A REVIEW ON MEDICINAL PLANTS FOR NEPHROPROTECTIVE ACTIVITY

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ABSTRACT

Medicinal plants may serve as a vital source of potentially useful new compounds for the development of effective therapy to combat a variety of kidney problems. Many herbs have been proven to be effective as nephroprotective agents while many more are claimed to be nephroprotective but there is lack of any such scientific evidence to support such claims. Developing a satisfactory herbal therapy to treat severe renal disorders requires systematic investigation of properties like acute renal failure, nephritic syndrome and chronic interstitial nephritis. Herbal medicines possess curative properties due to the presence of their chemical components. The present review is aimed to elucidate the list of nephroprotective medicinal plants, which are scientifically proved in treating renal disorders.

Keywords: Medicinal plants, nephroprotective agents, renal disorders.

INTRODUCTION

Nephrotoxicity is one of the most common kidney problems and occurs when body is exposed to a drug or toxin. A number of therapeutic agents can adversely affect the kidney resulting in acute renal failure, chronic interstitial nephritis and nephritic syndrome because there is an increasing number of potent therapeutic drugs like aminoglycoside antibiotics, NSAID’s, chemotherapeutic agents have been added to the therapeutic arsenal in recent years. Exposure to chemical reagents like ethylene glycol, carbon tetrachloride, sodium oxalate and heavy metals such as lead, mercury, cadmium and arsenic also induces nephrotoxicity. Prompt recognition of the disease and cessation of responsible drugs are usually the only necessary therapy. Nephroprotective agents are the substances which possess protective activity against Nephrotoxicity. Medicinal plants have curative properties due to the presence of various complex chemical substances. Early literatures have prescribed various herbs for the cure of renal disorders. Co-administration of various medicinal plants possessing nephroprotective activity along with different nephrotoxic agents which may attenuate its toxicity. The term renal failure primarily denotes failure of the excretory function of kidney, leading to retention of nitrogenous waste products of metabolism in the blood. In addition to this, there is a failure of regulation of fluid and electrolyte balance along with endocrine dysfunction. The renal failure is fundamentally categorized into acute and chronic renal failure.

Acute renal failure (ARF) refers to the sudden and usually reversible loss of renal function which develops over a period of days or weeks. There are many causes for acute renal failure which mainly includes acute tubular necrosis that commonly accounts for 85% of incidence. Mostly acute tubular necrosis occurs either due to ischemia or toxins. The toxins may be exogenous or endogenous. The exogenous agents are radio contrast agents, cyclosporine, antibiotics, chemotherapeutic agents, organic solvents, acetaminophen and illegal abortifacients. Chronic renal failure (CRF) is an irreversible deterioration in the renal function which classically develops over a period of years, leading to loss of excretory metabolic and endocrine functions. Various causes of renal failure has been recognized like hypertension, diabetes mellitus, antineoplastic agents like cyclophosphamide, vincristin and cisplatin etc.

Agents Which Causes Nephrotoxicity

Drugs, diagnostic agents & chemical are well known to be nephrotoxic. The following are some of the important nephrotoxic agents.

A) Heavy metal: Mercury, arsenic, lead, bismuth

B) Antineoplastic agents

Alkylation agents: Cisplatin, cyclophosphamide

Nitrosoureas: Streptozocin, Carmustine, Lomustine & Semustine

Antimetabolites: High dose Methotrexate, Cytosine Arabinoside, high dose 6-thioguanine, 5-fluorouracil

Antitumor antibiotics: Mitomycin, Mithramycin, Doxorubicin

Biologic agents: Recombinant leukocyte and interferon

C) Antimicrobial agents: Tetracycline, Acyclovir, Pentamidine, Sulphadiazine, Trimethoprin, Rifampicin, Amphotericin B

D) Aminoglycosides: Gentamycin, Amikacin, Kanamycin, Streptomycin

E) Miscellaneous

Radiocontrast agents: Non-steroidal anti-inflammatory agents (NSAID’s): Ibuprofen, Indomethacin, Aspirin etc.

Nephropathies Caused Due To Different Toxic Mechanisms

Cisplatin Toxicity

Cisplatin is a potent antitumor drug, but its clinical use is limited due to renal toxicity. Cisplatin decreases antioxidants and anti oxidant enzymes leading to enhanced generation of reactive oxygen metabolites and lipid peroxidation. It is reported that many Indian medicinal plants show beneficial effects against renal injury. An early report indicated that nephrotoxicity might occur in as many as 50 to 75% of patients receiving this drug, and is dose limiting. It is used intensively in man, being effective in ovarian & bladder carcinoma, neuroblastoma, head and neck carcinoma, and lymphoma as well as thyroid endometrial neoplasm. However, the most significant activity is observed in testicular cancer. The clinical use of cisplatin is often complicated by nephrotoxicity, ototoxicity, gastrointestinal disturbances like nausea, vomiting and myelosuppression. Early clinical trials of cisplatin in cancer patients showed a striking incidence of persistent azotamia and acute renal failure. Experimental studies have shown that there is an abrupt fall in the effective renal plasma flow within 3 hrs of the i.p. dose of cisplatin. It is known to be filtered by the glomeruli and concentrated in the glomuerular filtrate from which it is activated in the presence of a low intra cellular chloride concentration. The low intracellular concentration of chloride facilitates the displacement of chloride by the water molecule yielding a positively charged, hydrated and hydroxylated complex. Hydration of cisplatin induces formation of monochloro monoaquodiamino platin or diaquo diammineplatin. These agents alkylate the purine and pyrimidine bases of nuclear material. Renal damage is seen in proximal tubular S:portion, the distal tubule and collecting duct. Other proposed explanation of the nephrotoxicity of cisplatin involve the possibility that it include generate reactive metabolites that bind covalently to tissue macromolecules. The nephrotoxic
effects might also be due to sulphhydril binding of heavy metal. A reduction in sulphhydril groups in the rat renal cortex has been demonstrated; this occurred before any significant change in renal function could be detected, suggesting that this biochemical change may be a primary event. Cell fractions have shown that the greatest decline of sulphhydril groups occurs in the mitochondrial & cytosol fractions; these also had the highest concentrations of platinum. A recent study found that cisplatin induced proximal tubule injury could be ameliorated by the administration of hydroxyl radical scavengers. In these studies cisplatin (5mg/kg BW) caused lipid peroxidation. The hydroxyl radical scavenger prevented acute renal failure by altering tubule damage & enhancing the regenerative response of damaged tubule cells protection from cisplatin toxicity has generally focused on providing free radical scavengers.

**Acetaminophen Toxicity**

Acetaminophen is also known as paracetamol. It is a widely used analgesic and antipyretic drug that is safely employed for a wider range of treatments. Overdose of acetaminophen in humans is fairly common and is often associated with hepatic and renal damage. Although nphrotoxicity is less common than hepatotoxicity in acetaminophen overdose, renal tubular damage and acute renal failure can occur even in the absence of liver injury and can even lead to death in humans and experimental animals. Studies are going on throughout the world in search of protective molecules that would provide maximum protection to the liver, kidney as well as other organs and practically very little or no side effects would be exerted during their function in the body. A number of herbs are traditionally used in different countries during in response to drug or toxin induced hepatic and renal disorders.

There are three pathways for acetaminophen metabolism which includes conjugation with sulfate, glucurone and metabolism by cytochrome p450 oxidase enzyme system. 90% of ingested dose is metabolized through glucorionidation and sulfation pathway and 5% through cytochrome p450 oxidase enzyme system. Metabolism by cytochrome p450 enzyme system produces a metabolite, N-acetyl-p-benzoquinine imine (NAPQI) which is toxic to liver and kidney. In therapeutic dose, this is rendered ineffective by reduced glutathione, an antioxidant compound in the liver and NAPQI-reduced glutathione is excreted by kidneys. In acetaminophen overdose, sulfation and glucorionidation pathways become saturated. The amount and rate of formation of NAPQI is greatly increased, depleting body's reduced glutathione stores and outstripping its capability to make new glutathione. NAPQI then binds covalently with cells causing their death, resulting in liver and kidney dysfunction. Indeed several biological compounds with antioxidant properties proved effective in protecting the kidneys against deleterious effects of acetaminophen overdose.

**Gentamicin Toxicity**

Aminoglycoside antibiotics have been widely used for gram-negative bacterial infections. However, their nephrotoxicity and ototoxicity are the major limitations in clinical use. Among several aminoglycoside antibiotics, the grade of nephrotoxicity has been reported to be in the following order as, neomycin > gentamicin > tobramycin. Gentamycin Nephrotoxicity occurs in about 15-30% of treated patients, is manifested clinically as non-oliguric renal failure, with a slow rise in serum creatinine and hypoosmolar urinary output developing after several days of treatment. Gentamicin is filtered through glomeruli into tubular urine, that binds with anionic phospholipids; such as phosphatidylinoisitol or phospholipidylserine, in brush border membrane of proximal tubular cells reabsorbed actively via pinocytosis process into tubular cells, taken up by lysosomes and thereafter produces phospholipidois. The drug enters into the cells by adsorptive/receptor mediated endocytosis after binding to acidic phospholipids and megalin and is found essentially in lysosomes. Animals treated with low, therapeutically relevant doses of aminoglycosides show both lysosomal phospholipidosis and apoptosis in proximal tubular cells.

The following are some of the medicinal plants for review which possess nephroprotective activity.

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### Table 1: List of Nephroprotective plants

<table>
<thead>
<tr>
<th>Botanical Name</th>
<th>Family</th>
<th>Part used</th>
<th>Chemical constituents</th>
<th>Screening method</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Aerva lanata</td>
<td>Rutaceae</td>
<td>Whole plant</td>
<td>Botulin, β-sitosterol, Stigmasterol, Kaempferol, Propionic acid, β-carbolene, Aerosides and Aervolaine.</td>
<td>Gentamycin induced</td>
<td>Paller et al., 1990*</td>
</tr>
<tr>
<td>2. Crataeva nurvala</td>
<td>capparidaceae</td>
<td>Fruit</td>
<td>Kaemferol-3-O-a-D-glucoside, Quercitin-3-O-a-D-glucoside, Flavonoids, Glucosinolates, Steroids, Lupeol and Tannins.</td>
<td>Gentamycin induced</td>
<td>Kore et al., 2011†</td>
</tr>
<tr>
<td>3. Orthosiphon stamineus</td>
<td>Laminaeae</td>
<td>Whole plant</td>
<td>Flavonoids, Phenols, Carbohydrates, Steroids, Tannins, Glycosides, Terpins and Saponins</td>
<td>Gentamycin induced</td>
<td>Kannapan et al.,2010‡</td>
</tr>
<tr>
<td>4. Strychnos potatorum</td>
<td>Loganiaceae</td>
<td>Seed</td>
<td>Flavonoids, Phenols, Saponins, Alkaloids, Steroids, Tannins, Glycosides, and Lignins.</td>
<td>Gentamycin induced</td>
<td>Ruby Varghese et al., 2011§</td>
</tr>
<tr>
<td>5. Aerva javanica</td>
<td>Amaranthaceae</td>
<td>Fresh roots</td>
<td>Isoquercetin, 5-methylmellein, 2-hydroxy-3-O-β-primeveroside naphthalene-1,4-dione, Apigenin7-O-glucoside, and Kaempferol</td>
<td>Cisplatin induced</td>
<td>Vinit movaliya et al., 2011¶</td>
</tr>
<tr>
<td>6. Carica papaya</td>
<td>Caricaceae</td>
<td>Seed</td>
<td>Flavonoids, Phenols, Alkaloids, Protein, Sterols, Terpenoids, Carbohydrates, Steroids, Tannins, Glycosides, Terpins and Saponins</td>
<td>Cisplatin induced</td>
<td>Subal debnath et al., 2010¶</td>
</tr>
<tr>
<td>7. Ficus religiosa L</td>
<td>Moraceae</td>
<td>Latex</td>
<td>Flavonoids, Amino acids and Tannins.</td>
<td>Cisplatin induced</td>
<td>Yogesh chand yadav et al., 2011∥</td>
</tr>
<tr>
<td>8. Pedalium murex Linn</td>
<td>Pedaliaceae</td>
<td>Dried fruits</td>
<td>Flavonoids, Flavone, Alkaloids, Triterpenoids, Carbohydrates, Glycosides and Saponins.</td>
<td>Cisplatin induced</td>
<td>Shelke et al., 2009∥∥</td>
</tr>
<tr>
<td>9. Vernonina cinerea</td>
<td>Compositae</td>
<td>Aerial parts</td>
<td>Triterpenoids, like α-amyrin, β-amyrin and lupeol.</td>
<td>Cisplatin induced</td>
<td>Sreedevi et al., 2011∥∥∥</td>
</tr>
</tbody>
</table>

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* et al., 1990
† et al., 2011
‡ et.al., 2010
§ et al., 2011
¶ et.al., 2011
∥ et al., 2011
∥∥ et al., 2009
∥∥∥ et al., 2011
| 10. | *Acorus calamus* | Araceae | Aerial parts | Monoterpane, Phenylpropanoid, Flavonoids, Quinone and basarone, Glycoproteins. | Acetaminophen induced | Palani et al., 2010 |
| 12. | *Indigofera barberi L* | Fabaceae | Whole plant | Flavonoids, Phenolic acid and sterols. | Acetaminophen induced | Palani et al., 2008 |
| 13. | *Pimpinella tirupatiensis* | Apiaceae | Whole plant | Alkaloids, Flavonoids, Flavones, Volatile oils, β-Bisabolone, Δ-3-Carene, Cis-Carveol, Enol, Δ-Carveol and Methylenegranate. | Acetaminophen induced | Palani et al., 2009 |
| 14. | *Curcuma longa* | Zingiberaceae | Rhizome | Curcumin, Turmeric oil, Terepenoids, Curcumin(Terpen), Starch and Albuminoids. | Cadmium induced | Eduardo Molina-Jijon et al., 2011 |
| 15. | *Drynaria fortunei* | Polypodiaceae | Whole plant | Arsenic, Ca2+, Cu2+, Glucose, Iron, Mg, Mn, Hg, Naringin, K+, Na+, Starch and Zinc. | Silver chloride induced | Kore et al., 2011 |
| 16. | *Eruca sativa* | Crassulaceae | Seeds | Flavanoids | Mercuric chloride induced | Sarwar Alam et al., 2007 |
| 17. | *Moringa oleifera* | Moringaceae | Seeds | Vitamin A, Nicotinic acid, Ascorbic acid, Vitamin B, Fatty acid, Glucose, Sucrose, Citric acid, Malic acid, Succinic acid, Fumaric acid and Oxalic acid. | Fluoride induced | Ranjan et al., 2009 |
| 18. | *Tamarindus indica* | Caesalpinaceae | Fruit pulp | Polysaccharides, Catechin, Nasturtium, Tamarind, Phosphatidic acid, Phosphatidicle choline, Ethanolamine, Serine, Inositol, Alkaloid, Citric acid, Tartraric acid and Pottasium bitartrate. | Fluoride induced | Ranjan et al., 2009 |
| 19. | *Tectona grandis* | Verbenaceae | Bark | Lapachol, Dehydro-α-lapachone, Methyl quinizarin and Squalene. | Allocan induced | Ghasias et al., 2010 |
| 22. | *Euphorbia neriifolia* | Euphorbiaceae | Leaves | Saponins, Flavonoids and Tannins | N-nitroso dimethylamine induced | Pracheta et al., 2011 |
| 24. | *Punica granatum* | Punicaceae | Fruit peel | Ellagic acid, Ellagitannins and gallic acid | Ferric nitrito tri acetate induced | Mahgoub Mohammed et al., 2010 |

**Aerva lanata**

*Aerva lanata* is also called as Pasanbhedha, Chaya, Gorakghanja belongs to the family Amaranthaceae. The *Aerva lanata* plant is reported to have α-amyrin, campesteryl, β-sitosterol, its palmitate, chrysins and flavonoid glucosides. Canthin-6-one and β-carboline alkaloids were isolated from *Aerva lanata*. Four new alkaloids viz., aervine, methylaervine, aeroside and aervoline were isolated. The plant was reported for various activities such as diuretic, hepatoprotective, anti diabetic, antimicrobial, anthelmintic and demulcent activity. *Aerva lanata* also shows its effect on cisplatin and gentamycin model of acute renal failure.

The ethanolic extract of the entire plant of *Aerva lanata* was studied for its nephroprotective activity in cisplatin and gentamycin induced acute renal injury in abino rats of either sex. In the curative regimen, the extract at dose levels of 75, 150 and 300 mg/kg showed dose-dependent reduction in the elevated blood urea and serum creatinine and normalized the histopathological changes in the cisplatin model. In the gentamycin model the rats in the preventive regimen also showed good response to the ethanol extract at 300 mg/kg. The results suggest that the ethanolic extract of *Aerva lanata* possesses marked nephroprotective activity with minimal toxicity and could offer a promising role in the treatment of acute renal failure caused by nephrotoxins like cisplatin and gentamycin.

**Crataeva nurvala**

*Crataeva nurvala* Buch-Ham belongs to the Family Capparidaceae commonly known as Varuna, is an evergreen tree indigenous to India. Moreover, pharmacological study reveals the potentiality of *Crataeva nurvala* extract and its active principle, particularly luteolin as diuretic, anti-inflammatory, antioxidant, cardio-protective, hepatoprotective, lithotriptic, anti-rheumatic, anti-periodic, contraceptive, anti-protozoal, rubifacient and vesicant.

The alcoholic extract of *Crataeva nurvala* 250 and 500 mg/kg for 10 days showed protective activity against cisplatin 5 mg/kg induced nephrotoxicity. The results suggested, that the alcoholic extract has significantly altered the dysfunction of renal proximal tubule cells by decreasing the concentration of blood urea nitrogen, creatinine, lipid peroxidation, glutathione and catalase.
Administration of aqueous extract of *Crataeva nurvula* 200 and 400 mg/kg for 28 days showed protective activity against ethylene glycol induced nephrotoxicity\(^4\).

**Orthosiphon stamineus**

Orthosiphon stamineus Benth. is a medicinal herb belonging to the family Lamiaceae. The plant has extensively been exploited traditionally to treat several human ailments. Leaves of this plant have been used as diuretic, and to treat rheumatism, abdominal pain, kidney and bladder inflammation, edema, gout and hypertension\(^5\). Scientific studies have found that the leaves exhibit dynamic pharmacological properties such as, antioxidant, antibacterial, hepatoprotective, anti-inflammatory, cytotoxic, diuretic, antihypertensive and vasodilatory properties\(^6\). More than twenty phenolic compounds were isolated from this plant including lipophilic flavones, flavonol glycosides and caffeic acid derivatives such as rosmarinic acid and 2,3-dicaffeoyltartaric acid, were identified and quantified by HPLC\(^7\).

The methanolic extract of *orthosiphon stamineus* benth was evaluated for its nephroprotective activity using rat model. Gentamycin is an extensively used aminoglycoside antibiotic. It has been reported to produce nephrotoxicity even at normal therapeutic dose level. The drug was administered intra peritoneally at a dose of 80mg/kg weight for 9 days. Histopathological sections showed marked glomerular, peritubular and blood vessel congestion. These increased levels of serum creatinine, blood urea, urinary protein and extent of renal damage were decreased by the methanolic extract of *Orthosiphon stamineus* at both dose levels that is 100 and 200 mg/kg body weight in rats\(^2\).

**Strychnos potatorum**

*Strychnos potatorum* Linn commonly referred to as clearing root belongs to the family Loganiaceae\(^8\). According to Ayurveda, the seeds are acidic, astringent, lithuric and cure stranguary, urinary discharges, head ailments etc\(^9\). Roots cure all types of leucoderma whereas fruits are useful in eye diseases, thirst, poisoning and hallucinations. The ripe fruit is emetic, diaphoretic, aleuteric, cures inflammation, anemia, jaundice\(^10\). According to Unani system of medicine, seeds are bitter, astringent to bowels, goronhrea, colic etc\(^11\). Since kidney is involved in the clearance of toxins and xenobiotics, it may be more prone to attack by various challenges. A large number of these agents may cause damage to these organs by oxidative stress.

The ethanolic extract of *Strychnos potatorum* seeds was evaluated for its nephroprotective effect by using rat models. Hence, the study concludes that the seeds of *Strychnos potatorum* possess marked nephroprotective activity and could have a promising role in the treatment of acute renal injury induced by nephrotoxins, especially gentamicin\(^12\).

**Aerva javanica**

Aerva javanica Juss. ex Schult is medicinal plant belonging to the family Amaranthaceae\(^13\). Aerva javanica is reported as anthelmintic, diuretic, demulcent. It is used for the treatment of headache\(^14\). The decoction of the plant is administered to remove swellings, applied to acne like conditions of the face\(^15\).

The aqueous extracts of *Aerva javanica* roots were studied for the scientific evaluation of nephroprotective activity. Various parameters like body weight, blood urea, serum creatinine, serum protein, total protein, serum albumin, urine volume and pH, tissue protein, GSH and TBARS level were compared with controls on 16th day after treatment. The study concludes that cisplatin injury was evidenced by the elevated biochemical markers and histopathological features of acute tubular necrosis. The aqueous extract at the dose level of 400 mg/kg body weight was found to normalize the elevated biochemical markers and bring about a marked recovery in kidneys as evidenced by using microscopy\(^16\).

**Carica papaya**

Carica papaya Linn belonging to family caricaeae, is commonly known as papaya, Pawpaw, Melon tree\(^17\). *Carica papaya* is a rich source of phytoconstituents mainly carpeine, dehydrocarpaines, pseudocarpaines. It has various traditional remedies and pharmacological activities like antioxidant, wound healing, hepatoprotective, anti-inflammation, anti-bacterial, analgesic, heart tonic, anthelmintic and to treat ringworm, high blood pressure, stomachache, skin sores, fungal infections, cancer and prevents rheumatism, psoriasis\(^18\).

The aqueous seed extract of *Carica papaya* Linn. has been evaluated by carbon tetrachloride induced renal injury in wistar rats as a dose and time-dependent study. The study showed that *Carica papaya* Extract has nephroprotective effect on Carbon tetro chloride renal injured rats, an effect which could be mediated by any of the phytocomponents present in it in either antioxidant and or free radical scavenging mechanism\(^19\).

**Ficus religiosa**

*Ficus religiosa* (L.), commonly known as papal belonging to the family Moraceae. plants have been used in traditional Indian medicine for various range of ailments. Traditionally the bark is used as an antibacterial, antiprotozoal, antiviral, astringent, anti diarrhoeal. The extract significantly decreased the cisplatin used for skin diseases\(^20\). The leaves reported antivenom activity and regulates the menstrual cycle\(^21\). *Ficus religiosa* fruits contain flavonols namely kaempferol, quercetin, and myricetin\(^22\).

To evaluate the possible potential, nephroprotective and curative role of the methanolic extract of *Ficus religiosa* L. Latex was used against cisplatin (5mg/kg, i.p.) Induced Nephrotoxicity. The blood was collected from the retro orbital sinus of rats and determined for urea and creatinine levels in serum of each group after then rats were sacrificed for quantitative estimation of various enzymes and ATPas content in kidney tissue. A single dose of cisplatin induced shows the increased levels of urea & creatinine in serum and it was significantly recovered by 400mg/kg in curative and protective groups. The enzyme estimation in kidney tissue has found that increased malondialdehyde and decreased reduced glutathione (GSH). The results conclude that the study data confirmed nephrotoxicity induced by cisplatin due oxidative stress and methanolic extract of *Ficus religiosa* L. latex may have nephroprotective and curative activity\(^23\).

**Pedalium murex**

*Pedalium murex* is used as herbal medicine belongs to the family Pedaliaceae. Fruit extract of this plant contains many phytochemicals such as carbohydrates, flavonoids, alkaloids, glycosides, steroids, phenols, saponins, tannins and fixed oils & fats\(^24\). The effects of these bioactive components showed diverse pharmacological properties such as antioxidant, anti-diabetic, anti bacterial\(^25\), astringent, anti-inflammatory activity\(^26\) and nephroprotective property\(^27\).

The ethanolic extract of dried fruits of *Pedalium murex* Linn was evaluated for its nephroprotective activity. Nephrotoxicity was induced in Wistar rats by intraperitoneal administration of Cisplatin 5mg/kg. Effect of concurrent administration of *Pedalium murex* ethanolic extract at a dose of 250 mg/kg given by oral route was determined using serum creatinine and blood urea and change in body weight as indicators of kidney damage. Cystone was used as standard drug. The extract significantly decreased the cisplatin induced nephrotoxicity. The results showed that the ethanolic extract of dried fruits of *Pedalium murex* has an excellent nephroprotective activity as compared to cystone\(^28\).

**Vernonia cinerea**

*Vernonia cinerea* less belonging to family Asteracae\(^29\). Mainly it consists of 30%fatty oil, plant contains β-aminoylacetic acid, β-aminobenzoic acid, and its acetate, β-sitosterol, stigmasterol, α-spinasterol, kcl and also contains flavonoids, glycosides, tannins and carbohydrates\(^30\). The different parts of *vernonia cinerea* less has been possess Hypoglycemic and anti-diabetic activity\(^31\), anti-pyretic activity\(^32\), antibacterial activity\(^33\), diuretic and anti-diuretic activity\(^34\), anti-inflammatory activity\(^35\), free radicals and No
scavenging activity.\textsuperscript{15,16} The alcoholic extracts of aerial parts of vernonia cinerea has been examined for the effect of petroleum ether, ethyl acetate on cisplatin-induced nephrotoxicity at a dose of 6mg/kg, i.p. in albino rats. The alcoholic extract showed pronounced curative activity and the ethyl acetate extract has exhibited good prophylactic activity and petroleum ether extract showed moderate protection for both curative and prophylactic effects against cisplatin-induced toxicity.\textsuperscript{17}

CONCLUSION

From this study, it is clear that the medicinal plants play a prominent role against various diseases. A variety of medicinal plants and plants extracts have been reported for its significant nephroprotective activity in animal models. The nephroprotective activity is probably due to the presence of flavonoids in all the few medicinal plants. The results of this study indicate that extracts of leaves and plants of some medicinal plants have good potentials for use in kidney damage. The present review study give evidential explore mechanism of action of medicinal plants against experimentally induced nephrotoxicity. Hence, the review of the study is concluded that the herbal drug possesses nephroprotective activity and it has been proven by different animal models which gives many links to develop the future trials.

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