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NEPHROPROTECTIVE ACTIVITY OF *TAMARINDUS INDICA* FRUIT EXTRACT ON CISPLATIN INDUCED NEPHROTOXICITY ON ANIMAL MODELS

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ABSTRACT

Medicinal plants are naturally gifted with invaluable bioactive compounds which form the backbone of traditional medicines. Cisplatin is a highly effective chemotherapeutic agent; its clinical use is severely limited by serious side effects as nephrotoxicity. The present study was undertaken to evaluate the ethanolic extract of *Tamarindus indica* fruits pulp for its protective effects on cisplatin induced nephrotoxicity in rats. The Institutional Animal Ethics Committee (IAEC) approved the experimental protocol and the conditions in the animal house approved by Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). Rats were divided into 5 groups; normal control, vehicle, nephroprotective drug (lipoic acid), EETI 200 mg/kg and 400 mg/kg p.o. Anti-oxidant enzymes such as superoxide dismutase and catalase were assayed, and EETI exhibited significantly improved CAT, GPx, MDA, and SOD levels. The levels of renal function markers such as serum creatinine, serum urea, blood urea nitrogen, serum urea, and serum uric acid levels also evaluated, and a significant retrieval was found in a dose-dependent fashion. There was a significant reduction in lipid peroxidation and rise in GSH levels with ethanolic extract of *Tamarindus indica* fruits treatment. These results suggest that the ethanolic extract of *Tamarindus indica* fruits may be useful in reducing the cisplatin induced nephrotoxicity.

Keywords: *Tamarindus indica,* nephroprotective, cisplatin, serum blood urea nitrogen, superoxide dismutase

INTRODUCTION

Plants are essential components of the universe. Medicinal plants have been playing an essential role in the development of human culture. After various observations and experimentations many medicinal plants were identified as source of important medicine. Medicinal plants have been used since prehistoric period for the cure of various diseases. About 8,000 herbal remedies have been described in Ayurveda. The Rig-Veda (5000 BC) has recorded 67 medicinal plants, Yajurveda 81 species, Atharvaveda (4500-2500 BC) 290 species, Charak Samhita (700 BC) and Sushrut Samhita (200 BC) had described properties and uses of 1100 and 1270 species respectively. In compounding of drugs and these are still used in the classical formulations and in the Ayurvedic system of medicine. From time immemorial man depended on plants derived medicines, it is evident that the fascination for plants is as old as mankind itself **[1-4]**.

Figure 1: *Tamarindus indica* plant and fruit

Tamarindus indica Linn. (family-Fabaceae) is distributed in Africa, America, Mexico, Asia and Arabian countries. It is a large, slow growing, long living ever green tree with a trunk of diameter up to 1.5 -2.0 meter. Fruits are usually between 5- 14 cm in length and approximately 2 cm wide. The pulp dehydrates to a sticky paste, enclosed by a few course strands of fibres **[5-8]**.

The present study was focused on the assessment of nephroprotective activity of the ethanolic extract of fruit pulp of *Tamarindus indica* on cisplatin induced nephrotoxicity in Wistar Albino rats.

MATERIALS AND METHODS

Collection and authentication of plant

The fruit pulp of *Tamarindus indica* was collected from Valanchery, Kerala, India. The plant *T. indica* belonging to the family of Fabaceae was identified and authenticated by Prof. Dr. A. Balasubramanian, Director of A.B.S. Botanical Conservation, Research and Training Centre, Karipatti, Salem, Tamil Nadu, India.

Shade drying and cutting of the fruit pulp

The fruit pulp of *T. indica* were collected and shade dried at the room temperature and then cut it in to small pieces, which was used for the extraction for further studies.

Maceration

Fresh fruit pulps of *Tamarindus indica* were cut into small pieces, seeds were removed and air dried. The dried pieces of *T. indica* fruit pulp, weighing 100 g, were soaked in 500 ml of 95% ethanol in a round bottom flask for about 24 h.

Extraction

Extracting values of crude drug are useful for their evaluation, especially when the constituents of a drug cannot be readily estimated by any other means. Further, these values indicate the nature of the constituents present in a crude drug **[9-12]**.

Pharmacological Evaluation

Drugs and chemicals

All the drugs, chemicals and reagents were procured from S.D. Fine Chemicals, Mumbai, India. All chemicals and reagents used were of analytical reagent.

Ethical approval

The Institutional Animal Ethics Committee (IAEC) approved the experimental protocol and the conditions in the animal house approved by Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). The study was conducted in accordance with IAEC guidelines (IAEC approval No: IAEC/XLVIII/03/CLBMCP/2016 dated on 04/05/2016).

Animals

Albino Wistar rats of either sex (150-200 g) was procured from the animal house of King's Institute, Guindy, Chennai, Tamil Nadu, India. Prior to the experiment the rats were housed in a clean polypropylene cage (6 rats/cages) for a period of 7 days under temperature (25-30^{0}C), relative humidity (45-55%).

Selection of animal species

The preferred rodent species was the rat. Normally females were used. Females were generally slightly more sensitive. Healthy young adult animals of commonly used laboratory strains were employed. Females were nulliparous and non-pregnant. Each animal, at the commencement of its dosing, were between 8 to 12 weeks old.

Administration of doses

The test substance was administered in a single dose by gavages using an oral feeding

needle. Animals were fasted prior to dosing (e.g. with the rat, food but not water should be withheld over-night, with the mouse, food but not water was withheld for 3-4 h). Following the period of fasting, the animals were weighed and the test substance administered. After the substance has been administered, food was withheld for a further 3-4 h in rats.

Observation

Animals were observed individually after dosing at least once during the first 30 min, periodically during the first 24 h, with special attention given during the first 4 h, and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead. However, the duration of observation was not fixed rigidly. It was determined by the toxic reactions, time of onset and length of recovery period and extended when considered necessary. The times at which signs of toxicity appeared and disappeared were important, when toxic signs were to be delayed. All observations were systematically recorded with individual records being maintained for each animal.

Experimental protocol

The nephroprotective activity was tested on five groups of Albino Wistar rats of either sex, each group consisting of six animals.

Group-I: Served as normal control received 0.5 % DMSO (Dimethyl sulfoxide); for 15 days.

Group-II: Served as nephrotoxic control, received vehicle (0.5% DMSO); for 15 days.

Group-III: Received the standard nephroprotective drug, [Lipoic acid (50mg/kg; p.o)] dissolved in DMSO for 15 days.

Group-IV: Received ethanolic extract of *T. indica* (200 mg/kg; p.o) dissolved in DMSO for 15 days.

Group-V: Received ethanolic extract of *T. indica* (400 mg/kg; p.o) dissolved in DMSO for 15 days.

On the 10^{th} day 2 h after the administration of standard nephroprotective drug (lipoic acid) and *T. indica* (200 & 400 mg/kg) II-V groups received cisplatin (7.5 mg/kg; i.p.).

Blood collection techniques used in the present study

At the end of the experimental period, i.e. on the 15^{th} day animals were sacrificed under mild ether anaesthesia. The blood was collected by retro-orbital vein puncture using a fine capillary to an anti-coagulant tube and allowed to stand for 30 min at 37°C and then centrifuged to separate the serum to evaluate the biochemical markers.

Preparation of kidney homogenate

The kidney was quickly removed and perfused immediately with ice-cold saline (0.9% NaCl). A portion of the kidney was

homogenized in chilled Tris-HCl buffer (0.025 M, pH 7.4) using a homogenizer. The homogenate obtained was centrifuged at 5000 rpm for 10 min, supernatant was collected and used for various biochemical assays.

Estimation of serum blood urea nitrogen (BUN)

The blood urea was estimated by Berthelot method (Fawcett and Scott, 1960) using the commercially available kit (Kamineni Life Sciences Pvt. Ltd. Hyderabad, Telangana, India). 1000 µl of working reagent-I containing urease reagent, and a mixture of salicylate, hypochlorite and nitroprusside was added to 10 µl of serum, 10 µl of standard urea (40 mg/dl) and 10 µl of purified water to prepare test, standard and blank, respectively. All the test tubes were mixed well and incubated at 37 °C for 5 min. Then 1000 µl of reagent-II containing alkaline buffer, was added to all the test tubes, which were incubated at 37 °C for 5 min. Urease catalyses the conversion of urea to ammonia and carbon dioxide. The ammonia thus released reacts with a mixture of salicylate, hypochlorite and nitroprusside to yield into phenol, a blue- green coloured compound. The intensity of the colour produced is directly proportional to the concentration of urea in the sample and is measured spectrophotometrically at 578 nm. The blood urea was calculated using the following formula:

Blood urea (mg/dl) = Absorbance of test/Absorbance of standard X 40

Blood urea nitrogen (mg/dl) = Serum urea X 0.469

Estimation of superoxide dismutase (SOD)

0.1 ml of Tissue homogenate was added to the tubes containing 0.75 ml ethanol and 0.15 ml chloroform (chilled in ice) and centrifuged. To 0.5 ml of supernatant, added 0.5 ml of 0.6 mM EDTA solution and 1 ml of 0.1 M carbonate-bicarbonate (pH 10.2) buffer. The reaction was initiated by the addition of 0.5 ml of 1.8 mM epinephrine (freshly prepared) and the increase in absorbance at 480 nm was measured. The unit for superoxide dismutase (SOD) is nmoles/mg of protein.

Estimation of catalase (CAT)

0.1 ml of the tissue homogenate was added to the reaction mixture containing 1ml of 0.01M phosphate buffer (pH 7.0) pre-warmed to 37°C, 0.4 ml of distilled water and the mixture was incubated at 37°C. The reaction was initiated by the addition of 0.5 ml of 0.2M hydrogen peroxide and the reaction mixture was incubated at 37°C for one minute. The reaction was terminated by the addition of 2 ml of dichromate-acetic acid reagent after15, 30, 45, and 60 seconds. Standard hydrogen peroxide in the range of 4-20 µl/ moles was taken and treated in the same manner. All the tubes were heated in a boiling water bath for 10 minutes, cooled and the green colour that developed was read at 590 nm against blank containing all

components except the enzyme. Catalase activity was expressed in U/mg protein.

Estimation of glutathione peroxidase (GPx)

Tissue homogenate (approximately 0.5 mg protein) was incubated with 0.1 ml of 5mM GSH, 0.1 ml of 1. 25 mM H_2O_2, 0.1ml of 25 mM NaN_3 and phosphate buffer (0.05 mM, pH 7) in a total volume of 2.5 ml at 37 ^{0}C for 10 min. The reaction was stopped by adding 2 ml of 1.65 % $(HPO_3)_2$- and the reaction mixture was centrifuged at 1500 rpm for 10 min. 2 ml of the supernatant was mixed with 2 ml 0.4 M Na_2HPO_4 and 1ml of 1mM DTNB. The absorbance of the yellow-coloured complex was measured at 412 nm after incubation for 10 min at 37 ^{0}C against distilled water. A sample without the tissue homogenate processed in the same way was kept as non-enzymatic reaction

Estimation of reduced glutathione (GSH)

The supernatant (0.1ml) was made up to 1.0ml with 0.2M sodium phosphate buffer (pH 8.0). Standard GSH corresponding to concentrations ranging between 2 and 10 n moles were also prepared. Two ml of freshly prepared DTNB solution was added and the intensity of the yellow colour developed was measured in a spectrophotometer (Genesys 10-S, USA) at 412 nm after 10 min. The values are expressed as n moles GSH/g sample **[13-22]**.

RESULTS

The *Tamarindus indica* was selected, on the basis of ethnobotanical information which reveals its uses against one of the most hazardous diseases.

Table 1: Percentage yield of ethanolic extract of *Tamarindus indica* (EETI)

Drug	*Tamarindus indica* Linn.
Solvent	Ethanol
Colour	Dark brownish
Consistency	Semi solid
Percentage yield	22 % w/w

Table 2: Effect of the EETI on serum blood urea nitrogen on cisplatin induced nephrotoxicity in rats

Groups	Drug treatment	BUN
I	Normal control (0.5% DMSO)	23.66±0.505
II	Nephrotoxic control cisplatin (0.75%)	58.77±0.792
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	24.15±0.50***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	30.102±0.94*
V	Cisplatin (0.75%) + EETI (400 mg/kg)	24.55±0.55***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control.*

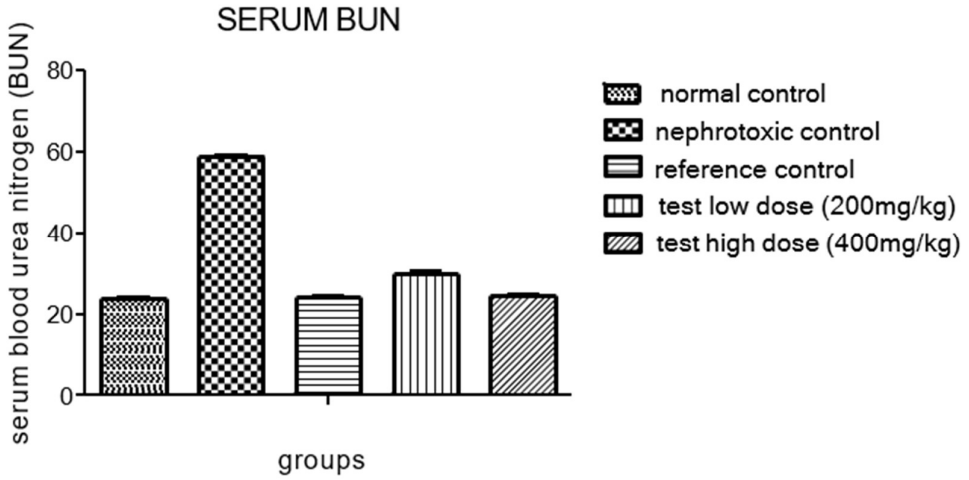

Figure 2: EETI on serum blood urea nitrogen parameters on cisplatin induced nephrotoxicity in rats

Table 3: Effect of EETI on malondialdehyde (MDA) in cisplatin induced nephrotoxic rats

Groups	Drug treatment	MDA
I	Normal control (0.5% DMSO)	7.61±0.470
II	Nephrotoxic control cisplatin (0.75%)	15.44±0.409
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	7.86±0.118***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	8.77±0.427**
V	Cisplatin (0.75%) + EETI (400 mg/kg)	7.66±0.238***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control*

Table 4: Effect of EETI on superoxide dismutase (SOD)

Groups	Drug treatment	SOB
I	Normal control (0.5% DMSO)	19.56±0.591
II	Nephrotoxic control cisplatin (0.75%)	7.53±0.423
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	18.50±0.44***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	11.89±0.303*
V	Cisplatin (0.75%) + EETI (400 mg/kg)	15.57±0.375***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control*

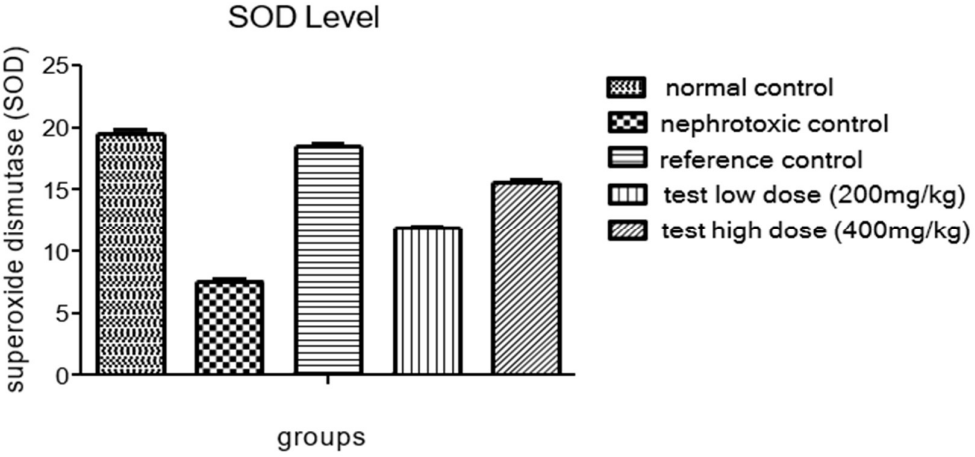

Figure 3: Effect of EETI on superoxide dismutase (SOD)

Table 5: Effect of EETI on catalase in (CAT) cisplatin induced nephrotoxic rats

Groups	Drug treatment	CAT
I	Normal control (0.5% DMSO)	220.31±0.52
II	Nephrotoxic control cisplatin (0.75%)	104.94±0.37
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	200.03±0.612***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	158.39±4.091**
V	Cisplatin (0.75%) + EETI (400 mg/kg)	181±0.265***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control*

Table 6: Effect of EETI on glutathione peroxidise (GPx) in cisplatin induced nephrotoxic rats

Groups	Drug treatment	GPx
I	Normal control (0.5% DMSO)	23.29±0.45
II	Nephrotoxic control cisplatin (0.75%)	14.48±0.448
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	21.39±0.37***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	16.33±0.399**
V	Cisplatin (0.75%) + EETI (400 mg/kg)	19.26±0.228***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control.*

Table 7: Results of the effect of EETI on Reduced glutathione (GSH)

Groups	Drug treatment	GSH
I	Normal control (0.5% DMSO)	20.15±0.776
II	Nephrotoxic control cisplatin (0.75%)	8.28±0.201
III	Reference control cisplatin (0.75%) + lipoic acid (50 mg/kg)	18.47±0.488***
IV	Cisplatin (0.75%) + EETI (200 mg/kg)	14.37±0.280**
V	Cisplatin (0.75%) + EETI (400 mg/kg)	16.33±0.566***

*Values were given in Mean ±SEM; *P<0.05, ** P<0.01 and** *P<0.001 vs nephrotoxic control*

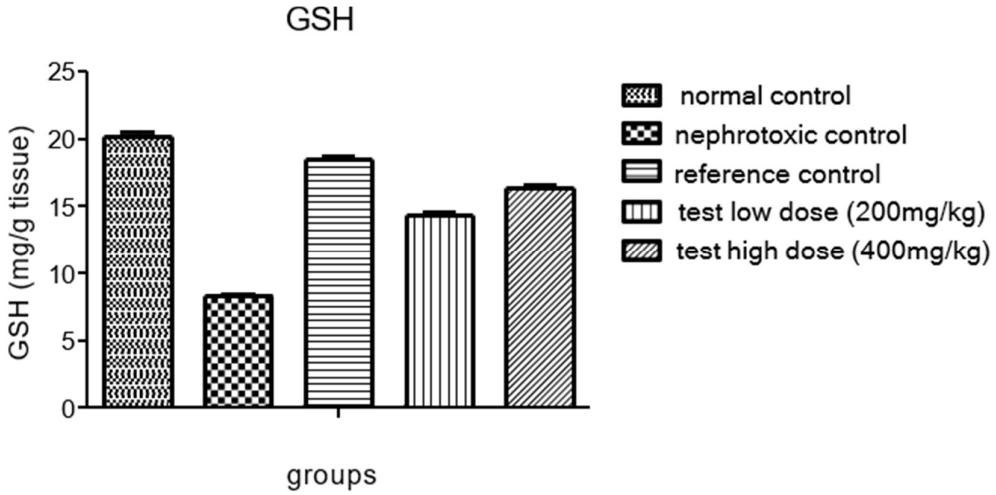

Figure 4: Results of the effect of EETI on Reduced glutathione (GSH)

DISCUSSION

In most of the developing nation's natural sources in particular the phytomedicine is the sole source within the financial and physical reach of the needy people. Furthermore, people had gained faith felt satisfied and happy that most of the therapeutically effective chemical molecules have come from plants. Natural medicine flourished by sharing knowledge both locally as well as across nations. Hence the natural products must continue to hold their quality and significant efforts have to be made to trap the real potential of natural source of medicine.

In present study, the rats treated with single dose of cisplatin shown marked reduction of body weight as compared to normal group also caused a marked reduction of glomerular filtration rate, which is accompanied by increase in serum creatinine level and decline in creatinine clearance indicating induction of acute renal failure. With *T. indica* at the dose level of 200 and 400 mg/kg body weight for 15 days significantly lowered the serum level of

creatinine with a significant weight gain, increased urine output and creatinine clearance when compared with the nephrotoxic control group. Cisplatin administration to control rats produced a typical pattern of nephrotoxicity which was manifested by marked increase in serum blood urea nitrogen. *Tamarindus indica* supplementation to cisplatin treated rats recorded decrement in levels of blood urea nitrogen in plasma.

The elevated level of malondialdehyde (MDA), a marker of lipid peroxidation, indicates increased free radical generation in the cisplatin induced nephrotoxicity. Cisplatin induced increment in malondialdehyde content of plasma was significantly prevented by *Tamarindus indica* treatment in the present study. Therefore, the significantly lower levels of malondialdehyde in the kidney tissues of treated groups as compared with the cisplatin group indicate attenuation of lipid peroxidation. This was probably due to less damage by oxygen free radicals with *Tamarindus indica*. The involvement of oxygen free radicals in tissue injury is well established. Decrement in activity levels of renal superoxide dismutase (SOD), Catalase (CAT) and Reduced Glutathione (GSH) following cisplatin treatment are in accordance with previous report on cisplatin induced suppression of endogenous enzymatic anti-oxidant machinery. *T. indica* treatment efficiently prevented cisplatin induced decrease in activity levels of superoxide dismutase, Catalase and Reduced Glutathione. A relationship between nephrotoxicity and oxidative stress has been confirmed in many experimental models.

Biological systems protect themselves against the damaging effects of activated species by several means. These include free radical scavengers and chain reaction terminators such as GPX system. Glutathione peroxidise (GPx) is a seleno-enzyme two third of which is present in the cytosol and one-third in the mitochondria, it catalyses the reaction of hydro-peroxides with reduced glutathione to form glutathione disulphide (GSSG) and the reduction product of the hydro-peroxide. Effect of *T. indica* on glutathione peroxidise in experimental rats' study were significantly reduced in cisplatin treated rats than in the experimental control rats. Decrement in the activity of renal GPx following cisplatin treatment are due to suppression of endogenous enzymatic antioxidant machinery. Supplementation with *T. indica* to cisplatin treated rats resulted in near normal activity of glutathione peroxidise. Based on the above results, it was concluded that *T. indica* exerted statistically significant nephroprotective activity against cisplatin induced nephrotoxic rats.

CONCLUSION

The present study was undertaken to scientifically evaluate the nephroprotective activity of the ethanolic extract of fruit pulps of *Tamarindus indica*. The administration of cisplatin during experimentation is effectively induced apoptosis and necrosis, which was similar to acute renal failure in human. Therefore, it is an effective and an ideal model for nephrotoxicity research. The evaluation of renal parameters on nephrotoxic rats with EETI showed significantly elevate the attenuated body weight, urine volume, creatinine clearance and significantly reduce in elevated serum creatinine level, which supports its nephroprotective activity. The cisplatin induced rats showed elevated levels of serum blood urea nitrogen and lipid peroxidation parameter like malondialdehyde which was significantly decreased with treatment of EETI, which proves it having nephroprotective activity. The Nephrotoxic rats also showed the reduced levels of enzymatic anti-oxidant like sulfoxide dismutase, glutathione peroxidise and catalase, and non-enzymatic antioxidant like Reduced glutathione, which was significantly increased with treatment of EETI, which showed its anti-oxidant activity due to the flavonoids which is present in the extract. In summary, the fruit pulp of *Tamarindus indica* in an ethanolic extract showed statistically significant nephroprotective activity. The plant extract proved to have nephroprotective potentials may because of its known flavonoid contents and antioxidant properties. There is a scope for further investigation on the histopathology of liver and spleen and clinical studies that are required to elucidate the active phytoconstituents with potent nephroprotective activity. Further studies will be necessary to establish the probable mechanism of action of the nephroprotective activity of the fruit extract of *Tamarindus indica* Linn.

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Declarations

Author contributions

All authors contributed to experimental work, data collection, drafting or revising the article, gave final approval of the version to be published, and agreed to be accountable for all aspects of the work.

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Notes

The authors declare no competing financial interest.

Ethical approval

The study was approved by the Institutional Ethics Committee.

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