WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 10, Issue 12, 1815-1828. Research Article ISSN 2277–7105

# STUDIES ON NEPHROPROTECTIVE ACTIVITY OF *TEPHROSIA PURPURIA LINN* ALCOHOLIC EXTRACT BY CISPLATIN INDUCED MODEL USING ALBINO RATS

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Article Received on
06 Aug. 2021,

Revised on 27 Aug. 2021,
Accepted on 17 Sept. 2021

DOI: 10.20959/wjpr202112-21825

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## ABSTRACT

**Background:** In traditional system of medicine *Tephrosia purpuria linn* plant is extensively used for its activities such as Anti-inflammatory, anti-ulcer, cardio-protective, anti-emetic, anti-fungal, anti-diarrhoeal, hepatoprotective effects. Further no scientific data is available on the nephroprotective activity of this plant, hence in the present study the nephroprotective activity of this plant was explored in experimental rats model. **Objectives:** The nephroproctive activity for the alcoholic extracts of *Tephrosia purpuria linn* whole plant was performed in albino rats by Cisplatin induced nephrotoxicity model. **Methods:** Alcoholic extract of *Tephrosia purpuria linn* plant was prepared by using Soxhlet extraction process. The extract was subjected for $LD_{50}$ studies in mice upto dose level of 2000 mg/kg, p.o. Nephroprotective activity was performed for alcoholic extract of *Tephrosia purpuria linn* plant by Cisplatin induced nephrotoxicity model. Serum samples were analyzed for biochemical parameters Alkaline phosphate, Blood urea nitrogen, Cholesterol, Creatinine, Albumin and Uric acid using semi auto-analyzer. **Results and Discussion:** The $LD_{50}$ studies indicated that none of the mice produced abnormal behavior or mortality rate. Alcoholic extract of *Tephrosia purpuria linn* showed significant Nephroprotective effect. Bio chemical parameters levels were also significantly altered after treatment with different dose of the alcoholic extract in nephrotoxic rats. **Conclusion:** Preliminary phytochemical investigation indicated presence of phytoconstituents like flavonoids, saponins, steroidal triterpines, tannins, carbohydrates and glycosides in the alcoholic extract. *Tephrosia purpuria linn* whole plant alcoholic extract showed significant neproprotective action.

**KEYWORDS:** Alcoholic extract of *Tephrosia purpuria linn*, Phytoconstituents Nephroprotective activity, Cisplatin, Bio chemical parameters.

## INTRODUCTION

Excretion of substances takes place by excretory organs like skin, salivary glands, lungs, gastrointestinal tract, liver and the kidneys. Among all the above channels of excretion kidneys occupy first place and perform excretory, homeostasis and endocrine functions. The urinary system consists of the organs which produce urine and eliminate it from the body thus plays a vital role to maintain the body homeostasis.[1]

According to Smith "the composition of the blood and its internal environment is determined not only by what the mouth ingests but also by what kidney keeps". Hence the main function of the kidneys is to excrete waste products especially the nitrogenous and sulphur containing end products of protein metabolism, maintenance of hydrogen ion concentration of body fluids and electrolytes, water balance of the body and osmotic pressure in blood and tissue.

The term renal failure primarily denotes failure of the excretory function of kidneys, leading to retention of nitrogenous waste products of metabolism in the blood which leads to failure in regulation of fluid and electrolyte balance along with endocrine dysfunction. The renal failure is fundamentally categorized into acute and chronic renal types. Chronic renal failure (CRF) is an irreversible deterioration in the renal function. The cause of CRF has been attributed to hypertension, diabetes mellitus, antineoplastic agents like cyclophosphamide, vincristine and cisplatin etc.

Acute renal failure (ARF) refers to the sudden and reversible loss of renal function, which developed over a period of days to weeks. Acute tubular necrosis is more common accounting for 85% of incidence, occurs either due to ischemia or toxins. The exogenous agents causing ARF are radio contrast agents, cyclosporine, antibiotics, chemotherapeutic agents, organic solvents, acetoaminophen and illegal abortifacients etc.[2]

Kidney diseases are the 9$^{th}$ leading cause of death in United States. Approximately, 19 million adults of United States have chronic kidney disease and estimated 80,000 persons have kidney failure diagnosed annually. Two community based studies have shown a prevalence of chronic renal failure of 0.16% and 0.79% in India. Till date for end stage renal failure, renal replacement is the only therapy. In caseof non-availability of kidneydialysis is the only alternative which unfortunately is severely limited by several constraints including a good amount of expenditure and no exclusive drug has been reported so far, as such in any category of medical treatment.

Herbal drugs are prescribed widely because of their effectiveness, fewer side effects and relatively low cost. According to WHO survey 80% of the population living in the developing countries relies almost on traditional medicine for their primary health care needs. The chemical constituents obtained from plants may be pharmacologically screened for developing novel agents. Phytochemicals are compounds found in plants that are not required for normal functioning of the body, but have a beneficial effect on health or play an active role in amelioration of diseases.[3]

Earlier investigations revealed that 61 different plant families have potential to cure renal diseases. This further includes 143 species of ethnomedicinally important nephroprotective plants in Maharashtra and 78 species found in Khandesh region. Kidney protective herbals contain a variety of chemical constituents like tannins, alkaloids, cardiac glycosides, steroids, terpenoids and flavonoids.[4]

The use of alternative medicinal therapy has increased the interest of pharmacologists and herbalists over the past decades as plants have provided a source of inspiration for novel drug components and made large contribution to health and well being of human beings.

The presently used nephroprotective drugs like ACE Inhibitor, Angiotensin receptor blocker, Furosemide, Erythropoietin, Rutin, are reported to produce unwanted side effects like cough, elevated potassium level, low blood pressure, dizziness, headache, drowsiness, abnormal taste (metallic or salty taste), rash, chest pain, increased uric acid levels, increased BUN and creatine levels, allergic reaction, pancreatitis, liver dysfunction, decreases in WBC, angioedema, muscle cramps, weakness, unusual tiredness, confusion, unusual dryness mouth, nausea, vomiting, fast/irregular heart beat and high blood pressure.[5]

**Cisplatin Induced Nephrotoxicity:**[6,7,8] Cisplatin is chemically known as cis-diamine-dichloro-platinum which is a highly effective anti-neoplastic DNA alkylating agent used in the treatment of wide variety of cancers such as breast cancer, head cancer, testes and ovarian cancer etc.

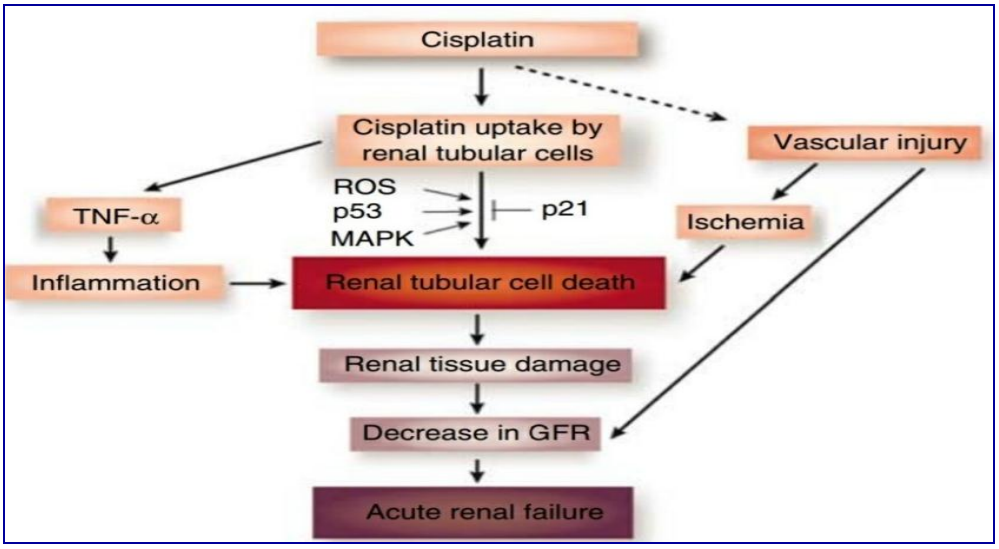


**Fig No 1: Mechanism of Cisplatin induced Nephrotoxicity.**

Higher dose of cisplatin in cancer treatment causes reversible and irreversible adverse reactions such as ototoxicity, gastrotoxicity, myelosupression, nephrotoxicity and allergic reaction. Cisplatin induced free radical production causes oxidative renal damage, possibly due to depletion of non-enzymatic and enzymatic antioxidant systems.

Once cisplatin is filtered by the glomerular filter of the Bowman's capsule, it enters renal cells by passive and or/ facilitated mechanisms. Exposure of cisplatin to the tubular cells activates cell death signalling pathways like MAPK, P53, ROS or cytoprotective. Cisplatin also induce TNF- alpha formation in the tubular cells, which triggers a robust inflammatory response, which causes tubular cell injury and necrotic death of the tubular cells. Cisplatin may also induce injury in renal vasculature, leading to ischemic tubular cells death and decreased GRF. These pathological events may result in acute renal failure.

**Literature Survey**

Studies on plant based products are going on throughout the world for the search of novel and highly effective/protective molecules that would provide maximum protection to the liver, kidney as well as other organs and practically with very little or no side effects exert during their function in the body. A number of herbs are traditionally used in different countries for treating chemical/drug or toxin induced hepatic and renal disorders.[2]

Currently, studies are being conducted worldwide to identify active molecules from the plant origin that can protect the kidney and other organs with few or no side effects. Therefore, this present study is planned to identify herbal medicines capable of protecting chemical/drug

induced nephrotoxicity in experimental animals. To accomplish this, cisplatin is selected to induce oxidative stress on kidney cells, which was then planned to treat with various herbal extracts prepared as mentioned earlier to determine if any of these medicines exert a nephroprotective effect.[9]

The polyphenolic compounds are found in fruits, vegetables, nuts and seeds as well as most types of tea and in red wine. Flavanoids have been reported to exhibit a wide range of biological effects and play a protective role against chronic diseases such as cancer and cardiovascular diseases. They have antioxidant and antiproliferative properties.[5]

The major components of most of the mushrooms extracts revealed that the component of the extracts contains polysaccharide, protein complex, terpenoids show a significant antioxidant activity. A polysaccharide protein complex isolated from *Pleurotus ostreatus* was reported to used as a nephroprotective agent.[7]

Literature has shown that *Harungana madagascariensis* contained high concentrations of glycosides, flavanoids, alkaloids, saponins and tannins. The protection offered by the aqueous extract could have been due to the presence of any of the active principles taking into account that flavonoids, particularly quercetin, in other nephroprotective medicinal plants have been reported of inhibiting xenobiotic-induced nephrotoxicity in experimental animal models due to their potent anti-oxidant or free radicals scavenging effects. In addition, alkaloids have also been reported to strongly inhibit lipid peroxidation induced in isolated tissues via its antioxidant activity. Any of these with their combination could be responsible for the nephroprotective effect. In view of the above, the possible mechanisms for nephroprotective actions of different extracts could be via their antioxidant and/or free radical scavenging activities.

Several independent animal and human studies have reported and confirmed the high safety profile of the plant extracts in the treatment of various diseases. In addition, previous phytochemical studies of *Phyllanthus amarus* extract has reported with the isolation and structural determination of bioflavonoids (e.g. quercetin), lignans (e.g., phyllanthine and hypophyllanthine) and alkaloids. Taking into account that flavonoids particularly quercetin (isolated from other medicinal plants identified with nephroprotective) have been reported of inhibiting xenobiotic-induced nephrotoxicity in experimental animal models due to its potent antioxidant free radicals scavenging effects.[3]

A diet rich in carotenoids is associated with a number of health benefits. Interestingly the use of tomatoes and tomato-based products has been increased as a consequence of many epidemiological studies showed the presence and protective action of phytoconstituents carotenoids, in particular lycopene on cancer and cardiovascular diseases. Lycopene is a major carotenoid, available primarily from tomatoes and its products. Of all carotenoids, lycopene has been shown to exhibit the highest physical quenching rate constant with ROS. The administration of carotenoids with fruits and vegetables instead of carotenoid supplements such as lycopene is rarely associated with nephroprotective effects.

## MATERIAL AND METHODS

**Plant material:** The whole plant of *Tephrosia purpuria* was collected from the fields in and around areas of Raichur, Karnataka in the month of August and September in 2011 and was authenticated by Dr. V.Hemanth Kumar, Professor, Head Department of Pharmacognosy and Phytochemistry, V.L. College of Pharmacy, Raichur, Karnataka.

**Aqueous extract of *Tephrosia purpuria linn* (*AETP*) preparation:** The whole plant was collected and dried under shade and subjected for size reduction to prepare coarse powder with mixer grinder, later the powder was packed in a soxhlet apparatus and extracted with purified water for 18 h (until solvent in the siphon tube of the soxhlet apparatus get decolorized). The extract dried on a water bath at $45^0$ C to get a solid mass. The dried extract was stored in an airtight container and stored in refrigerator at 4-$8^0$ C.

**Experimental Animal care:** Male albino rats used for the acute and chronic experiments were handled in accordance with international principles guiding the use and handling of experimental animals. The albino rats were maintained on standard pellet feed and potable water *adlibitum* at ambient temperature 28 - 30 ºC and 55 ± 5% relative humidity and standard (natural) photoperiod of approximately 12 h of light (06:30 h – 18:30 h) alternating with approximately 12 h of darkness (18:30 h - 06:30 h).[10]

**Acute toxicity studies:**[10] Acute toxicity study of methanol and aqueous extracts will be carried out by using male albino rats with body weight 120-150g, maintained under standard animal husbandry. The animals will be fasted for 3 h prior to the experiment and each extract has to be administering as single dose and observed mortality up to 48 h study period for acute toxicity and 14 days for chronic toxicity. Based on the acute toxicity profile the next dose was determined as per OECD guidelines (No.425).

**Cisplatin induced nephrotoxicity model for alcoholic extract of *Tephrosia purpuria* (*AETP*) for Nephroprotective activity:** [10]Wistar albino Rats were divided into 9 groups of 6 animals in each group (n=6).

**Group I:** Animals were treated with (vehicle) distilled water for 6 days and was kept as normal control.

**Group II:** Animals were treated with distilled water (vehicle) daily 1 h before cisplatin injection at a dose of (10 mg/kg i.p.) for 5 days as toxicant control.

**Group III:** Received with Rutin 20 mg/kg body weight daily 1 h before cisplatin injection for 5 days.

**Group IV, V, VI:** Animals were treated by oral route with different doses of aqueous extract of *Tephrosia purpuria* (low, medium, high) respectively daily 1 h before cisplatin injection for 5 days.

**Group VII, VIII, IX-** Groups were treated daily orally with three different doses of (low, medium, high) aqueous extract for 7 days respectively on 8$^{th}$ day 1 h after extract treatment Cisplatin was administered via intra peritoneal route at a dose of 10 mg/kg body weight.

Then the animals were fasted for 14 h and on 9$^{th}$ day animals anaesthetized with ether and the blood was collected from the retro-orbital puncture, centrifuged and the serum separated was subjected for the estimation of Alkaline phosphate, Blood urea nitrogen, Cholesterol, Creatinine, Albumin and Uric acid. The biochemical parameters were estimated as per the standard procedure prescribed by the manufacturer's instruction manual provided with the biochemical kits. (Erba Diagnostics Mannheim GmbH, Germany) using semi auto-analyzer.

## RESULTS

**Table No. 1: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Alkaline Phosphate estimation (n=6).**

| a) Alkaline Phosphate | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 95.12 | 298.6 | 142.5 | 221 | 215.1 | 171.5 |
| B | 93.23 | 291.5 | 165.2 | 199.5 | 198.5 | 187.5 |
| T | 82.15 | 321 | 152.2 | 211.3 | 194.2 | 172.9 |
| HB | 89.69 | 312.5 | 173.5 | 215.6 | 169.9 | 168.2 |
| BT | 78.96 | 319.1 | 151.2 | 214.8 | 175.9 | 159.9 |
| HT | 87.21 | 313.6 | 143.5 | 185.2 | 189.8 | 169.9 |
| Mean | **87.73** | **309.38*****| **154.68***** | **207.9**** | **190.57***** | **171.65***** |
| SEM | 6.28 | 11.77 | 12.30 | 13.24 | 16.25 | 9.01 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

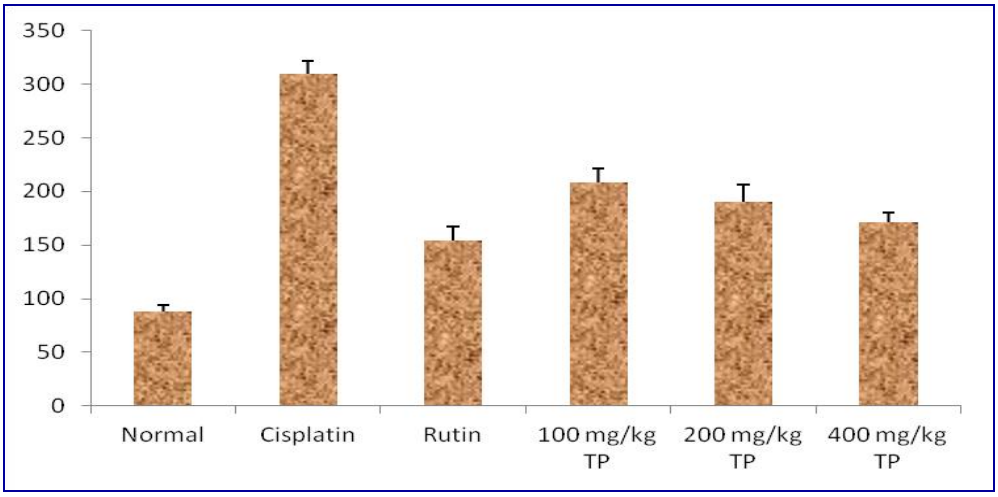


**Fig No. 2: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Alkaline Phosphate estimation (n=6).**

**Table No. 2: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Blood Urea Nitrogen estimation (n=6).**

| b) Blood Urea Nitrogen (BUN) | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 7.82 | 48.6 | 19.8 | 28.8 | 25.9 | 19.9 |
| B | 6.75 | 38.9 | 21.3 | 26.7 | 23.8 | 25.1 |
| T | 7.45 | 37.8 | 25.6 | 25.4 | 19.9 | 18.3 |
| HB | 6.78 | 34.6 | 22.9 | 29.1 | 24.1 | 19.5 |
| BT | 7.41 | 41.9 | 18.9 | 28.3 | 24.9 | 21.8 |
| HT | 9.38 | 43.8 | 24.7 | 29.8 | 28.3 | 22.3 |
| Mean | **7.60** | **40.93*****| **22.2***** | **28.02***** | **24.48***** | **21.15***** |
| SEM | 0.97 | 4.94 | 2.67 | 1.65 | 2.77 | 2.44 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

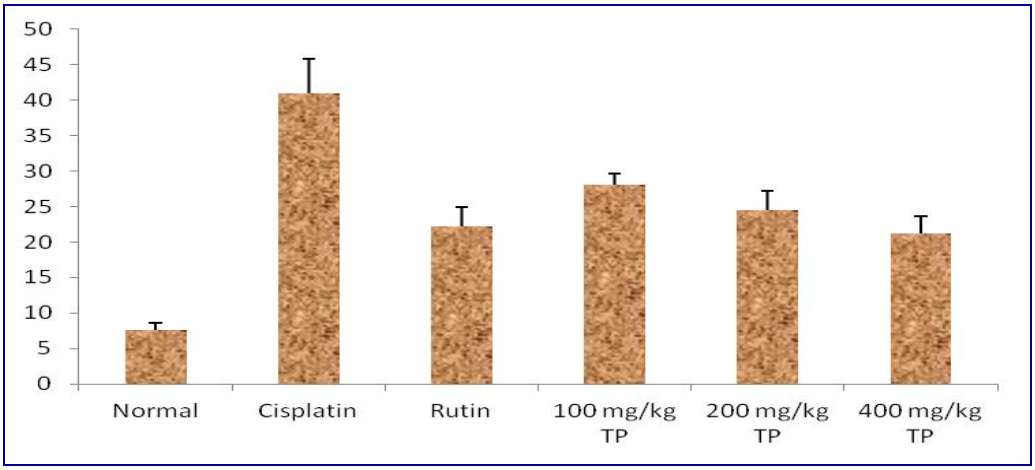


**Fig No. 3: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Blood Urea Nitrogen estimation.**

**Table No. 3: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Creatinine estimation (n=6).**

| c) Creatinine | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 0.91 | 1.82 | 1.06 | 1.35 | 1.28 | 1.24 |
| B | 0.82 | 1.54 | 1.21 | 1.41 | 1.23 | 1.19 |
| T | 0.95 | 1.63 | 1.12 | 1.39 | 1.39 | 1.21 |
| HB | 0.96 | 1.79 | 1.28 | 1.38 | 1.3 | 1.26 |
| BT | 0.79 | 1.65 | 1.35 | 1.34 | 1.28 | 1.23 |
| HT | 0.82 | 1.59 | 1.22 | 1.36 | 1.26 | 1.28 |
| Mean | **0.88** | **1.67*****| **1.21**** | **1.37*** | **1.29*** | **1.24**** |
| SEM | 0.07 | 0.11 | 0.11 | 0.03 | 0.05 | 0.03 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

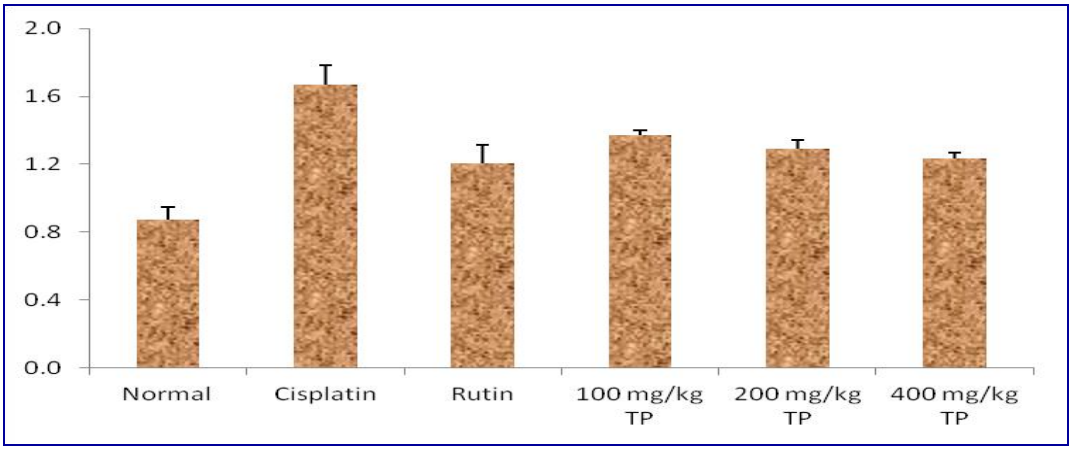


**Fig No. 4: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Creatinine estimation.**

**Table No. 4: Nephroprotective activity of AETP by Cisplatin induced toxicity Cholesterol estimation (n=6).**

| d) Cholesterol | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 192.5 | 287.2 | 184.1 | 243.2 | 205.2 | 194.2 |
| B | 172.1 | 295.3 | 181.9 | 238.9 | 198.3 | 189.1 |
| T | 175.2 | 301.4 | 192.5 | 234.2 | 192.7 | 182.5 |
| HB | 180.1 | 319.8 | 172.4 | 252.1 | 208.1 | 201.4 |
| BT | 158.9 | 279.5 | 169.9 | 241.8 | 203.5 | 196.5 |
| HT | 165.8 | 298.6 | 183.2 | 242.6 | 193.4 | 184.8 |
| Mean | **174.1** | **296.97**** | **180.67**** | **242.13*** | **200.20*** | **191.42**** |
| SEM | 11.66 | 13.76 | 8.29 | 5.90 | 6.39 | 7.24 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

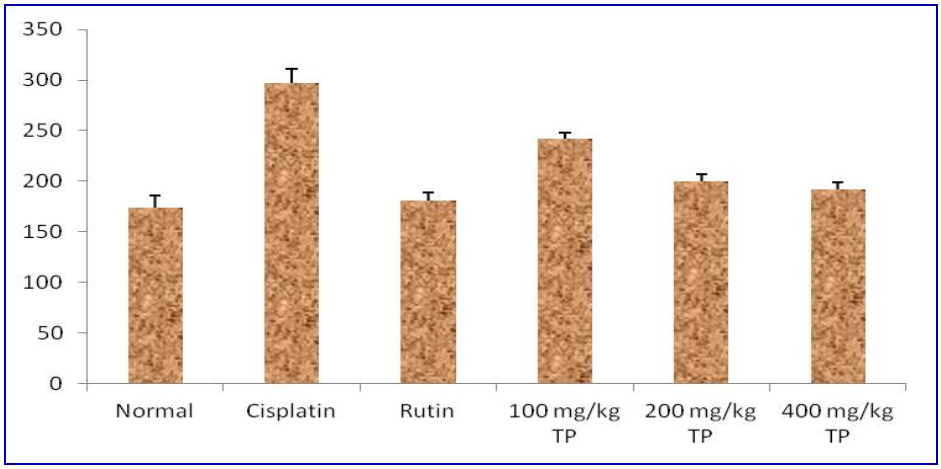


**Fig No. 5: Nephroprotective activity of AETP by Cisplatin induced toxicity Cholesterol estimation.**

**Table No. 5: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Albumin estimation (n=6).**

| e) Albumin | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 3.51 | 2.32 | 4.73 | 3.21 | 3.45 | 3.97 |
| B | 3.53 | 2.28 | 4.9 | 3.18 | 3.48 | 3.84 |
| T | 3.97 | 2.33 | 4.82 | 3.29 | 3.5 | 3.95 |
| HB | 5.02 | 2.21 | 3.98 | 3.34 | 3.47 | 3.79 |
| BT | 3.87 | 2.39 | 4.85 | 3.38 | 3.42 | 3.69 |
| HT | 3.98 | 2.24 | 4.97 | 3.19 | 3.46 | 3.88 |
| Mean | **3.98** | **2.30**** | **4.71***** | **3.27**** | **3.46**** | **3.85***** |
| SEM | 0.55 | 0.07 | 0.37 | 0.08 | 0.03 | 0.10 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

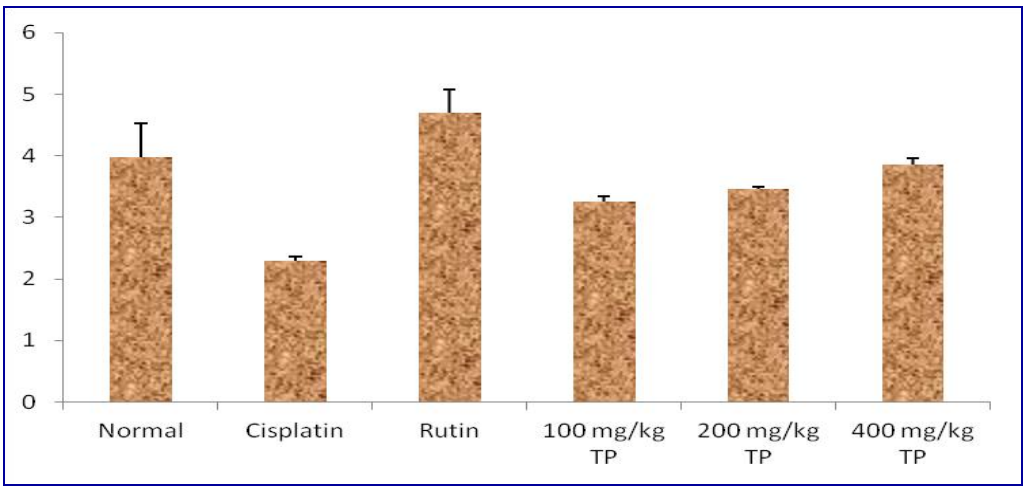


**Fig No. 6: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Albumin estimation.**

**Table No. 6: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Uric acid estimation (n=6).**

| f) Uric acid | | | | | | |
|---|---|---|---|---|---|---|
| **Animals** | **Normal** | **Toxicant (mg/kg)** | **Standard (mg/kg)** | ***AETP* dose (mg/kg)** | | |
| | | **Cisplatin** | **Rutin** | **Low** | **Medium** | **High** |
| | | **10** | **20** | **100** | **200** | **400** |
| H | 5.69 | 13.68 | 9.47 | 10.52 | 9.78 | 9.42 |
| B | 6.38 | 12.96 | 9.89 | 11.87 | 10.02 | 9.75 |
| T | 6.28 | 13.59 | 9.64 | 10.35 | 10.23 | 9.91 |
| HB | 5.92 | 13.92 | 9.43 | 10.95 | 9.91 | 9.54 |
| BT | 6.13 | 11.78 | 9.53 | 10.42 | 10.05 | 9.71 |
| HT | 5.89 | 14.2 | 9.25 | 10.78 | 10.18 | 9.78 |
| Mean | **6.05** | **13.36*****| **9.54**** | **10.82*** | **10.03*** | **9.69**** |
| SEM | 0.26 | 0.88 | 0.22 | 0.56 | 0.17 | 0.18 |
| P < 0.05*, 0.01** and 0.001*** | | | | | | |

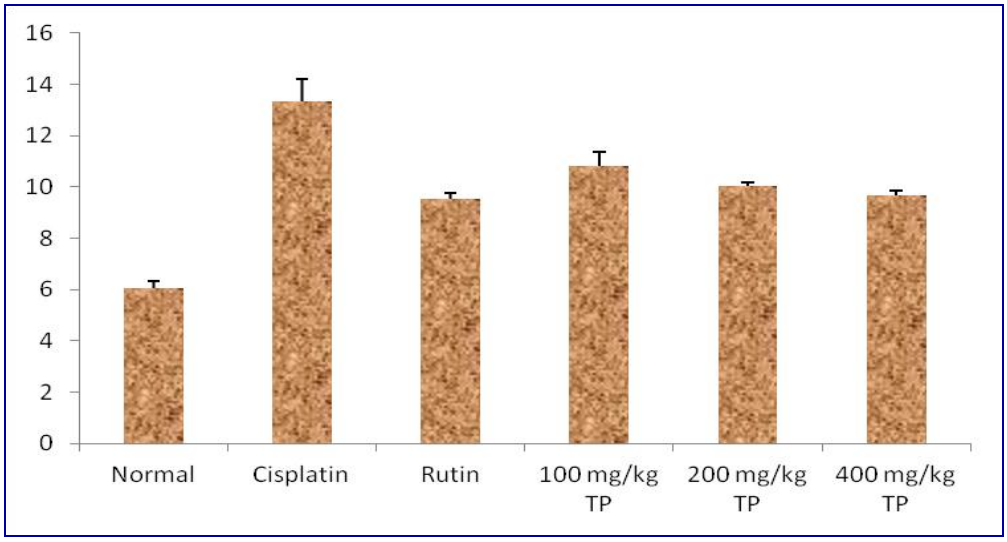


**Fig No. 7: Nephroprotective activity of *AETP* by Cisplatin induced toxicity Uric acid estimation.**

## DISCUSSION

A significantly increased level of serum alkaline phosphatase was observed in blood serum samples of the cispaltin treated group in comparison to the normal group (Table 1 and figure 2). However, supplementation with alcoholic extract of *Tephrosia purpuria* at 100, 200 and 400 mg/kg doses significantly diminished ALP compared to cisplatin group. Highest effect was observed with 400 mg/kg of extract.

In cisplatin treated group of animals the concentration of BUN was considerably increased than the normal animals which indicated severe nephrotoxicity. Treating alcoholic extract of

*Tephrosia purpuria* at 100, 200 and 400 mg/kg doses significantly decreased the concentration of serum BUN compared to cisplatin treated group (Table 2 and figure 3).

Cisplatin administered rats had encountered nephrotoxicity as evidenced by elevation in the levels of creatinine compared to normal animals (Table 3 and figure 4). Treatment with alcoholic extract of *Tephrosia purpuria* at 100, 200 and 400 mg/kg significantly lowered the levels of creatinine when compared with the toxic control group.

Single intraperitoneal administration of cisplatin induced significant rise in serum Cholesterol as seen in the cisplatin treated rats. However, elevation in the level of Cholesterol was significantly attenuated by treatment with the alcoholic extract of *Tephrosia purpuria* (Table 4 and figure 5) at 100, 200 and 400 mg/kg in dose related fashion. The effect of 400 mg/kg extract was comparable to that of standard rutin.

Quantification of serum biomarkers revealed a sharp rise in albumin in the Cisplatin alone-treated group (Group-II) on comparing with the normal group indicating the induction of nephrotoxicity. However, in a dose-dependent fashion, the albumin level significantly decreased (Table 5 and figure 6) presenting the potency of alcoholic extract of *Tephrosia purpuria* towards nephrotoxicity.

A considerable enhancement in the levels of uric acid was observed for the cisplatin alone-treated group on comparing with the normal group (Table 6 and figure 7). Contrarily, a significant decline ($p<0.01$) was noted for uric acid with an administration of the alcoholic extract of *Tephrosia purpuria* at 100, 200 and 400 mg/kg, in the experimental model after inducing nephrotoxicity with cisplatin, indicating the efficiency of the alcoholic extract of *Tephrosia purpuria* with respect to the nephroprotective activity.

**SUMMARY**

Cisplatin (Cis-diamminedichloroplatinum2) is one of the most potent anticancer drugs. High doses of Cisplatin produced impairment in the kidney function. The cisplatin induced nephrotoxicity is characterized by increased lipid peroxidation and change in renal clearance. The cisplatin inhibit antioxidants enzymes in renal tissue and increases the thiobarbuturicacid- reactive substance (TBARS). All the 3 different doses of *AETP* have significantly reduced serum blood urea nitrogen and creatinine levels implying the improvement of GFR in the kidneys.[42,43]

In the present study it was observed that chronic administration of the drug Cisplatin to rats increased the serum levels of marker enzymes like creatinine, BUN, and uric acid as these are stored in the renal cells and increased the levels of these marker enzymes in the serum indicate a damage to the renal cells. Pre-treatment with *AETP* at 3 different doses significantly decreased the levels of alkaline phosphate, blood urea nitrogen, Cholesterol, creatinine, and uric acid levels, where albumin levels were increased which indicated nephroprotective activity of alcoholic extract of *Tephrosia purpuria linn* plant against cisplatin induced nephrotoxicity model.

## CONCLUSION

Preliminary phytochemical investigation of *AETP* extract for phytochemicals like flavonoids, tannins, saponins, glycosides, carbohydrate, proteins, steroid and triterpenes whose presence indicates nephroprotective activity of alcoholic extract of *Tephrosia purpuria linn*. In acute toxicity study of the *AETP* there is no mortality or abnormal behavior were recorded in mice even at the highest dose tasted of 2000 mg/kg, p.o. The nephroprotective activity of the alcoholic extract was evaluated by Cisplatin induced nephrotoxicity in rat models. Standard reference Rutin (20 mg/kg) has exhibited a significant nephroprotective activity in Cisplatin (10 mg/kg) induced nephrotoxicity models in rats. From the study it could be concluded that *AETP* showed a significant nephroprotective effect against Cisplatin induced renal damage as depicted by its protective activity on functional and decreased biochemical parameters levels except albumin in semi auto analyzer. The medium and higher doses of *AETP* (200 and 400 mg/kg) treated groups showed better nephroprotective activity when compared to 100 mg/ kg dose with the extract.

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