.e) and those treated with ethanolic extract and inebriated with paracetamol (fig.d) both revealed reduced vacuole alterations[18].

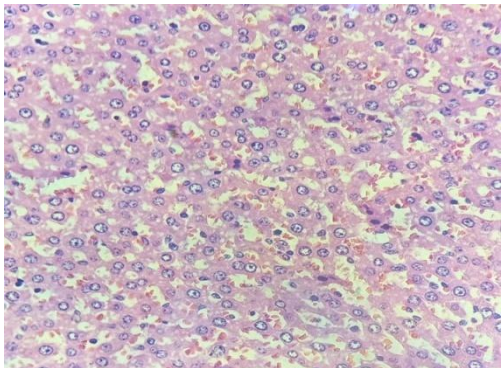

Fig.a Liver section of rat of normal control group

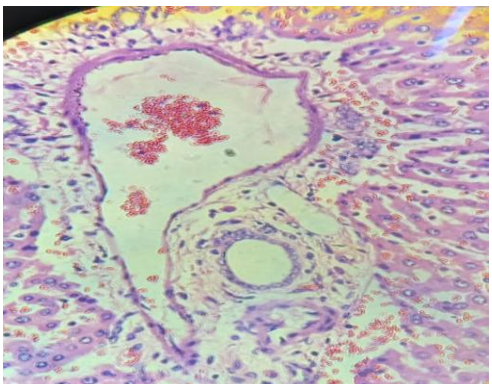

Fig.b liver section of the rat of toxic control group

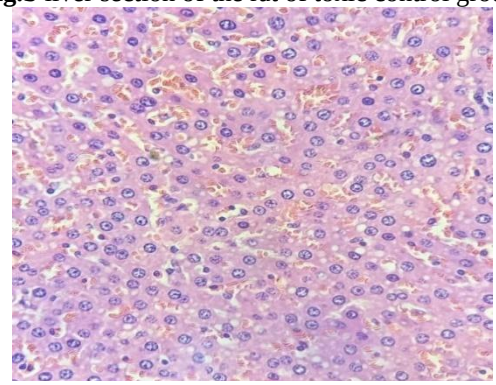

Fig.c Liver section of rat treated with silymarin

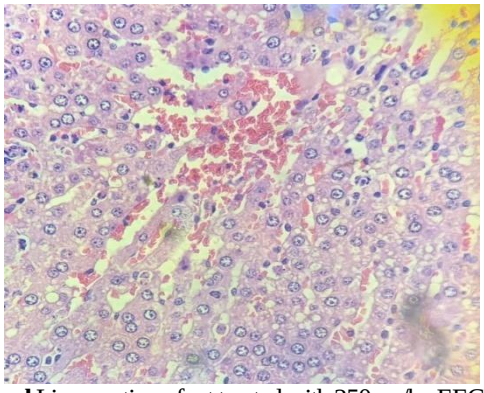

Fig.d Liver section of rat treated with 250mg/kg EECR

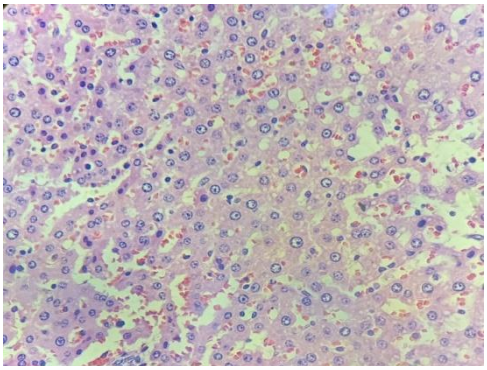

Fig.e Liver section of rat treated with 500mg/kg EECR

CONCLUSION

According to reports, the creation of the extremely reactive free radical, which destroys polyunsaturated fatty acids, is the cause of paracetamol's hepatotoxicity. It causes hepatotoxicity in test subjects by changing the liver microsomal membrane (animals). The ethanolic extract of Cyperus rotundus L. rhizomes effectively prevented the paracetamol-induced hepatotoxicity, confirming the extract's preventive efficacy against experimentally induced liver damage in rats. the increase in SGPO, SGOT, ALKP, and TB as well as a decline in TPTN level[19].

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