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A Review On Herble Drugs Used In The Treatment Of Nephroprotective Activity

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I. INTRODUCTION;

Man and his domesticated animals have since the time immemorial been largely dependent on plants for the essential for their existence by way of food , clothing, shelter and medicines etc,

1. Since disease, deCay and death always coexisted with life , the study of disease and thier treatment must have also been contemporaneous with the dawn of the human intellect. The primitive man must have used as therapeutical agents and remedial measures those things which he was able to procure most easily.there is no authentic record of medicines used by the primitive man. But the Rigveda which is the oldest book in the library of man supplies curious information on the subjects. In his work on plants and animal under domestication, Darwin says "From innumerable experiments made through dire necessity by savages of every land, with the result handed down by tradition, the nutritious, stimulating and medicinal properties of the most of unpromising plants were probably first discovered."

2. The doctrine of signatures would all account for the use of several plants as medicinal agents. The reason for the extensive use of vegetable drugs may be the fact that plants are everywhere at hand, their number is very great and their focus are distinct and peculiar and these are procured without trouble.

It is greatly to the credit of people of India, that they were acquainted with a far large no. of medicinal plants than the natives of any other country on the face of the earth.

3. Many indian fruits, grains and vegatables employed as useful dietary aricles forms achief factor in the cure of diseases, as well as preservation of health and good nutrition.

4. Herbs have always been the principle form of Medicine in india and they are becoming references to the kidney can be found in the old tastement. Before the time of the christ, Greek physicians prescribed botanical material to promote diuresis and employed blood letting and other means for removal of excess body fluids.

5.Hyppocrates (460-375BC) was skilled in microscopic detail of urine analysis. Artaeus of cappadoicia(30-90AD) and Galan (130-200AD) recognized kidney as the organ responsible for urin formation.

6.By the middle of 1800,the structural complexity of mammalian kidney was revealed &unraveled through improved optics &microscopy.if a few names had to be chosen among the pioneers, we could mention **Marcello Malpighi** and **Loreuzo Bellin** in **Italy**&**AntoineFerrein** in **France** for birth of renal anatomy.Sir William Bowman in England & Karl Ludwig in Germany for renal physiology & Richard Bright in London &PierraRayer in Paris for the diseases of kidney. Kidney is an important excretory organ in the human body. The function of kidney is not only to excrete metabolic waste products, but also to maintain the acid base balance, endocrine function like erythropoietin production.

7. Ancient literature has prescribed various herbs for the cure of kidney disease. The term "Pashanabeda" has been sited in literature to identify a group of plants, which have been extensively used in indigenous system of medicine to dissolve urinary calculi & stones. Eg: Aervalanata, Crataevanurvala, Pongamiaprinnata etc. Some other plants mentioned in literature include T.terrestris, O.sanctum, Zea mays etc.

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II. DEFINATION

nephroprotective. [nephro- + protective] Tending to preserve kidney function, esp. when the kidneys are exposed to unusual or unique stresses. nephroprotection (′shŏn)

III. 3. ANATOMY AND PHYSIOLOGY OF KIDNEY.

Paired kidneys are reddish bean shaped organs about 10-12cm long, 5-7cm wide, 3cm thick and has a mass of 135-150g.10 The kidneys lie on the posterior abdominal wall, one on each side of vertebral column, behind the peritoneum and below diaphragm. They extend from the level of 12th thorasic vertebrae to 3rd lumbar vertebrae.Near the centre of concave boarder is a deep vertical tissure called the renal hilum, through which the ureter emerges from the kidney along with blood vessels, lymphatic vessels & nerves.

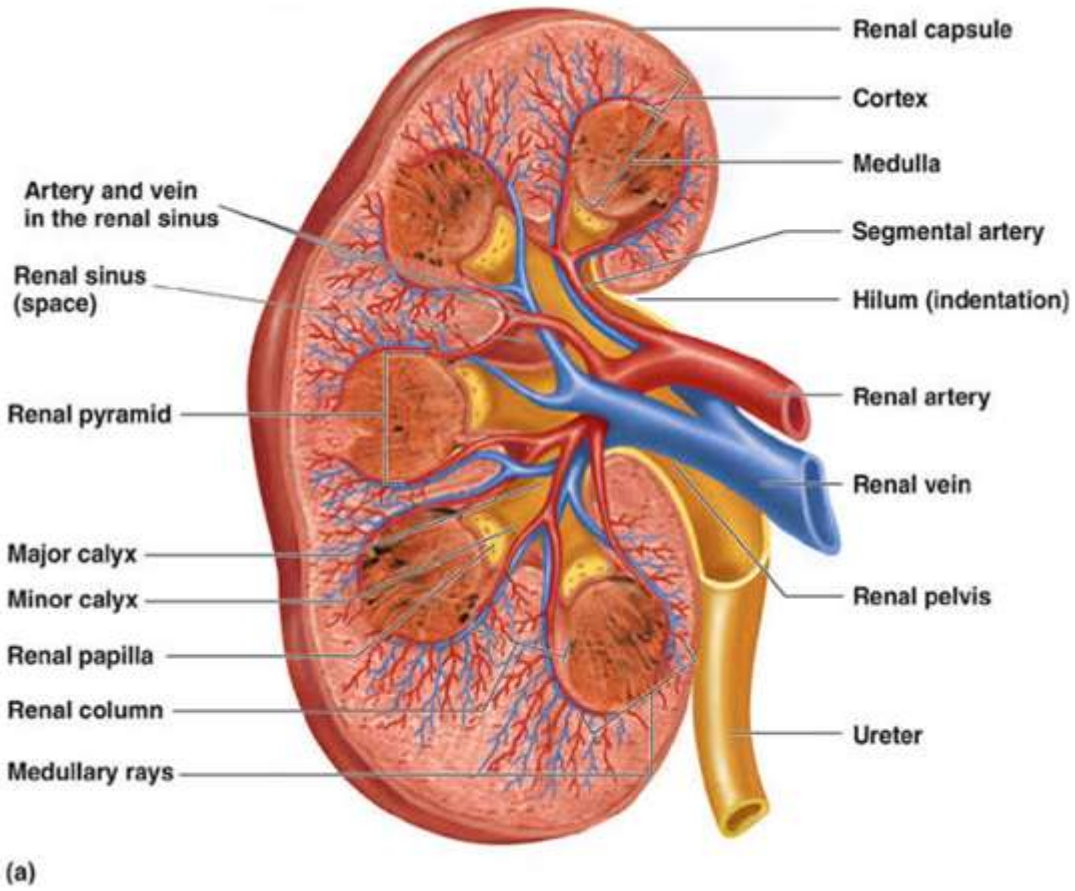

Fig. no.1 Kidney

The kidney consists of two distinct region, outer renal cortex & inner renal medulla. The urine collects to calyx and then to renal pelvis which empties into ureter. The functional unit of kidney is nephron and there are about 1million nephron in each kidney.10

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3.1 STRUCTURE OF NEPHRON

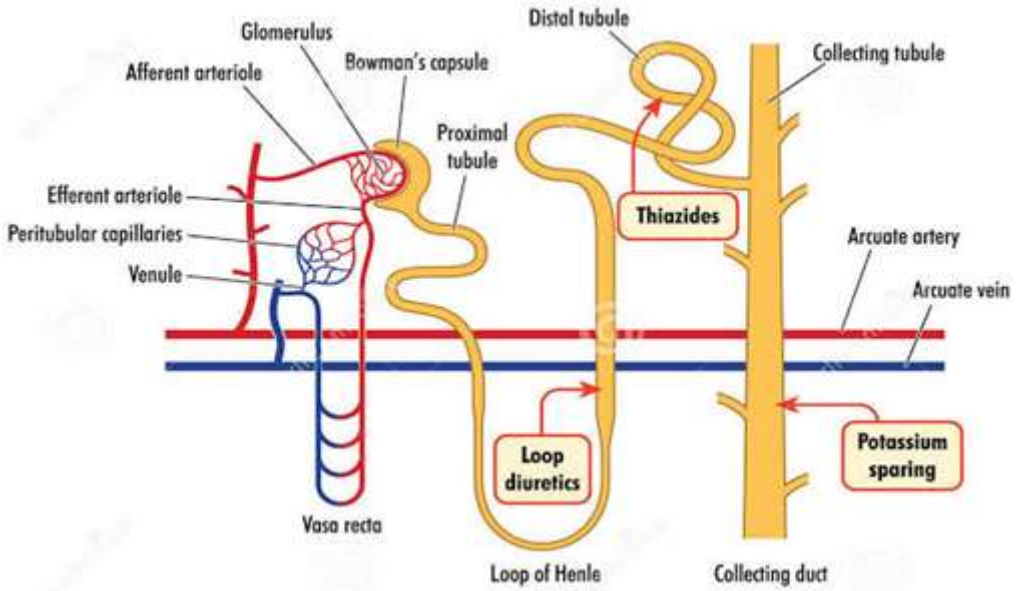

Fig. no.2 Nephron

It consists of a tubule closed at one end, and the other end opening into a collecting tubule
The closed end form Bowmann's capsule, which encloses the glomerulus.
The remaining parts of nephron is about 3cm long & consist of
Proximal convoluted tubule (PCT)
Henley's loop
Distal convoluted tubule
These nephrons are packed tightly to make up the kidney parenchyma.9

3.2Function of Kidney

The main function of kidney can be categorized as
-Formation of urine
-Water & electrolyte balance
-Production of hormones & enzymes

In the resting adult, kidney receives 1.2-1.3litres of blood/min. In an adult, the GFR averages 120ml/min. The collecting duct of kidney is an area of fine control of ultrafiltrate composition & volume, where final adjustment in electrolyte composition is made by the action of mineralocortioid& ADH. The hypertonicity of medullary interstitium plays an important role in concentrating the urine. The kidney not only excretes the metabolic substances, but also toxic agents from the body.6 Hence kidney becomes one of the important targets for the toxicity of agents more than other organs in body. Factors that make kidney particularly prone to actions of nephrotoxicity include,

High levels of toxins are delivered to the kidney's large blood supply.
The large surface area of renal tubular epithelium provide site for toxin interaction & uptake.
The availability of specific transport mechanisms that mediate cellular uptakes.
The normal concentrating mechanism of kidney can increase concentration of toxins.
The presence of the metabolic processes in the renal tubular cell, can release toxic components & induce damage.

IV. HISTOPATHOLOGYOFRENAL FAILURE

The term renal failure primarily denotes failure of the excretory function of kidney, leading to the retention of nitrogenous waste products of metabolism in blood. In addition, there is failure of regulation of fluid & electrolyte balance along with endocrine dysfunction.7 The renal failure is fundamentally categorized into acute renal failure & chronic renal failure.

4.1 ACUTE RENAL FAILURE

Acute renal failure is characterized by azotemia that progresses rapidly over several hours or days. It may or may not accompanied by oliguria

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& there is a sudden & reversible loss of renal function.

4.1.1 Histopathology of kidney with ARF

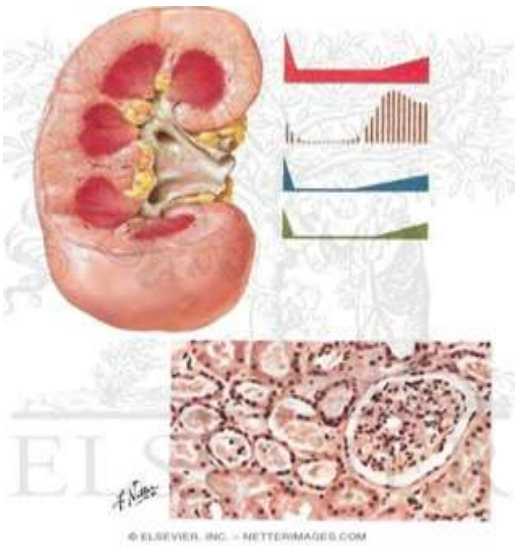

Fig. no.3Kidney with ARF

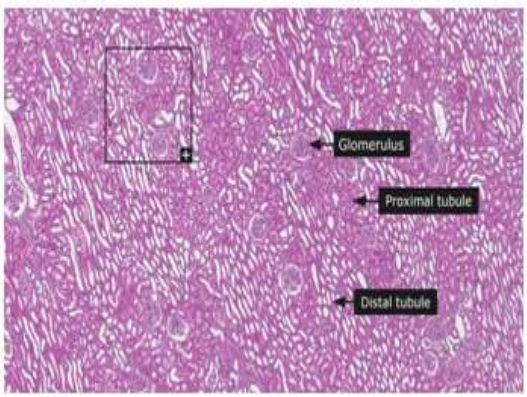

Fig.no.4 Histopathology of normal kidney

Early recognition of ARF is critical, because it is often asymptomatic. It is detected by measuring serum creatinine level & is more specific than measurement of blood urea nitrogen (BUN). There are many causes of ARF which could be,

4.1.2 Pre renal ARF

It is due to under perfusion of kidney. It accounted for 21% of ARF cases. It can be thought of as "a good kidney looking at a bad world." It is quickly reversible with appropriate therapy.

4.1.3 Post renal ARF

It is caused by obstruction of urinary tract. It accounted for 10% of cases.

4.1.4 Intrinsic ARF

It is due to disease in parenchyma. It accounted for 69% of cases. Among the renal causes of acute renal failure, acute tubular necrosis is more common accounting for 85% of incidence. ATN occurs due to either ischaemia or toxins. The toxins can be either exogenous or endogenous. The exogenous agents are radiocontrast agents, cyclosporins, antibiotics, chemotherapeutic agents, organic solvents, acetaminophen, & illegal abortifacients.

4.2 CHRONIC RENAL FAILURE.

It is a syndrome characterized by progressive & irreversible deterioration of renal due to slow destruction of renal parenchyma, eventually terminating in death when sufficient no. of nephrons have been damaged.8 Various causes are glomerulonephritis, diabetes mellitus, chronic pyelonephritis, hypertension.9antineoplastic agents like cyclophosphamide, viniristne, cisplatin etc.

V. NEPHROTOXIC AGENTS

Drugs, diagnostic agents & chemical are well known to be nephrotoxic. The following are some of the important nephrotoxic agents.11

A) Heavy metal

Mercury, arsenic, lead, bismuth

B) Antineoplastic agents

1. Alkylating agents

Cisplatin, cyclophosphamide

Nitrosoureas: Streptozotocin, Carmustine, Lomustine&Semustine

2. Antimetabolites

High dose Methotrexate, Cytosine Arabinose, high dose 6-thioguanine, 5-flurouracil

3. Antitumour antibiotics

Mitomycin, Mithramycin, Doxorubicin

4. Biologic agents

Recombinant leukocyte and interferon

C) Antimicrobial agents

Tetracycline, Acyclovir, Pentamidine, Sulphadiazine, Trimethoprin, Rifampicin

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AmphotericinB

D) Aminoglycosides
Gentamicin, Amikacin, Kanamycin, Streptomycin

E) Miscellaneous
Radiocontrast agents
Non-steroidal anti-inflammatory agents: Ibuprofen, Indomethacin, Aspirin etc.

VI. ANIMAL MODELS USED IN EXPERIMENTAL STUDIES

IN VIVO MODELS
GM treated albino rat
Cisplatin treated albino rats
Cisplatin treated rabbits
GM treated guinea pigs
Mercuric chloride treated mice
Ethylene glycol treated mice

IN VITRO MODELS
-Vero cells

VII. PHARMACEUTICAL EVALUATION OF SOME IMPORTANT PLANT.

7.1 EffectsAervalanata on GENTAMICIN & CISPLATIN models of acute rebal failure

Fig. no. 5 AervaLanata Plants

The ethanol extract of entire plant of Aervalanata was studied for its nephroprotective activity in cisplatin & gentamicin induced acute renal injury in albino rats of either sex. In the curative regimen, the extract at dose levels of 75,150 & 300mg/kg showed dose dependant reduction in the elevated blood urea and serum creatinine & normalized the histopathological changes in the cisplatin model. In the gentamicin model, the rats in the preventive regimen also showed good response to the ethanol extract at 300mg/kg. The findings suggest that the ethanol extract of Aervalanata 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 & gentamicin.

7.2 Protective effect of Pongamiapinnata flowers againts CISPLATIN & GENTAMICIN induced Nephrotoxicity rats.

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Fig.no.6 Pongamiapinnata flowers

When ethanolic extract of flowers of Pongamiapinnata (300 &600mg/kg) was administered orally in rats followed by cisplatin (5mg/kg ip), toxicity of cisplatin as measured by loss of body weight, elevated blood urea & serum creatinine declined significantly. Similarly in gentamicin (40mg/kg sc) induced renal injury, the extract 600mg/kg normalized the raised blood levels of urea & serum creatinine levels. Reversal of cisplatin & gentamicin renal cell damage was confirmed on histopathological examination. The results suggested that the protective effects is through antioxidant property of two flavonoids kaempferol and 3,5,6,7,8-penta methoxy flavone.

7.3 Salviae radix Extract prevwnts CISPLATINinducedACUTE RENAL Failure IN RABBITS

Fig.no.7 Salviae radix

The present study was carried out to determine if Salviae radix extract (SRE) exerts a beneficial effect against cisplatin induced renalfailure in rabbits. Rabbits were pretreated with SRE orally followed by cisplatin injection (5mg/kg ip). Cisplatin injection caused a reduction in GFR, which was accompanied by an increase in serum creatinine levels. The fractional Na+ excretion and lipid peroxidation were also increased. All these changes were prevented by SRE pretreatment.

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Cisplatin treatment invitro in renal cortical slices increased LDH release and lipid peroxidation, which were prevented by SRE and its effect may be attributed to its antioxidant action.

7.4 Protective effect of GYCYRRHIZIN ON GENTAMICIN Induced ACUTE RENAL failure IN RATS

Fig. no.8 Gycyrrhizin on Gentamicin

The effects of glycyrrhizin (200 mg/kg/day) on renal function in association with the regulation of aquaporin 2 water channel in rats with gentamicin (100 mg/kg/day)-induced acute renal failure was investigated. Polyuria in rats with gentamicin-induced acute renal failure was associated with down-regulation of renal aquaporin 2 in the inner and outer renal medulla, and cortex. Glycyrrhizin administration restored the expression of aquaporin 2 with paralleled changes in urine output. Changes in renal functional parameters, such as creatinine clearance, urinary osmolality, and solute-free reabsorption, accompanying acute renal failure were also partially restored after administration of glycyrrhizin. Histological changes in rats with gentamicin-induced acute renal failure were also abrogated by glycyrrhizin treatment. The above results suggest that glycyrrhizin treatment could ameliorate renal defects in rats with acute renal failure induced by gentamicin.

Fig. no.9Ginko biloba plant

7.5 Ginkgo biloba extract AMELIORATES GM induced NEPHROTOXICITY IN RATS

The effect of Ginkgo biloba (EGb), a plant extract with an antioxidant effect, has been studied on gentamicin-induced nephrotoxicity in male wistar rats. Ginkgo biloba extract (300 mg/kg BW) was administered orally concurrently with gentamicin (80 mg/kg BW). Estimations of urine creatinine, glucose, blood urea, serum creatinine, plasma and kidney tissue MDA were carried out after gentamicin treatment. Kidneys were examined using histological techniques.

Blood urea and serum creatinine were increased with gentamicin. Creatinine clearance was significantly decreased with gentamicin. Changes in blood urea, serum creatinine and creatinine clearance induced by gentamicin were significantly prevented by Ginkgo biloba extract. There was a rise in plasma and kidney tissue MDA with gentamicin, which were significantly reduced to normal with Ginkgo biloba extract. Histomorphology showed necrosis and desquamation of tubular epithelial cells in renal cortex with gentamicin, while it was normal with Ginkgo biloba extract. These data suggest that supplementation of Ginkgo biloba extract may be helpful to reduce gentamicin nephrotoxicity.

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7.6 Effect of Cassiaauriculata Root extract on CISPLATIN & GM induced renal injury

Fig.no. 10 Cassia auriculata

The ethanol extract of the roots of Cassia auriculata was studied for its nephroprotective activity in cisplatin- and gentamicin-induced renal injury in male albino rats. In the cisplatin model, the extract at doses of 300 and 600 mg/kg body wt. reduced elevated blood urea and serum creatinine and normalized the histopathological changes in the curative regimen. In the gentamicin model, the ethanol extract at a dose of 600 mg/kg body wt. reduced blood urea and serum creatinine effectively in both the curative and the preventive regimen. The extract had a marked nitric oxide free-radical-scavenging effect. The findings suggest that the probable mechanism of nephroprotection by C.auriculata against cisplatin- and gentamicin-induced renal injury could be due to its antioxidant and free-radical-scavenging property.

7.7 AGED GARLIC Extract Attenuates GM Induced Renal damage And Oxidative stress i RATS

Fig.no.11Aged Garlic

Aged garlic extract (AGE), an antioxidant, has a protective role in this experimental model of male Wistar rats were studied. AGE was given at a dose of (1.2 mL/kg/12 hours) followed by GM (70 mg/kg/12 hours). Nephrotoxicity was made evident by:

1) The increase in blood urea nitrogen and plasma creatinine

2) The decrease in plasma glutathione peroxidase (GPx) activity and the urinary increase in N-acetyl-beta-D-glucosaminidase activity and total protein

3) Necrosis of proximal tubular cells.

4) Increase in the renal levels of oxidative stress markers: nitrotyrosine and protein carbonyl groups and the decrease in manganese superoxide dismutase (Mn-SOD), GPx, and glutathione reductase (GR) activities.

These alterations were prevented or ameliorated by AGE treatment. Furthermore, AGE prevented the GM-induced The protective effect of AGE was associated with the decrease in the oxidative stress and the preservation of Mn-SOD, GPx, and GR activities in renal cortex. These data suggest that AGE may be a useful agent for the prevention of GM-nephrotoxicity.

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7.8 The effect of Nigella sativa OIL on GM NNephrotoxicity In RATS

Fig.no.12 Nigella sativa oil

In this work, tested whether oral treatment of rats with N. sativa oil (0.5, 1.0 or 2.0 ml/kg/day) would ameliorate nephrotoxicity of GM (80 mg/kg/day im) concomitantly with the oil. Nephrotoxicity was evaluated histopathologically and by measurement of concentrations of urea, creatinine and total antioxidant status (TAS) in plasma and reduced glutathione (GSH) and TAS in kidney cortex. The results indicated that GM treatment caused moderate proximal tubular damage, significantly increased the concentrations of creatinine and urea, and decreased that of TAS and GSH. Treatment with N. sativa oil produced a dose-dependent amelioration of the biochemical and histological indices of GM nephrotoxicity that was significant at the two higher doses used, and it increased GSH and TAS concentrations in renal cortex and enhanced growth. The results suggest that N. sativa may be useful in ameliorating signs of GM nephrotoxicity in rats.

7.9 Flavonoid of Drynariafortunei Protects Against ARF

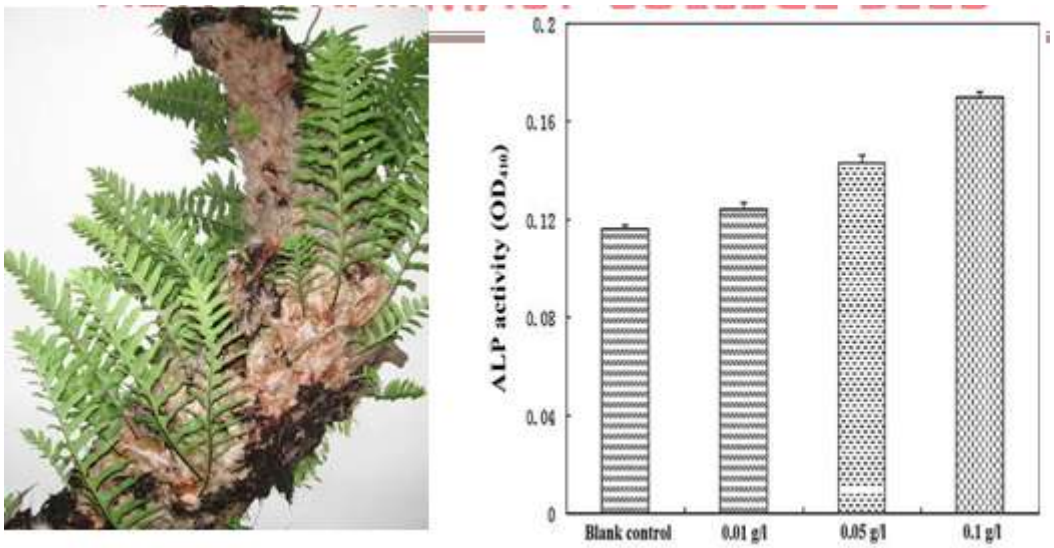

Fig.no.13Flavonoid of DrynariaFortunei

The flavonoid fraction (FF) from Drynariafortunei was investigated to determine its biological activity expression in three acute renal failure animal models Guinea pigs & mercuric chloride treated mice. Guinea pigs received 100 mg/kg of gentamicin & 10 mg/kg of FF. FF treatment prevented the GM toxicity, ie; the increase in BUN and creatinine levels. Mice were

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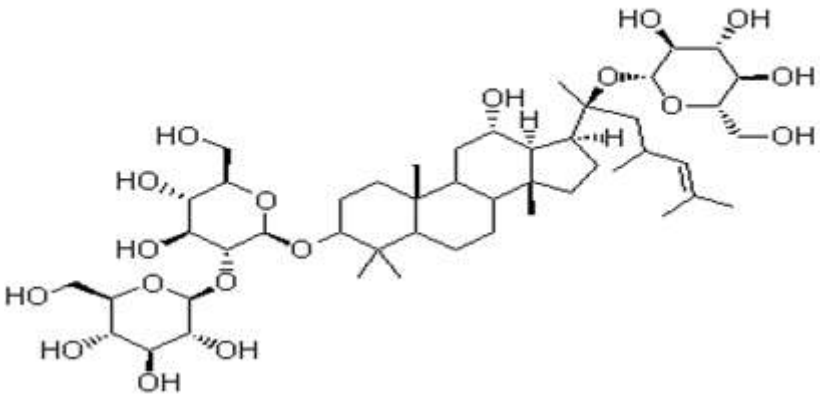
treated once with 6 mg/kg of mercuric chloride, followed by 10 mg/kg of FF. BUN and creatinine levels were found to be significantly higher on the mercuric chloride treatment and is ameliorated by FF treatment. In conclusion, the present study suggests that FF prevents nephrotoxicity, improves kidney function and promotes kidney primary epithelial tubular cell regeneration.

7.10 The roll of GINSENOID-Rd In CISPLATIN Induced ARF

Ginsenoside-Rd has been proved to decrease the severity of renal injury induced by cisplatin, in which proximal urinaferous tubules represent the main site of injury. When ginsenoside-Rd was given orally at a dose of 1 or 5 mg/kg body weight/day prior to cisplatin injection, the activities of the antioxidation enzymes superoxide dismutase and catalase were higher, while malondialdehyde levels in serum and renal tissue were lower in the treated rats than in the controls. The levels of urea nitrogen and creatinine in serum were decreased in rats given ginsenoside-Rd. Decreased urinary levels of glucose, sodium and potassium reflected a protective action against the renal dysfunction caused by cisplatin. In addition, it was demonstrated that ginsenoside-Rd affected cultured proximal tubule cells exposed to cisplatin.



7.11 Nephroprotective action of Tribulusterrestris AND Crataevanurvala In ALBINO RATS

Fig.no.14Tribulus terrestrial And Crataevanurvala

Nephrotoxic model was developed in male albino rats by administering GM. The aqueous extract of fruits of T.terrestris (65 or 130mg/kg) and C.nurvala (70 or 145mg/kg) after GM administration. Urine was examined for sugar, albumin, RBC & epithelial cells. Histopathological changes were also noted. The drug showed a dose dependant nephroprotective action against GM toxicity. The results indicate that the two

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indigenous plants would ameliorate renal effects in albino rats with acute renal failure induced by GM.

7.12 EFFECT OF Ocimum sanctum AQUEOUS LEAF EXTRACT ON GM INDUCED NEPHROTOXICITY IN RATS

Fig.no.15Ocimum sanctum

Nephrotoxicity was induced in rats by GM (180mg/kg/day ip). O.sanctum aqueous leaf extract (OS) was given orally at a dose of 100 mg/kg/day along with GM. Concurrent administration of OS significantly prevented rise in levels of serum creatinine, blood urea & plasma MDA which elevated by GM. It also significantly prevented the histological damage caused by GM. The results suggested that OS probably by virtue of its antioxidant property prevented GM induced nephrotoxicity in rats.

7.13 The effect of treatment with the medicinal plant Rhazya stricta On GM Induced NEPHROTOXICITY

Fig.no. 16Rhazya stricta leaves

Crude water extract of R. Stricta leaves (0.25, 0.5 and 1 g/Kg) was given orally to rats and thereafter, concomitantly with GM (80 mg/Kg/day). Nephrotoxicity was evaluated histopathologically and biochemically by measuring the concentrations of urea and creatinine in serum, reduced glutathione (GSH), lipid peroxidation and superoxide dismutase (SOD) activity in kidney cortex. The results suggested that a dose-related melioration in the indices of toxicity was noted when the two higher doses of the plant extract were given. The two higher doses, significantly and dose-dependently increased SOD activity and GSH concentration, and decreased that of lipid peroxides in the kidney cortex. These results suggest that R. stricta water extract may

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contain compounds that could potentially ameliorate GM nephrotoxicity in rats.

7.14 Renoprotective effect of GRAPE SEED Extract in ETHYLENE GLYCOL Include NEPHROTOXIC MICE

Grape seed extract in ethylene glycol (EG) induced nephrotoxicity in mice was studied for its nephroprotective activity. Mice received grape seed extract 100mg/kg BW was given after EG (2ml/kg BW po) administration. Grape seed extract in mice produced significant reduction of urinary LDH, blood urea, creatinine & dilated tubules lined by normal intact epithelium indicating recovery. The results suggest that the renoprotective effect of Vitis vinifera seed extract is due to improvement in antioxidant status.

7.15 Cytoprotective Role of Solanum nigrum Against GM Induced Kidney cell (VERO CELL) Damage invitro

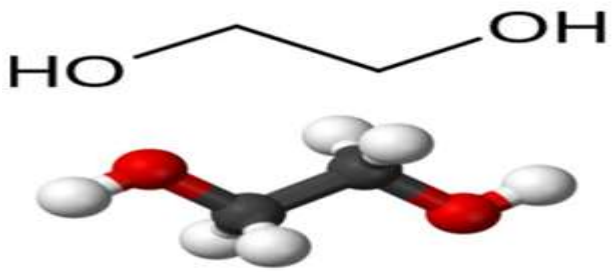

Fig.no.17Solanum nigrum

The 50% ethanol extract of the whole plant of Solanum nigrum was tested in vitro for its cytoprotection against gentamicin-induced toxicity on Vero cells. Cytotoxicity was significantly inhibited as assessed by the Trypan blue exclusion assay and mitochondrial dehydrogenase activity (MTT) assay. The test extract also exhibited significant hydroxyl radical scavenging potential, thus suggesting its probable mechanism of cytoprotection.

7.16 Evaluation of Nephroprotective Effect of Indian Medicinal Plants (IMPs) In Experimental GM Induced Nephrotoxicity32

The effect of administration of IMPs, Withaniasomnifera, Emblica officinalis, Glycyrrhiza glabra on BUN, serum creatinine, bodyweight MDA, renal histopathology were evaluated with administration of GM (150mg/kg/day) in female rats. Concurrent administration of IMPs & alpha lipoic acid prevented the rise in BUN, serum creatinine, kidney MDA to varying degrees. Thus IMPs show promise as protective agents against experimental nephrotoxicity.

VIII. DISCUSSION

This study was conducted to establish the nephroprotective activity of plants. Various models have been used to substantiate the nephroprotective activity of herbals. They were GM in albino rats, cisplatin in rabbits, mercuric chloride in mice, ethylene glycol in mice etc. These nephrotoxic agents caused nephropathy mainly due to their free radical generation in kidney tissues. And the kidney damage was indicated by changes in renal function parameters like creatinine, BUN, and the enzymes suchn as GPx, SOD and was also confirmed histopathologically. Above works certified that, by ameliorating all the allied effects, mainly due to antioxidant property the plants like A.lanata, P.pinnata, C.auriculata, S.radix, G.glabra, G.biloba, N.sativa, D.fortunei, T.terrestris, C.nurvala, O.sanctum, S.nigrum, V.vinifera have nephroprotective activity.

IX. CONCLUSION:

As we gone through various studies on treatment of kidney disorders, we can conclude that herbal plants play a unique role in medicine. There is no synthetic drug which relieve overall insufficiency of kidney. But indigenous plants possess tissue rejuvenator property which is anyway unavoidable. To Indians, who are brought

upon Indian food, soul & climate with Indian habits of life and environment, Indian drugs naturally suit better and safer than European constitution built upon their peculiar food, climate, habits and manner of life. This may perhaps be the reason why in numerous cases,where synthetic medicines fails, Indiginous system of medication succeed.

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