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**Therapeutic effectiveness of “*Kathaka Kadir*
Dhathri” decoction for the management of chronic
kidney disease – A pharmacological review**

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Abstract

Chronic kidney disease (CKD) is a progressive disease with no cure and high morbidity and mortality that occurs commonly in the general adult population, especially in people with diabetes and hypertension. It is debilitating, increasing in incidence worldwide, and a financial and social burden on health systems. Kidney failure, the final stage of CKD, is life-threatening if untreated with kidney replacement therapies. “*Kathaka Kadir* *Dhathri*” (KKD) is a decoction used for CKD patients in Ayurvedic medicine. However, there are no any scientific investigation or study have been done to identify the effect of this combinations. This review article focuses on effective alternative therapies to improve the prevention and treatment of CKD, using plants included into KKD to identify, rigorously test pre-clinically and clinically, and avoid any toxic outcomes to obtain optimal therapeutic benefit from medicinal plants. It may prove to be a filtering tool to researchers into complementary and alternative medicines to find out the current trends of using plant-based therapies for the treatment of kidney diseases, including CKD. Three mechanistic processes that are well-documented in CKD pathogenesis are inflammation, fibrosis, and oxidative stress. *In vitro* and *in vivo* experiments using plant-based therapies for pre-clinical research demonstrate some robust therapeutic benefits. According to this review, most of the plants included in to KKD have showed significant pharmacological effects on kidney and they are already known to ameliorate kidney dysfunction through antioxidant action, with subsequent benefits on inflammation and fibrosis.

Keywords: Chronic kidney disease, Decoction, *Kathaka Kadir* *Dhathri*,

Introduction

Chronic kidney disease (CKD) is an insidious, multifactorial, and slowly progressive disease, defined using altered kidney structure or dysfunction present for three months or more¹. It is recognized as having changed from a subspecialty issue to a global health concern². CKD is one of the major public health burdens in developed and developing countries. Although statistics differ from country to country, the prevalence of this disease has tended to increase from year to year recently³. CKD increases rapidly with age, with rates among those aged 75 and over, twice as high as for 65-74 year olds, and around 7 times as high as those aged 18-54 (42%, 21% and 6%, respectively). Prevalence is generally higher in lower socioeconomic groups (14% compared with 8% in higher socioeconomic groups)⁴. CKD is graded into six progressive stages (Stage 1, 2, 3a and 3b, 4, and 5) based on glomerular filtration rate (GFR). While not progressive in everyone, CKD progresses in many people to total and permanent kidney failure, or Stage 5 CKD, previously termed end-stage kidney disease (ESKD)⁵.

Sri Lanka has a population of approximately 20 million and agriculture is a major component of the economy. CKD is a growing problem in Sri Lanka. Hospital admissions due to diseases of the genitourinary system have nearly doubled during the period of the last two decades. For the year 2016, the eighth driving reason of hospital mortality was noted as diseases of the urinary tract under which the death because of CKD were reported (Annual Health Statistics, Sri Lanka, 2016). The increased incidence of CKD is mainly attributed to the rise in prevalence of type-2 diabetes mellitus and hypertension among the Sri Lankans⁶.

In recent years, herbal medicine has demonstrated its potential as an alternative therapy for treatment of numerous diseases, and remains an important source for the discovery of new drugs that has attracted greater attention recently. There has also been evidence showing that herbal medicine is

increasingly popular for promoting healthcare and preventing CKD. Interest in the health-benefiting properties of plant-based therapies is driven, in part, by the potential to prevent onset, or ameliorate progression, of certain diseases and reduce health care costs. The financial burden to health systems for CKD hospitalizations and treatments, worldwide, is immense and expected to increase. Complementary and alternative medicines have been used for CKD patients for many years, with reportedly disparate results for improving outcome⁷.

“*Kathaka Kadira Dhathri*” decoction (KKD) mentioned at Sri Lanka Ayurvedic pharmacopeia which is widely used by the Ayurvedic physicians to the CKD patient in our country for the management of chronic kidney disease. This decoction includes thirteen herbal plant material namely *Strychnos potatorum*, *Acacia chundra*, *Phyllanthus emblica*, *Sena auriculata*, *Coscinium fenestratum*, *Rubia cordifolia*, *Curcuma longa*, *Cissampelos pareira*, *Cyperus rotundus*, *Terminalia chebula*, *Hygrophila auriculata*, *Mangifera indica* and *Calamus zeylanicus*. However, there are no any scientific investigation or study have been done to identify the effect of this drug combinations. Therefore, it is significant to study of this plant combination with a scientific review. This review will describe the pathogenetic mechanisms of CKD that may be modulated by these therapies and emphasize and summarize information on plants included in to KKD using examples from pre-clinical and clinical studies.

Materials and Methods

PubMed, Medline, Google Scholar, Web of Science, for relevant articles published in English were used for this review of pre-clinical and clinical plant-based therapies for CKD and common causative diseases for CKD. Only English articles were included.

Results

Pathogenesis of chronic kidney disease

Aging, male gender, diabetes, hypertension and lifestyle are the most common causes for the

CKD⁸. Tubulointerstitial fibrosis and chronic inflammation, tubular atrophy, glomerulosclerosis and proteinuria are the Common pathogenesis mechanisms can be seen in CKD patients⁹. Chronic inflammation occurs through a series of biological signaling pathways involving the vasculature and the immune system, leading to the accumulation of inflammatory mediators in the tissue¹⁰. Fibrosis is a multifaceted cellular response primarily driven by various profibrotic and inflammatory cytokines such as transforming growth factor- β (TGF- β), tumor necrosis factor- α (TNF- α), platelet-derived growth factor, fibroblast growth factor-2, and some of the interleukins (ILs). Mechanistically, fibrosis and inflammation are exacerbated by oxidative stress¹¹. There is another key mechanism for kidney fibrosis is epithelial-mesenchymal transition (EMT) which is induced by various factors, including TGF- β , IL-1 β and angiotensin II¹².

Hypoxia and inflammation coexist and have interactive roles in CKD. Hypoxia promotes inflammation by increasing vascular permeability, a central process in the movement of inflammatory cells that facilitate the production of inflammatory mediators. In contrast, inflamed tissues are often severely hypoxic because of increased metabolic demands. Some important inflammatory signaling pathways in CKD involve mitogen-activated protein kinase (MAPK), the transcription factor nuclear factor kappa B (NF- κ B), p65 and some of the ILs. The MAPK family consists of extracellular signal-regulated kinase, Jun N-terminal kinase and p38 MAPK. NF- κ B is a key player in the production of pro-inflammatory cytokines and chemokines, such as TNF- α , IL-1 β , IL-6, chemokine motif ligand 2 and macrophage inflammatory protein-2¹³. Hypoxia is closely regulated by hypoxia-inducible factor (HIF). HIF plays a critical role in inflammation and fibrosis during CKD through determining gene transcription, activation of multiple signaling pathways and epigenetic regulation, and contributes to the pathogenesis of comorbidities of CKD such as anemia, and aberrant angiogenesis¹⁴. Tissue hypoxia also causes mitochondrial dysfunction and oxidative stress, which lead to the generation of reactive

oxygen species and reactive nitrogen species. Other leading causes of oxidative stress are proteinuria, uremic toxins, hyperglycemia and increased activity of the intra-renal angiotensin system.

Acute kidney injury involves tubular epithelial cell apoptosis and/or necrosis. If the injury is mild, an adaptive repair process can lead to complete recovery. In contrast, if severe or prolonged injury occurs, this may be followed by maladaptive repair that progresses to chronic inflammation, vascular rarefaction, nephron loss, fibrosis and finally progression to CKD. One of the main causes of acute kidney injury is ischemia-reperfusion. Ischemia also causes release of damage-associated molecular pattern molecules (DAMPs) by damaged cells. The DAMPs promote and exacerbate the inflammatory response¹⁵. Inflammatory mediators such as inducible nitric oxide synthase, and pro- and anti-apoptotic signaling pathways also act in the continuum of disease progression from acute kidney injury to CKD. Ultimately, tubulointerstitial fibrosis develops, thereby impairing local oxygenation. Thus, a time-dependent, combined and cumulative interplay of fibrosis, inflammation and tubular cell loss leads finally to progression to CKD.

Multiple factors and many signaling pathways are responsible for chronic kidney disease (CKD). Ischemia-reperfusion injury (IRI) is one of the key causes of acute kidney injury (AKI) and apoptosis. Hypoxia produces oxidative stress and reactive oxygen species. Tubular inflammation stimulates immune cells to produce and accumulate extracellular matrix (ECM), causing malfunctioning of kidney structure. Chronic tubular inflammation causes vascular rarefaction and nephron loss. The transcription factor nuclear factor- κ B (NF- κ B) stimulates proinflammatory cytokines. Reactive oxygen species, proinflammatory cytokines and kidney structure malfunction cause kidney fibrosis and, ultimately, CKD.

Phytochemicals and pharmacological actions of plant materials.

Medicinal plants are regarded as an acceptable, cheap, easily available and relatively safe source of many active compounds for pharmaceuticals. The beneficial effect of medicinal plants on kidney disease is often derived from their ability to boost the natural antioxidant defense mechanisms in the body. Different types of phytochemicals such as flavonoids, vitamins, resveratrol, anthocyanin, curcumin and phenolic acid are often found in the plant-based medicines and may act as antioxidant and give various pharmacological actions.

Strychnos potatorum seeds mainly containing with of alkaloids, flavonoids, glycosides, lignins, phenols, saponins, steroids, tannins¹⁶. The seeds of *S. potatorum* were studied using carrageenan-induced hind paw edema and cotton pellet granuloma models. Finally, were found to normalize the increased alkaline, acid phosphatases, and lipid peroxide levels indicating their membrane stabilization and free radical scavenging properties, and exhibited dose dependent anti-inflammatory activity in acute and subacute inflammatory models, and its effect was also comparable with the standard drug diclofenac sodium¹⁷. *S. potatorum* seeds possess hepatoprotective and antioxidant activities against CCl₄-induced acute hepatic injury. Hepatoprotective action is by reducing the serum marker enzymes like serum glutamate oxaloacetate transaminase (SGOT) and serum glutamate pyruvate transaminase (SGPT). They also reduced the elevated levels of alkaline phosphatase (ALP) and serum bilirubin. Histopathological studies confirmed the hepatoprotective activity of *S. potatorum* when compared with the CCl₄ treated control groups¹⁸. Diuretic activity of *S. potatorum* seeds was evaluated by using Wistar albino rats. The result indicated that the total urine volumes of the treated rats were evaluated nearly two and half fold then compared with the control group which was treated with saline. Excretion of cations (sodium and potassium ions) and anions (chloride ions) also increased significantly with respect to

the control group. The diuretic effect was comparable with that of the standard drug furosemide. The increase of cations in the urine on treatment with *S. potatorum* seed extract was dose-dependent¹⁹.

In the heartwood of *Acacia chundra* content catechin, Camphor, phytol, vitamin E acetate, 2-ethyl-3-methyl-1-butene, butyl phosphonic acid, ellagic acid, quercetin, rutin, and kaempferol are present in the leaves and the bark shows the presence of catechin (methanol extract), quercetin, kaempferol (ethyl acetate), kaempferol (methanol extract) ascorbic acid, riboflavin, thiamine, niacin, and carotenoids²⁰. The antidiabetic potential of ethanolic bark extract of *A. chundra*, was investigated, using α -glucosidase and α -amylase inhibition assays. Both assays demonstrated a good anti-diabetic impact; however, the α -amylase inhibition assay demonstrated the greatest inhibitory effect²¹.

Phyllanthus emblica are rich with multiple nutraceuticals which are iron, calcium, carotene, niacin, phosphorous, riboflavin, and thiamine among several others. Ascorbic acid (vitamin C) is the most abundant constituents of *P. emblica* fruit the seed contains a certain type of fixed oil such as linoleic, oleic, linolenic, palmitic, stearic, myristic acid; along with multiple essential oils and phosphatides. The bark is rich in both tannin and leucodelphinidin and the root is enriched with lupeol, ellagic acid, also a significant amount of tannin is found in the leaf and fruit as well. A specific amount of D-fructose, D-glucose, D-myo-inositol and free sugars are found from this fruit whereas, tannins, gallic acid and pyrogallol are the active principles of this fruit. Emblicanin A and Emblicanin B, pedunculagin and punigluconin are the major tannins reported from this plant. Other compounds isolated from this plants are, malic acid, arginine, aspartic acid, astragalin, β -carotene, β -sitosterol, chebulagic acid, chebulic acid, chebulagic acid, chebulinic acid, corelogic acid, corilagin, cysteine, emblicol, gibberellins, glutamic acid, glycine, histidine, isoleucine, kaempferol, leucodelphinidin, methionine, phenylalanine, phyllantidine, phyllemblic acid, quercetin, riboflavin, rutin,

thiamin, threonine, tryptophan, tyrosine, valine, zeatin, etc²². *P. emblica* significantly reversed the effects of ischemia-reperfusion on major antioxidant enzymes such as superoxide dismutase, catalase, glutathione peroxidase and lipid peroxidation activities. This study supports the claim that anti-oxidants of *Phyllanthus emblica* may act as cardioprotective agents²³. The study was carried out to investigate the anti-apoptosis effect of the extract from *Phyllanthus emblica* for the prevention of contrast-induced acute kidney injury (CI-AKI) and the findings suggested that pretreatment with *Phyllanthus emblica* extract provided the anti-apoptotic effect against CI-AKI in the rat model²⁴. The study showed that *Phyllanthus emblica* reduces nucleation of struvite crystals which is also called as infection stones occur in the urinary system of humans particularly the women community²⁵. Study relived that the *Phyllanthus emblica* reduced the elevated levels of serum creatinine and urea nitrogen; thiobarbituric acid-reactive substance levels of serum, renal homogenate in aged rat by the mechanism of Reduction of iNOS and COX-2 expression levels by inhibiting NF- κ B activation; reduction of elevated expression level of bax, aproapoptotic protein²⁶.

Phytochemical constituents of *Sena auriculata* are alkaloids, terpenoids, phenols and tannins, sugar saponins, flavonoids, quinines, steroids and proteins in commonly²⁷. Roots presence with anthraquinone glycosides and flavone glycoside. And also, some compounds like root bark are a chalcone 3,6, -dihydroxy-4-methoxychalcone, and two leucoanthocyanins. The methanol extract of *S. auriculata* roots have potent hepatoprotective activity against ethanol and anti-tubercular drug-induced hepatotoxicity²⁸. The study found that the ethyl acetate fraction of *S. auriculata* has more effective analgesic and anti-inflammatory activity compared to petroleum ether²⁹. The ethanolic root extract of *S. auriculata* has nephroprotective activity in gentamicin and cisplatin induced renal damage, because of the antioxidant property³⁰.

Coscinium fenestratum contain alkaloids berberine, dihydroberberine, 12, 13-dihydro-

8-oxo berberine, tetrahydroberberine, oxyberberine, and noroxy hydrastinine³¹. The antidiabetic potential of the alcoholic stem extract of *Coscinium fenestratum* was evaluated in the STZ- nicotinamide induced type 2 diabetic model and Significant reduction in fasting blood glucose along with serum triglyceride, and cholesterol levels were observed in the normal as well as in the treated diabetic animals³². Alcoholic extract of the stems of *C. fenestratum* was studied for its carbohydrate metabolism effect and antioxidant status in streptozotocin–nicotinamide induced type 2 diabetic rats. Oral administration of *C. fenestratum* stem extract in graded doses caused a significant increase in enzymatic antioxidants such as catalase, superoxide dismutase, glutathione synthetase, peroxidase, and glutathione peroxidase whereas a significant decrease was observed in the levels of gluconeogenic enzyme, glucose-6-phosphatase and alanine aminotransferase in treated diabetic rats as well as the serum creatinine and urea levels also declined significantly³³. Extract of *C. fenestratum* stem bark has hepato-renal protective effect in STZ-induced diabetic rats³⁴.

Mollugin is the active compound of *Rubia cordifolia* which has anti-inflammatory action including arthritis and uteritis³⁵. The hepatoprotective activity of an aqueous-methanol extract of *Rubia cordifolia* was investigated against acetaminophen and CCl₄ induced hepatic damage in mice and pretreatment of rats with plant extract lowered significantly the serum GOT and GPT levels³⁶. The hypoglycemic activity of the alcoholic extract of roots of *Rubia cordifolia* was studied in normal, glucose fed and alloxan-induced diabetic rats. The normal rats were treated with single dose of plant extract reduced the blood glucose level significantly and the rats pre-treated with plant extract improved oral glucose tolerance compared to glucose fed rats, exogenously injected insulin with plant extraction caused 26% potentiation of hypoglycemic effect at 6 h as compared with alone insulin treatment³⁷. The protective effect of *R. cordifolia* against lead nitrate-induced immune response impairment and kidney oxidative damage was studied using male Swiss albino mice. The result

showed the plant extract has a significant reversal of lead nitrate-induced toxicity on oxidative stress and immunological parameters such as lipid peroxidation and catalase and glutathione in renal tissues³⁸. The extract significantly decreased the cisplatin induced nephrotoxicity as inferred from the tissue antioxidant status in the drug administered mice. Remarkable change was observed in serum creatinine and urea levels and also Lipid peroxidation in the kidney and liver tissues was also considerably reduced in *Rubia cordifolia* extract treated mice³⁹.

Curcuma longa is rich in sources of polyphenolic curcuminoids and responsible for the yellow color of turmeric. Turmeric contains volatile Oil, its main content a variety of Monoterpenes, Sesquiterpenes and diterpenes. Other chemicals contain in turmeric are protein, fat, minerals, carbohydrates and moisture and Eugenol, Epiprocurcumenol; Eucalyptol; Eugenol; Feruloyl-p-coumaroyl-methane also present. Further chemical compounds are copper/zinc, campesterol, stigmasterol, beta-sitosterol, cholesterol, fatty acids and metallic elements potassium, sodium, magnesium, calcium, manganese, iron⁴⁰. Turmeric demonstrated both hepatoprotective and renoprotective characteristic similar to silymarin which is a flavonolignans extracted from the milk thistle *Silybum marianum*(L) mainly due to its antioxidant properties, as well as its ability to decrease the formation of pro-inflammatory cytokines⁴¹. The action of renal protection from diabetic nephropathy of turmeric had been studied in vitro using rats' mesangial cell line HBZY-1. Result showed restore advanced glycation end products (AGEs)-induced apoptosis to normal levels, and decrease the reactive oxygen species generation in mesangial cell by the action of curcumin and demethoxycurcumin⁴². Anti-Hepatotoxicity activity of curcumin and *C. longa* L. extract was studied in vivo using rats in acute or chronic CCl₄ induced hepatotoxicity and they observed recovering from CCl₄-induced hepatic toxicity in stress conditions⁴³. Several studies have proven that curcumin has strong antioxidant activity reducing the blood pressure elevation, vascular resistance and restored vascular

responsiveness also increasing the level of peroxides, superoxide dismutase activity, and total antioxidant concentration in liver homogenate⁴⁴.

Two novel tropoloisoquinoline alkaloids, named as Pareirubines A and B, had been isolated as antileukemic substances from *Cissampelos pareira* and also with skeleton alkaloids, grandirubrine and isoimerubrine. Pelosine was an amorphous white alkaloid indifferent from the plant. Cissamine and cycleanine have been reported from the roots also reported to contain l-curine. Menismine, pareirine and hayatinine are reported in root bark. Pareirubines A and B, alkaloids show effect as antileukemic agent⁴⁵. Significant anti-inflammatory activity of *C. pareira* was identified using 50% Ethanolic extract of roots in acute, subacute and chronic models of inflammation was assessed in rats by administration of dose (200, 400 mg/kg)⁴⁶. Hydrochloric root extraction of *C. Pareira* showed significant hepatoprotective action against CCl₄ induced Hepatotoxicity in rats and the result showed that elevated serum marker enzymes of AST, ALT, ALP and serum bilirubin were significantly reduced to near normal level in extract treated rats. Lipid peroxidation level was decreased significantly and in case of antioxidant enzymes SOD, catalase levels were increased⁴⁷. Anti-hyperglycemic potential of methanol root extraction of *C. pareira* Linn was evaluated by using in-vivo methods in streptozotocin- induced diabetic rats. Result showed the dose of 400mg/kg body weight was more effective with the highest glycemic changes⁴⁸. Nephro protective and antioxidant activity of *C. Pareira* had been found through the study of hydroalcoholic *Cissampelos pareira* whole plant extract using cisplatin induced nephrotoxic rats. *C. pareira* extract significantly increases the body weights, decreases the elevated urinary glucose levels in the urine, decreases the urea and creatinine levels in blood and increases the urinary creatinine levels in cisplatin induced nephrotoxic rats. In the in-vivo antioxidant study there was a dose-dependent decreasing and increasing of lipid peroxidation, glutathione levels in hydroalcoholic extract treated groups

respectively. The histopathological investigation also gave evidence to nephroprotective activity of *C. pareira*⁴⁹. The diuretic activity is observed using alcoholic extract of roots of *C. pareira* by Lipschitz method in albino rats. Single dose administration of standard Furosemide and alcoholic extract of roots of *C. pareira* significantly increased the urine output along with an increase in elimination of Sodium, Potassium, and Chloride ions. Alcoholic extract of roots of *C. pareira* 400 mg/Kg produced a comparable diuretic activity with standard Furosemide⁵⁰. Alcoholic extract of roots of *C. pareira* at (200 mg/kg and 400 mg/kg) doses showed curative effect in urolithiasis induced rats by preventing the formation, reducing number and disruption of calcium oxalate calculi formed in the kidneys⁵¹.

Cyperus rotundus presence of alkaloids, flavonoids, tannins, starch, glycosides, furochromones, monoterpenes, sesquiterpenes, sitosterol, fatty oil containing a neutral waxy substance, glycerol, linolenic, myristic and stearic acids⁵². Oral daily administration of 500 mg/kg of *C. rotundus* the extract (once a day for seven consecutive days) significantly lowered the blood glucose levels in rats with alloxan induced diabetes⁵³. Test the protective effects of *C. rotundus* tubers aquatic extraction the liver and kidney functions of male rats exposed to cadmium chloride poisoning (5 mg/kg body weight) for 30 days. Finally result showed that the *C. rotundus* tubers aquatic extract has protective effects and reduces the effects that cadmium chloride can cause in rats' liver and kidney functions through its antioxidant activity and removal of free radicals⁵⁴. Anti-platelet activity of ethanolic extract of *C. rotundus* was reported. Hence, *C. rotundus* extract and its active component (+)-nootkatone can be used for the prevention of platelet-linked cardiovascular diseases⁵⁵.

Terminalia chebula are a rich source of gallic acid. Major constituents are chebulagic acid and chebulic acid. Other compounds are tannic acid, gallic acid, ethyl gallate, ellagic acid, chebulinic acid, chebulanin, corilagin, terflavin A,

punicalagin, terchebulin, casuarinin, 2,4-chebulic- β -D-glucose, and glucose esterified with gallic acid to various degrees. Fruit; also contains tannic acid, gallic acid, resin etc. and some purgative principles of the nature of anthraquinone. Other classes of compounds identified in the fruits are shikimic acid and related compounds, sugars, triterpenoids, Arjunolic acid, and steroids. Also reported to have 18 amino acids and a small quantity of phosphoric, succinic, syringic and quinic acids. In *Terminalia chebula* 33% of the total phytoconstituents are hydrolysable tannins ((i.e., Gallo tannins and ellagitannins)) which are responsible for pharmacological activity that mainly effect on liver⁵⁶. The study showed, aqueous extract of the fruit of *Terminalia chebula* in wistar albino rats decreased the elevated levels of oxalate and phosphate in urine as well as kidney tissue homogenate as well as the extract supplementation also prevented the elevation of serum levels of Blood urea nitrogen, Creatine and Uric acid⁵⁷. The Fruit extraction of *T. chebula* has reduced the blood sugar level in normal and alloxan (120mg/kg) induced diabetic rats significantly⁵⁸. A fruit and seeds exhibited dose dependent reduction in blood glucose of streptozotocin induced diabetic rats both in short term and long-term study⁵⁹. A mixture of chebulic acid (CA) and its minor isomer, neo chebulic acid with a ratio of 2:1 isolated from ethanolic extract of *T. chebula* fruits showed strong hepatoprotective activity⁶⁰.

The whole plant of *Hygrophila auriculata* contains phytosterols, tannins, carbohydrates, flavonoids, terpenoids, and sterols. Oil from the seeds and reported the presence of uronic, palmitic, stearic, oleic, and linoleic acids. Apigenin-7-O-glucuronide and apigenin-7-o-glucoside were isolated from the flowers and lupeol, betulin, and stigmasterol were isolated from the plant. Alkaloids, steroids, tannins, proteins, flavonoids, carbohydrates, fats, and oils were isolated from the roots. Moreover, the leaves show the presence of alkaloids, carbohydrates, proteins, steroids, glycosides, flavonoids, tannins, phenolic compounds⁶¹. The alcoholic extract of *H. auriculata* (Schum.) at doses of 200 mg/kg showed a significant increase in the total urine

volume and concentrations of Na^+ , K^+ , and Cl^- in the urine in the male Wistar albino rats. This finding supports its traditional use as a diuretic⁶². The antihepatotoxic effect with treatment of methanolic extracts of the seeds of *H. auriculata* was studied on rat liver damage induced by a single dose of paracetamol and a significant hepatoprotective activity of the methanolic extract of the seeds was observed⁶³. Treatment of streptozotocin-induced diabetic rats with ethanolic extracts from the aerial parts of *H. auriculata* at doses 100 and 250 mg/kg for 3 weeks showed a significant reduction in the blood glucose levels, thiobarbituric acid reactive substances, and hydroperoxide in both liver and kidney. This also significantly increased the glutathione, glutathione peroxidase, glutathione S-transferase, and catalase. This study shows the antidiabetic activity along with potent antioxidant potential in diabetic conditions⁶⁴. Nephroprotective effect of methanolic extract of *H. auriculata* was studied using Cisplatin induced acute renal failure in rats. The results revealed that pretreatment with plant extract significantly reduced blood urea and serum creatinine levels elevated by Cisplatin administration. Thus, methanolic extract of *H. auriculata* significantly attenuated Cisplatin induced increase in malondialdehyde and decrease in reduced glutathione, catalase and superoxide dismutase and glutathione peroxidase activities in renal cortical homogenates. histopathological examination showed that plant extract markedly ameliorated Cisplatin-induced renal tubular necrosis⁶⁵. Antiuro lithiatic activity of *H. auriculata* was studied with ethylene glycol induced nephrolithiasis in male Wistar albino rats. The treatment with oral administration of

methanolic extraction of *H. auriculata* significantly reduced the elevated urinary oxalate, urinary calcium and serum uric acid with increase in reduced urinary magnesium hence ethylene glycol feeding also resulted in increased levels of calcium and oxalate in kidney which was decreased after the treatment with *H. auriculata*⁶⁶.

Mangifera indica are mainly contains polyphenolics, flavonoids and triterpenoids. It furthermore contains mangiferin (xanthone glycoside), isomangiferin, tannins and gallic acid⁶⁷. Seeds and Kernel contain Long-chain hydrocarbons and fatty acids include stearic acid, eicosanoid acid, linoleic, linolenic, oleic acid, arachidonic acid, and palmitic acid. Sterols include stigmasterol, sitosterol, and campesterol. Triterpenes and triterpenoids include α -pinene, β -pinene, myrcene, and limonene. Polyphenols and phenolic acids include ascorbic acid, mangiferin, quercetin, and gallic acid⁶⁸. *M. indica* has prophylactic impact against kidney injury by upgrading the kidney work by means of diminishing serum creatinine, urea and uric acid. Treatment of rats with 500 and 1000mg/kg methanolic seed kernel extract altogether expanded the level of reduced glutathione and superoxide dismutase activity while decreased the level of aggregate malondialdehyde and glutathione-S-transferase⁶⁹. Aqueous extract of *Mangifera indica* bark and leaves has showed diuretic activity by increasing Na^+/K^+ ratio in rats⁷⁰. *M. indica* fruit peel powder showed a significant reduction of blood glucose level and diabetes associated complications in rats. Similar results have been obtained in a study carried with a flour prepared from mango fruit pulp⁷¹.

Table 1: Phytochemicals and Pharmacological actions of the plants included into *KathakaKadiraDhathri* formula.

No	Scientific name	Part of used	Phytochemicals	Pharmacological actions
1	<i>Strychnos potatorum</i>	Seeds	Alkaloids, Flavonoid, Glycosides, Lignins, Phenols, Saponins, Steroids, Tannins	Anti-diabetic Anti-inflammatory Antioxidant Diuretic
2	<i>Acacia chundra</i>	Wood and bark	Catechin, Quercetin, Kampeferol, Ascorbic acid, Riboflavin, Thiamine, Niacin, Carotenoids	Anti-diabetic Antioxidant
3	<i>Phyllanthus emblica</i>	Dry fruits	Tannins, Gallic acid, Pyrogallol, fixed oils, carotene, Niacin, Riboflavin, Thiamine, Ascorbic acid	Anti-atherosclerotic action Antioxidant Cardio protective action Anti-apoptosis action Anti-diabetic
4	<i>Sena auriculata</i>	Roots	Alkaloids, Glycosides, Saponins, Phenols, Tannins, Terpenoids, Triterpenes, Anthroquinone, Sitosterols	Anti-diabetic Anti-hyperlipidemic Hepatoprotective action Anti-inflammatory Nephroprotective action
5	<i>Coscinium fenestratum</i>	Stems	Alkaloid (berberine), Saponin	Anti-diabetic Antioxidant Hepato-renal protective action
6	<i>Rubia cordifolia</i>	Stems	Mollugin Quinones Bicyclic hexapeptides compounds	Antitumor activity Hepatoprotective action Anti-diabetic Nephroprotective action Antioxidant
7	<i>Curcuma longa</i>	Rhizomes	Polyphenolic Curcuminoids (Curcumin i, ii, iii), Volatile oil, sterols, Fatty acids, Metallic elements	Hepatoprotective action Antioxidant Anti-hyperglycemic Antibacterial Antimicrobial Nephroprotective action
8	<i>Cissampelos pareira</i>	Roots	Alkaloids (Pareirubrine A and B), Curine, Menismin, Pareirine, Hayatinine	Anti-inflammatory Anti-leukemic Hepatoprotective action Anti-hyperglycemic Nephroprotective action Antioxidant Diuretic activity

9	<i>Cyperus rotundus</i>	Rhizomes	Alkaloids, Flavonoids, Tannins, Starch, Glycosides, Sitosterol, Furochromones, Monoterpenes, Triterpens, Sesquiterpenes.	Anti-diabetes Anti-hyperlipidemic Antioxidant Anti-platelet activity
10	<i>Terminal chebula</i>	Dry fruits	Chebolic acid, Tannic acid, Gallic acid, Shikimic acid, Triterpenoids, Arjunolic acid, Steroids, Tannins	Anti-hyperlipidemic Nephroprotective action Anti-diabetes Anti-spasmodic Antioxidant
11	<i>Hygrophila auriculata</i>	Whole plant	Phytosterols, Tannins, Flavonoids, Terpenoids, Sterols, Alkaloids, Steroids, Glycoside, Phenolic compounds	Hematopoietic activity Anti-hepatotoxic activity Anti-diabetic activity Nephroprotective action Antiurolithiatic activity
12	<i>Mangifera indica</i>	Seeds and kernel	Long-chain hydrocarbons, Eicosanoid acid, Linoleic acid, Linolenic acid, Oleic acid, Arachidonic acid, Palmitic acid, Sterols, Polyphenols, Phenolic acid	Nephroprotective action Anti-diabetes Diuretic activity
13	<i>Calamus zeylanicus</i>	Trunk		

There is no more literature to find out regarding *C. zeylanicus* which is endemic to Sri Lanka due to any scientific studies that have not been done.

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Conclusion

There is no doubt that each and every medicinal plants included into KKD decoction show significant effect on CKD. Clinical and pre-clinical trials of plant extracts demonstrate benefit for CKD. Therefore, the aim of recent research is to identify, rigorously test pre-clinically and clinically, and avoid toxic outcomes in order to obtain optimal therapeutic benefit from KKD. This review may prove to be a filtering tool to researchers into complementary and alternative medicines to find out the current trends of using medicinal plants and plant extracts for the treatment of kidney diseases, including CKD.

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