Abstract
Nephropathy develops progressively in diabetic patients such that it becomes a major contributor risk factor for death from cardiovascular complications. It is a common and disabling complication of diabetes mellitus with no effective therapy. Acute uric acid nephropathy (AUAN, also acute urate nephropathy) is a rapidly worsening (decreasing) kidney function (renal insufficiency) that is caused by high levels of uric acid in the urine (hyperuricosuria). Chronic Kidney Disease: Chronic renal failure (CRF) is the gradual reduction of kidney function that may lead to permanent kidney failure, or end-stage. Diabetic nephropathy (nephropatia diabetica), also known as Kimmelstiel-Wilson syndrome, or nodular diabetic glomerulosclerosis, and intercapillary glomerulonephritis, is a progressive kidney disease caused by angioopathy of capillaries in the kidney glomeruli. It is characterized by nephotic syndrome and diffuse glomerulosclerosis. It is due to longstanding diabetes mellitus, and is a prime indication for dialysis in many Western countries. Exploration of traditional medicine is a mysteriously interesting yet, scientifically significant and economically important task of ethno botanists. The people of India are well acquainted with a large number of indigenous medicinal plants than the natives of any other countries. Herbs are the principal form of medicine in India and they are becoming popular throughout the world.

Introduction
Nephropathy develops progressively in diabetic patients such that it becomes a major contributor risk factor for death from cardiovascular complications. It is a common and disabling complication of diabetes mellitus with no effective therapy. The progression of nephropathy in diabetes from the reference time point of diagnosis of diabetes is Incipient nephropathy (hyper filtration and persistent microalbuminuria) - after ca. 18 years. Overt nephropathy (persistent proteinuria and decline in glomerular filtration rate (GFR) - after ca. 29 years. End stage renal disease (ESRD) requiring renal replacement therapy - after ca. 39 years, and Diabetes is a common cause (30%) of chronic renal failure and is likely to be of increasing importance in the future as diabetes incidence increases - especially with early onset. Glycemic control is a risk factor for the development of incipient nephropathy and progression from incipient to overt nephropathy in type 1 and type 2 diabetes. Health care services have now prioritized the decreasing the risk of nephropathy by tight control of blood glucose and blood pressure and aggressive intervention to counter dyslipidaemia - as reflected in position statement reports. This is not always achievable - particularly because of limitations of current drug therapy. Hence, diabetic nephropathy is still a significant clinical problem that needs new effective therapy urgently and preferably therapies working independent of existing strategies. One such therapy emerging is high dose thiamine supplements. The link of hyperglycemia to renal dysfunction and development of nephropathy. High plasma glucose concentration leads to high cytosolic glucose concentration in renal endothelial cells and pericytes with consequent biochemical dysfunction: activation of protein kinase C<sub>β</sub>, hexosamine and polyol pathways; metabolic pseudo hypoxia, mitochondrial dysfunction and oxidative stress, and accumulation of advanced glycation end products (AGEs)<sup>[1,2]</sup>. Biochemical dysfunction in hyperglycemia linked to diabetic nephropathy<sup>[3]</sup> increased concentrations of trios phosphate glycolytic intermediates, glyceraldehyde-3-phosphate (GA3P) and dihydroxyacetonephosphate (DHAP), is the trigger for these processes. A pharmacological strategy that counters trios phosphate accumulation in hyperglycemia would suppress multiple pathogenic pathways and prevent the development of diabetic nephropathy. Activation of the reductive pentose phosphate pathway (PPP) by high dose thiamine therapy may achieve this by increasing transketolase (TK) activity and stimulating the conversion of GA3P and fructose-6-phosphate (F6P) to ribose-5-phosphate (RSP) – Figure 2. Reversal of biochemical dysfunction in hyperglycemia by high dose thiamine We first established this response in cell culture and then investigated the effect of high dose thiamine and Benfotiamine therapy on the development of incipient nephropathy in the streptozotocin (STZ)-induced diabetic rat model of diabetes with moderate insulin therapy. Incipient nephropathy developed over a 24-week period in the STZ diabetic rats, as judged by hyper filtration and microalbuminuria, and both high dose thiamine and Benfotiamine therapy prevented it.<sup>[1]</sup> Diabetic nephropathy (nephropatia diabetica), also known as Kimmelstiel-Wilson syndrome, or nodular diabetic glomerulosclerosis and intercapillary glomerulonephritis, is a progressive kidney disease caused by angioopathy of capillaries in the kidney glomeruli. It is characterized by nephrotic syndrome and diffuse glomerulosclerosis. It is due to longstanding diabetes mellitus, and is a prime indication for dialysis in many Western countries.<sup>[1,2]</sup> Epidemiology
The syndrome can be seen in patients with chronic diabetes (usually less than 15 years after onset) after about 5 years in type 1 diabetes. Clinical nephropathy secondary to glomerular disease usually manifests 15–25 years after diagnosis of diabetes and affects 25-35% of patients under the age of 30 years. It is the leading cause of premature death in young diabetic patients.(between 50 and 70 years old). The disease is progressive and may cause death two or
three years after the initial lesions, and is more frequent in men. Diabetic nephropathy is the most common cause of chronic kidney failure and end-stage kidney disease in the United States. People with both type 1 and type 2 diabetes are at risk. The risk is higher if blood-glucose levels are poorly controlled. Furthermore, once nephropathy develops, the greatest rate of progression is seen in patients with poor control of their blood pressure. Also people with high cholesterol level in their blood have much more risk than others.[2]

Pathophysiology

The earliest detectable change in the course of diabetic nephropathy is a thickening in the glomerulus. At this stage, the kidney may leak more serum albumin (plasma protein) than normal in the urine (albuminuria), and this can be detected by sensitive medical tests for albumin. This stage is called "microalbuminuria". As diabetic nephropathy progresses, increasing numbers of glomeruli are destroyed by progressive nodular glomerulosclerosis. Consequently, urine albumin increases to the point that it may be detected by ordinary urinalysis techniques. At this stage, a kidney biopsy generally shows diabetic nephropathy. The Armanni-Ebstein change (or Armanni-Ebstein cell) consists of deposits of glycogen in the tubular epithelial cells (pars straight of proximal convoluted tubule and loop of Henle). Because most diabetics are treated before this stage, it is very rare to see it at the present time. It appears in decompensated diabetics with glycosuria; it is a reversible alteration without functional.[4]

Complications

Possible complications include:

- hypoglycemia (from decreased excretion of insulin) (insulin isn’t secreted by the kidneys) (decreased excretion of insulin would cause hyperglycemia)
- rapidly progressing chronic kidney failure
- end-stage kidney disease
- hyperkalemia
- severe hypertension
- complications of haemodialysis
- complications of kidney transplant
- coexistence of other diabetes complications
- peritonitis (if peritoneal dialysis used)
- increased infections[3,4]

Kidney failure provoked by glomerulosclerosis leads to fluid filtration deficits and other disorders of kidney function. There is an increase in blood pressure (hypertension) and fluid retention in the body plus a reduced plasma oncotic pressure causes edema. Other complications may be arteriosclerosis of the renal artery and proteinuria.

Throughout its early course, diabetic nephropathy has no symptoms. They develop in late stages and may be a result of excretion of high amounts of protein in the urine or due to renal failure- edema: swelling, usually around the eyes in the mornings; later, general body swelling may result, such as swelling of the legs, blurry appearance or excessive frothing of the urine (caused by the proteinuria), unintentional weight gain [from fluid accumulation], anorexia (poor appetite) nausea and vomiting, malaise (general ill feeling), fatigue, headache, frequent hiccups, generalized itching

Types of nephropathy

Obstructive nephropathy:

Obstructive nephropathy: A condition which is characterized by obstructive of the urinary tract resulting in kidney disease and dysfunction.

Symptoms of Obstructive nephropathy

The list of signs and symptoms mentioned in various sources for Obstructive nephropathy includes: Urinary incontinence, Urinary hesitancy, Increased or decreased urine output, Abnormal urine flow, Dribbling after urination, Weak urinary stream, Increased urinary urgency, Increased nighttime urination, Sensation of incomplete bladder emptying, Burning urination, Stinging urination, Blood in urine, Abnormal urine color, Kidney damage, Asymptomatic in some cases, Increased blood pressure, Abdominal pain, Back pain, Flank pain, Nausea - acute cases, Vomiting - acute cases, Loss of appetite - acute cases.[6]

Acute uric acid nephropathy

Acute uric acid nephropathy (AUA, also acute urate nephropathy) is a rapidly worsening (decreasing) kidney function (renal insufficiency) that is caused by high levels of uric acid in the urine (hyperuricosuria).[7,8] Acute uric acid nephropathy is usually seen as part of the tumor lysis syndrome in patients undergoing chemotherapy or radiation therapy for the treatment of malignancies with rapid cell turnover, such as leukemia and lymphoma. It may also occur in these patients before treatment is begun, due to spontaneous tumor cell lysis (high incidence in individuals of all ages, but lymphoma) Acute uric acid nephropathy can also be caused by an acute attack of gout.[7,8]

Acute renal failure - is a syndrome characterized by rapid onset of renal dysfunction, chiefly oliguria or anuria, and sudden increase in metabolic waste-product in the blood and secondly

- Chronic kidney disease

Chronic Kidney Disease: Chronic renal failure (CRF) is the gradual reduction of kidney function that may lead to permanent kidney failure, or end-stage ... more about Chronic Kidney Disease.[9,10] Chronic Kidney Disease: Long-term and generally irreversible disease of the kidneys due to infection, obstruction, congenital diseases or generalised diseases causing failure of the kidneys’ normal functions. [9,10] Symptons of Chronic Kidney Disease

- Fluid retention, Puffiness in feet, Legs and face, Cough, Breathlessness, Night-time cough, Cough when lying down, Chest pain, “sighing” breaths, Palpitations, Infrequent urination, More frequent urination, Night time urination, Frotthy urine, Blood in urine, High blood pressure, Dizziness, Light-headedness, Fatigue, Nausea, Vomiting, Itchy skin, Yellow skin pigmentation, Pale skin, Loss of appetite, Muscle loss/wasting, Muscle weakness, Clumsiness, Sensory loss in hands and feet, Headache, Loss of concentration, Drowsiness, Coma, Bone weakness, Frequent bone fractures, Bone fractures from minimal trauma, Bone pain, Impotence, Infertility, Heavy periods[9,10]

- Characteristics of IGA nephropathy

IgA nephropathy – also known as Berger’s disease in the literature – is the most common form of primary glomerulonephritides. It occurs in individuals of all ages, but is most common in young adults. Diagnosis requires immunofluorescent examination of kidney tissue. Although the progression is slow and the disease was thought to have a benign course, on the basis of long term follow up 30–40% of patient’s needs dialysis 20 years after the diagnosis. The complex examination of potential factors playing a role in the progression of the disease is possible with analyzing the data obtained during the long term observation of IgA patients. The importance of the examinations shows the fact, the that the cause and the exact pathomechanism of the disease is not yet known. It is especially important in the light of the
fact, that the disease's prevalence is high and 30–40% of the patients need dialysis after 20–25 years. Follow up.[11]

Chinese herbs nephropathy
In 1993, a rapidly progressive interstitial renal fibrosis was reported in a group of young women who ingested pills containing Chinese herbs while attending a slimming clinic in Brussels. Subsequent investigation found that one of the prescribed Chinese herbs had been inadvertently replaced by other Chinese herbs containing the nephrotoxin aristolochic acid. Since the original description, there have been several reports worldwide of nephropathy associated with the inadvertent adulteration of herbal remedies taken for a variety of conditions with herbs containing aristolochic acid. Interestingly, the pathologic findings in Chinese herbs nephropathy bear a striking resemblance to those observed in the ill-defined Balkan nephropathy, suggesting that the latter might represent a chronic exposure to aristolochic acids.

There is an on-going concern regarding the use of Chinese herbs based upon a number of incidents of nephropathy involving renal failure due to severe interstitial fibrosis, apparently related to the ingestion of herbs from the Aristolochia genus. Carcinogenesis may have also played a role in the pathology. There are both facts and conjecture surrounding this issue, which has come to be known as "Chinese Herb Nephropathy" (CHN).

The original incident was related to a combination diet therapy used in a Belgian weight loss clinic that involved drugs, including serotonin, and herbs not normally combined for this purpose. While the herbs chosen for the therapy were Stephania tetrandra and Magnolia officinalis, neither of these herbs was implicated in the subsequent CHN.[12]

Treatment of nephropathy
Treatment is focused on preventing deposition of uric acid within the urinary system by increasing urine volume with potent diuretics such as furosemide. Raising the urinary pH to a level higher than 7 (alkalinization) is often difficult to attain, although sodium bicarbonate and/or acetazolamide are sometimes used in an attempt to increase uric acid solubility. [28]

The list of treatments mentioned in various sources for Chronic Kidney Disease includes the following list. Always seek professional medical advice about any treatment or change in treatment plans.

- Treatment of underlying cause - e.g., hypertension, diabetes, autoimmune diseases
- Diuretics
- Fluid restriction
- Dietary restrictions of potassium and phosphate containing foods
- Treatment of renal bone disease - Vitamin D supplements
- Treatment of acid-base imbalances - sodium bicarbonate
- Treatment of anemia - erythropoietin
- Renal dialysis - haemodialysis or peritoneal dialysis
- Renal transplant
- Regular monitoring of blood pressure, urea, creatinine and glomerular flow rate
- Vitamin D - possibly used for treatment of vitamin D deficiency
- Treatment of chronic renal failure is dependent upon the underlying cause of the kidney damage, the severity of the kidney damage, the age of the patient, and any other health problems that the patient might have. Treatments include:
  - Avoidance of medications that may contribute to further renal damage - NSAID's, IV contrast
  - Smoking cessation - people who continue to smoke progress to end stage renal failure sooner
  - Close management of high blood pressure, whether it is a cause or result of renal disease
  - Use of ACE inhibitors - in patients both with and without proteinuria has been shown to slow the progression of renal failure
  - Dietary management
    - Protein restriction - may have a role in slowing the progress of renal failure, and is important to manage uremic symptoms in advanced failure, but needs to be managed carefully to avoid malnutrition
    - Salt and water restriction as necessary to manage fluid overload
    - Phosphate restriction
    - Potassium restriction as necessary
  - Erythropoietin agonists - for management of anaemia associated with chronic renal failure
  - Phosphate binders - for management of hyperphosphatemia in chronic renal failure
  - Calcitriol and other Vitamin D supplements - for hypercalcemia and hyperparathyroidism associated with chronic renal failure
  - Sodium bicarbonate - for acid-base disturbance
  - Diuretics - may be used in situation of volume overload
  - Renal dialysis - used when there are manifestations of uremia and the GFR is < 10mL/min
  - Haemodialysis
  - Peritoneal dialysis
  - Renal transplantation[12,13]

The list of treatments mentioned in various sources for Nephropathy includes the following list. Always seek professional medical advice about any treatment or change in treatment plans.

- Treatment depends on the underlying cause of nephropathy
- Treatment of nephropathy is dependent upon the underlying cause of the kidney damage, the severity of the kidney damage, the age of the patient, and any other health problems that the patient might have. Treatments include:
  - Treatment of underlying cause - e.g., hypertension, diabetes, autoimmune diseases. This is very important in order to limit or slow the progression of renal disease
  - Avoidance of medications that may contribute to further renal damage - NSAID's, IV contrast
  - Smoking cessation - people who continue to smoke progress to end stage renal failure sooner
  - Close management of high blood pressure, whether it is a cause or result of renal disease
  - Use of ACE inhibitors - in patients both with and without proteinuria has been shown to slow the progression of renal failure
  - Dietary management
    - Protein restriction - may have a role in slowing the progress of renal failure, and is important to manage uremic symptoms in advanced failure, but needs to be managed carefully to avoid malnutrition
    - Salt and water restriction as necessary to manage fluid overload
    - Phosphate restriction
    - Potassium restriction as necessary
  - Erythropoietin agonists - for management of anemia associated with chronic renal failure
  - Phosphate binders - for management of hyperphosphatemia in chronic renal failure

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- Calcium supplements - for hypocalcaemia associated with chronic renal failure
- Calcitriol and other Vitamin D supplements - for hypocalcaemia and hyperparathyroidism associated with chronic renal failure
- Sodium bicarbonate - for acid-base disturbance
- Diuretics - may be used in situation of volume overload
- Renal dialysis - used when there are manifestations of uraemia and the GFR is < 10mL/min
  - Haemodialysis
  - Peritoneal dialysis
- Renal transplantation

**Medicinal plant**

Demand for medicinal plants is increasing in both developing and developed countries. Research on medicinal plants is one of the leading areas of research globally. However, there is a need to pay closer attention to the issue of bioactivity-safety evaluation and conservation of medicinal plants. Kidney failure is one of the most common diseases in India.

a) Acute renal failure - is a syndrome characterized by rapid onset of renal dysfunction, chiefly oliguria or anuria, and sudden increase in metabolic waste-product in the blood and secondly

b) Chronic renal failure - is a syndrome characterized by progressive and irreversible deterioration of renal function due to slow destruction of renal parenchyma eventually terminating in death. Many plants have been used for the treatment of kidney failure in traditional system of medicine throughout the world. Indeed along with dietary measures, plant preparation formed the basis of the treatment of the disease until the introduction of allopathic medicine. Ethnomedical plants can be used to help forestall the need for dialysis by treating the causes and effect of renal failure, as well as reducing the many adverse effect of dialysis (Yarnell et al., 2007) though; there are few chemical agents to treat acute renal failure. Studies reveal that synthetic nephroprotective agents have adverse effect besides reduce nephrotoxicity, Various environmental toxicant and clinically useful drugs, acetaminophen and gentamicin, can cause severe organ toxicities through the metabolic activation to highly reactive free radical (Adeneye et al., 2008)

Right from its beginning, the documentation of traditional knowledge, especially medicinal uses of plants, has provided many important drugs of modern day.

**Table no. 1** Protective effect of medicinal plants on nephropathy

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Name of Plant</th>
<th>Family</th>
<th>V. Name</th>
<th>Part use</th>
<th>Active principle</th>
<th>Rep. Fre.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Abutilon indicum L.</td>
<td>Malvaceae</td>
<td>Atibalaa</td>
<td>rt,b</td>
<td>Asparagines, Mucilages, Tannin, Alkaloids</td>
<td>L</td>
</tr>
<tr>
<td>2.</td>
<td>Acacia arabica (Willd)</td>
<td>Leguminoseae</td>
<td>Babul</td>
<td>l</td>
<td>Tannin, Flavonoid</td>
<td>L</td>
</tr>
<tr>
<td>3.</td>
<td>Achyranthes aspera L.</td>
<td>Acanthaceae</td>
<td>Aghada</td>
<td>r,b</td>
<td>Alkaloids, Saponin, Tannin Oil</td>
<td>M</td>
</tr>
<tr>
<td>4.</td>
<td>Allium cepa L.</td>
<td>Liliaceae</td>
<td>Onion</td>
<td>Bu</td>
<td>Essential oil organic sulphide, Flavonoid, Phenolic acid</td>
<td>H</td>
</tr>
<tr>
<td>5.</td>
<td>Andropogon muricatus Retz.</td>
<td>Gramineae</td>
<td>Kalavala</td>
<td>l, fl</td>
<td>Essential oil</td>
<td>M</td>
</tr>
<tr>
<td>6.</td>
<td>Anona Squamosa L.</td>
<td>Annonaceae</td>
<td>Custard apple</td>
<td>Ls</td>
<td>Alkaloid Aminoacids, Camphor, Anonane</td>
<td>H</td>
</tr>
<tr>
<td>7.</td>
<td>Arachis hypogaea L.</td>
<td>Fabaceae</td>
<td>Mung-phali</td>
<td>S</td>
<td>Vit</td>
<td>H</td>
</tr>
<tr>
<td>8.</td>
<td>Asclepias syriaca L.</td>
<td>Asclepiadaceae</td>
<td>Mohari</td>
<td>Rt</td>
<td>Glucol, Asclepiadin</td>
<td>L</td>
</tr>
<tr>
<td>9.</td>
<td>Asperagus racemosus Wild</td>
<td>Liliaceae</td>
<td>Shatawari</td>
<td>Rt</td>
<td>Oil, Saponin</td>
<td>H</td>
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<tr>
<td>10.</td>
<td>Azadirachta indica L.</td>
<td>Meliaceae</td>
<td>Nimb</td>
<td>L</td>
<td>Alkaloid, Steroid, Azadirin, Resin, Tannin, Fixed oils</td>
<td>H</td>
</tr>
<tr>
<td>11.</td>
<td>Bacopa monnieri L.</td>
<td>Scrophulariaceae</td>
<td>Brahmmi</td>
<td>L</td>
<td>Essential Oil, Alkaloid</td>
<td>M</td>
</tr>
<tr>
<td>12.</td>
<td>Barleria prionitis Linn.</td>
<td>Acanthaceae</td>
<td>Kate-Koranti</td>
<td>fl, l</td>
<td>Essential Oil, Flavonoid Glycoside, (\beta)-Sitosterol</td>
<td>M</td>
</tr>
<tr>
<td>13.</td>
<td>Basella alba L.</td>
<td>Basellaceae</td>
<td>Indian spinach</td>
<td>L</td>
<td>Iodine, Fluorine, Carotenoids, Flavonoid</td>
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<tr>
<td>14.</td>
<td>Boerhavia diffusa L.</td>
<td>Nyctaginaceae</td>
<td>Punarnava</td>
<td>Wp</td>
<td>Alkaloids, Triacetcotanol, (\beta)-Sitosterol, Glucos e Fructose</td>
<td>H</td>
</tr>
<tr>
<td>15.</td>
<td>Bombas ceiba L.</td>
<td>Bombaceae</td>
<td>Salmali</td>
<td>Fr</td>
<td>Tannins, (\beta)-Sitosterol, D-glucoside</td>
<td>M</td>
</tr>
<tr>
<td>No.</td>
<td>Name</td>
<td>Family</td>
<td>Common Name</td>
<td>Key Components</td>
<td>Remarks</td>
<td></td>
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<tr>
<td>16.</td>
<td>Brassica oleracea L</td>
<td>Brassicaceae</td>
<td>Cabbage</td>
<td>L, essential, aminoacid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>Butea monosperma Lam</td>
<td>Fabaceae</td>
<td>Falash</td>
<td>L, Glucoside,Buttern, proteolytic lipolytic enzyme, Flavonoid</td>
<td>H</td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>Cajanus cajan L Millsp</td>
<td>Fabaceae</td>
<td>Tuvar</td>
<td>Ls, Amino acid, galactosid</td>
<td>M</td>
<td></td>
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<tr>
<td>19.</td>
<td>Carica papaya L</td>
<td>Caricaceae</td>
<td>Papaya</td>
<td>Fr, Alkaloid, papain enzymes.</td>
<td>L</td>
<td></td>
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<tr>
<td>21.</td>
<td>Cassia fistula L</td>
<td>Caesalpiniaceae</td>
<td>Bahava</td>
<td>1 po, Glycoside, Tannin, Flavonoid.</td>
<td>L</td>
<td></td>
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<td>22.</td>
<td>Clitoria ternatea L</td>
<td>Papilionaceae</td>
<td>Aparajita</td>
<td>Rt, Teraoxeron, glucoside, oligosaccharide</td>
<td>H</td>
<td></td>
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<tr>
<td>24.</td>
<td>Cordia dichotoma Forst</td>
<td>Boraginaceae</td>
<td>Bhoker</td>
<td>Fr, Alkaloid, Tannin</td>
<td>H</td>
<td></td>
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<td>25.</td>
<td>Crataeva religiosa Buch,Ham</td>
<td>Capparidaceae</td>
<td>Varun</td>
<td>B, L, Linalool, linalyl acetate, thymol, β-caryophyllene α-pinene borneol, limonene, β-phellandrene, citral, citral, citral</td>
<td>M</td>
<td></td>
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<td>26.</td>
<td>Curculigo orchioidesGuert</td>
<td>Amaryllidaceae</td>
<td>Kalimudi</td>
<td>Rt, Saponine, curcuma, phenolic glycoside</td>
<td>H</td>
<td></td>
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<tr>
<td>27.</td>
<td>Gymnodon daeictylon Pers</td>
<td>Gramineae</td>
<td>Durva</td>
<td>Rt, β-ionone, 2-propionionic-hydroxybenzoic</td>
<td>M</td>
<td></td>
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<tr>
<td>28.</td>
<td>Cyperus rotundus L</td>
<td>Cyperaceae</td>
<td>Nagermotha</td>
<td>Rh, Essential oil, cyperen e, cyperol, starch β-sitosterol</td>
<td>H</td>
<td></td>
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<tr>
<td>29.</td>
<td>Datura metal L</td>
<td>Solanaceae</td>
<td>Datura</td>
<td>Lh, Alkaloid, scopolamine, ne, hyposcyamine, atrcin, orpin, vitatc</td>
<td>M</td>
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<td>30.</td>
<td>Daucus carota L</td>
<td>Umbelliferae</td>
<td>Carrot</td>
<td>Rt, I, Oil, carotol essential oil, Flavones</td>
<td>L</td>
<td></td>
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<td>31.</td>
<td>Dolichos biflorus L</td>
<td>Leguminosae</td>
<td>Kalith</td>
<td>S, Urease, lectin carbohydrate, carbohydrate</td>
<td>L</td>
<td></td>
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<td>32.</td>
<td>Ficus religiosa L</td>
<td>Moraceae</td>
<td>Piple</td>
<td>B, L, Arabinose, mannose, glucose β-sitosterol D-glucoside</td>
<td>H</td>
<td></td>
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<tr>
<td>33.</td>
<td>Gmeliana arborea(Roxb)</td>
<td>Verbenaceae</td>
<td>Jivanti</td>
<td>Fr, L, Volatile oil, sugar</td>
<td>H</td>
<td></td>
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<td>34.</td>
<td>Gossypium arboreum (L.)</td>
<td>Malvaceae</td>
<td>Cotton</td>
<td>L, Betaine, choline, Salicylic acid</td>
<td>M</td>
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<td>35.</td>
<td>Gymnema sylvestrer(Rez)R,Br</td>
<td>Asclepiadaceae</td>
<td>Gudmar</td>
<td>Lwp, Saponine, I-V, gymnemic acid</td>
<td>H</td>
<td></td>
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<td>36.</td>
<td>Helianthus annus L</td>
<td>Compositae</td>
<td>Sunflower</td>
<td>S &amp; rtJ, Albumin, globulin, glutelin, flotosterol</td>
<td>H</td>
<td></td>
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<tr>
<td>37.</td>
<td>Hemidesmus indicus L</td>
<td>Asclepiadaceae</td>
<td>Anant mul</td>
<td>rt 1s, Essential oil, Steroid, saponin, r esin tannine</td>
<td>M</td>
<td></td>
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<td>38.</td>
<td>Hibiscus sabdariffa L</td>
<td>Malvaceae</td>
<td>China Rose</td>
<td>L, Organic acid, anthocyanin, vitamin C</td>
<td>M</td>
<td></td>
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<tr>
<td>39.</td>
<td>Holarrhena antidysenterica</td>
<td>Apocynaceae</td>
<td>Kala-Kada</td>
<td>Bx, Alkaloid, tannin, Triterpenes</td>
<td>H</td>
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</tr>
<tr>
<td>40.</td>
<td>Hygraphe auriculata K,Schum.</td>
<td>Acanthaceae</td>
<td>Neermali</td>
<td>rt, 1, Fatty oil, Alkaloid, cal cium, phosphate, K, C, L</td>
<td>M</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Plant Name</td>
<td>Family</td>
<td>Part(s)</td>
<td>Chemicals</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>------------</td>
<td>---------------------------------------------------------------------------</td>
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</tr>
<tr>
<td>41</td>
<td>Jasminium grandiflorum L.</td>
<td>Oleaceae</td>
<td>Chameli</td>
<td>Alkaloid, essential oil, Ascorbic acid, Glucoside</td>
<td></td>
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</tr>
<tr>
<td>42</td>
<td>Leptadenia reticulata W. &amp; A</td>
<td>Asclepiadaceae</td>
<td>Jivanti</td>
<td>Stigma sterol, tocopherol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>43</td>
<td>Leptadenia reticulata W. &amp; A</td>
<td>Asclepiadaceae</td>
<td>Gokarna,</td>
<td>Stigma sterol, tocopherol</td>
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<tr>
<td>44</td>
<td>Linum usitatissimum L.</td>
<td>Linaceae</td>
<td>Aalsi</td>
<td>fixed oil, proteose, wax, resin, sugar, glucoside</td>
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<tr>
<td>45</td>
<td>Mangifera indica L.</td>
<td>Anacardiaceae</td>
<td>Mango plant</td>
<td>Flavonoid Phenolic acid, Vitamin ABCD</td>
<td></td>
<td></td>
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<tr>
<td>46</td>
<td>Menta arvensis L.</td>
<td>Labiatae</td>
<td>Podina</td>
<td>Essential oil, carvone</td>
<td></td>
<td></td>
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<tr>
<td>47</td>
<td>Michelia champaca L.</td>
<td>Magnoliaceae</td>
<td>Champa</td>
<td>Essential oil, fatty oil</td>
<td></td>
<td></td>
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<tr>
<td>48</td>
<td>Mimosapudica L.</td>
<td>Leguminosae</td>
<td>Lajalu</td>
<td>Alkaloids, Mimoseine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>Momordica dioica Robb ex Willd</td>
<td>Cucurbitaceae</td>
<td>Jangali karelaa</td>
<td>Glyceride, saponin</td>
<td></td>
<td></td>
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<tr>
<td>50</td>
<td>Moringa oleifera Lam</td>
<td>Moringaceae</td>
<td>Drumstick tree</td>
<td>Carene, nuciferine, acid, ascorbic acid, amino acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>51</td>
<td>Mucuna pruriens L.</td>
<td>Leguminosae</td>
<td>Khajkauri</td>
<td>Calcium, phosphorus, iron, sulphur, alkaloids</td>
<td></td>
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<tr>
<td>52</td>
<td>Murraya Koenigii L.</td>
<td>Rutaceae</td>
<td>Karry patta</td>
<td>Oil, ?caryophyllene, ?gurjunene, ?carbazol, Alkaloid</td>
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<tr>
<td>53</td>
<td>Musa paradicaea L.</td>
<td>Scistaminaceae</td>
<td>Banana</td>
<td>Albumin, globulin, glutelin, proteose</td>
<td></td>
<td></td>
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<td>54</td>
<td>Nelumbium nucifera gaerts</td>
<td>Nelumbonaceae</td>
<td>Lotus</td>
<td>Alkaloids, nuciferine, proteose sugar, vitamin</td>
<td></td>
<td></td>
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<tr>
<td>55</td>
<td>Nerium indicum Mill</td>
<td>Apocynaceae</td>
<td>Kaner</td>
<td>Glycoside, Digitoxigenin</td>
<td></td>
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<tr>
<td>56</td>
<td>Nyctanthus arbor weresa L.</td>
<td>Oleaceae</td>
<td>Parijat</td>
<td>Oil, manitol, Lannin, biotinogen</td>
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<tr>
<td>57</td>
<td>Ocimum sanctum L.</td>
<td>Labiatae</td>
<td>Tulasi</td>
<td>Eugenol, methyl, ethyl, carvacol</td>
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<tr>
<td>58</td>
<td>Paederia foetida L.</td>
<td>Rubiaceae</td>
<td>Hirenwel</td>
<td>Essential oil, Alkaloids, foetida</td>
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<td></td>
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<tr>
<td>59</td>
<td>Phaseolus mungo L.</td>
<td>Leguminoseae</td>
<td>Green gram</td>
<td>2.6% ash, Oil</td>
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<td>60</td>
<td>Philanthus neruri L.</td>
<td>Euphorbiaceae</td>
<td>Bhuiamla</td>
<td>Phyllanthin, hypophytanthin</td>
<td></td>
<td></td>
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<tr>
<td>61</td>
<td>Philanthus niruri L.</td>
<td>Euphorbiaceae</td>
<td>Bhui awala</td>
<td>Alkaloid, Flavonoids, Phyllanthin, hypophytanthin</td>
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<td></td>
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<tr>
<td>62</td>
<td>Pimpinella anisum L.</td>
<td>Umbelliferae</td>
<td>Rajanigandha</td>
<td>Volatile oil, flavonoid, Sterol</td>
<td></td>
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<tr>
<td>63</td>
<td>Raphanus sativus L.</td>
<td>Cruciferae</td>
<td>Radish</td>
<td>Essential oil, Glucoside, enzyme, methylene Immaculate</td>
<td></td>
<td></td>
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<tr>
<td>64</td>
<td>Rosa damascene (Mill)</td>
<td>Rosaceae</td>
<td>Rose</td>
<td>Essential oil</td>
<td></td>
<td></td>
</tr>
<tr>
<td>65</td>
<td>Saccharum officinarum L.</td>
<td>Poaceae</td>
<td>Sugar cane</td>
<td>Phenol, Glycolic acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>66</td>
<td>Santalum album L.</td>
<td>Santalaeae</td>
<td>Safed Chandan</td>
<td>Santalbic acid, palmitic acid, olic acid</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

49
Diabetic nephropathy is treated with medicines that lower blood pressure and protect the kidneys. These medicines may reverse kidney damage and are started as soon as any amount of protein is found in the urine (microalbuminuria). The use of these medicines before nephropathy occurs may also help prevent nephropathy in people who have normal blood pressure.

If you have high blood pressure, two or more medicines may be needed to lower your blood pressure enough to protect the kidneys. Medicines are added one at a time as needed. The American Diabetes Association recommends a target blood pressure of less than 130/80 millimeters of mercury (mm Hg). The level recommended by other groups may vary. Talk with your doctor about what your target blood pressure level should be. For more information on blood pressure, see the topic "High Blood Pressure".

If you take other medicines, avoid ones that damage or stress the kidneys, especially non-steroidal anti-inflammatory drugs (NSAIDs).

It is also important to keep your blood sugar within your target range. Maintaining blood sugar levels within your target range prevents damage to the small blood vessels in the kidneys.

Limiting the amount of salt in your diet can help keep your high blood pressure from getting worse. You may also want to restrict the amount of protein in your diet. If diabetes has affected your kidneys, limiting how much protein you eat may help you preserve kidney function. Talk to your doctor or dietitian about how much protein is best for you.

People who have diabetes are 2 to 4 times more likely than people who don’t have diabetes to die of heart and blood vessel diseases. Eating a low-fat diet can help prevent heart attack, stroke, and other large blood vessel disease (macrovascular disease).

Initial treatment

Medicines that are used to treat diabetic nephropathy are also used to control blood pressure. If you have a very small amount of protein in your urine, these medicines may reverse the kidney damage. Medicines used for initial treatment of diabetic nephropathy include:

- Angiotensine-converting enzyme (ACE) inhibitors, such as captopril, lisinopril, ramipril, and enalapril.
- ACE inhibitors can lower the amount of protein being lost in the urine. Also, they may reduce your risk of heart and blood vessel (cardiovascular) disease.
- Angiotensine II receptor blockers (ARBs), such as candesartan cilexetil, irbesartan, losartan potassium, and telmisartan. You may be given both an ACE inhibitor and an ARB. The combination of these medicines may provide greater protection for your kidneys than either medicine alone.

If you also have high blood pressure, two or more medicines may be needed to lower your blood pressure enough to protect your kidneys. Medicines are added one at a time as needed. The American Diabetes Association recommends a target blood pressure of less than 130/80 millimetres of mercury (mm Hg).

If you take other medicines, avoid ones that damage or stress the kidneys, especially non-steroidal anti-inflammatory drugs (NSAIDs).

It is also important to keep your blood sugar within a target range to prevent damage to the small blood vessels in the kidneys. The American Diabetes Association recommends that you keep your blood sugar levels at:

- 70 mg/dL to 130 mg/dL before meals and 110 mg/dL to 150 mg/dL at bedtime.

MODERN MEDICINE:

Diabetic nephropathy is treated with medicines that lower blood pressure and protect the kidneys. These medicines may reverse kidney damage and are started as soon as any amount of protein is found in the urine (microalbuminuria). The use of these medicines before nephropathy occurs may also help prevent nephropathy in people who have normal blood pressure.

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- 70 mg/dL to 130 mg/dL before meals and 110 mg/dL to 150 mg/dL at bedtime.
Recent treatment
As diabetic nephropathy progresses, blood pressure usually rises, making it necessary to add more medicine to control blood pressure and keep it below 130/80 mm Hg. Your doctor may advise you to take the following medicines that lower blood pressure. You may need to take different combinations of these medicines to best control your blood pressure. By lowering your blood pressure, you may reduce your risk of kidney damage. Medicines include:

1. A combination of Angiotensin-converting enzyme (ACE) inhibitors and Angiotensin II receptor blockers (ARBs). A combination of these medicines may be more effective in controlling blood pressure than either used alone.

2. Calcium channel blockers lower blood pressure by making it easier for blood to flow through the vessels. Examples include diltiazem (such as Cardizem SR, Dilacor XR, or Tiazac), verapamil (such as Calan SR or Isoptin SR), and diltiazem (such as Norvasc), and nifedipine (such as Adalat or Procardia XL).

3. Diuretics. Medicines such as chlorthalidone, hydrocholorothiazide, or spironolactone help lower blood pressure by removing sodium and water from the body.

4. Beta-blockers lower blood pressure by slowing your heartbeat and reducing the amount of blood pumped with each heartbeat. Examples include atenolol (Tenormin), carvedilol (Coreg), or metoprolol (such as Lopressor).

5. Continue to avoid other medicines that may damage or stress the kidneys, especially non-steroidal anti-inflammatory drugs (NSAIDs). And it is still important to keep your blood sugar within your target range, limit salt in your diet, restrict the amount of protein you eat, keep your cholesterol at a healthy level, eat a low-fat diet, get regular exercise, and do not smoke.

Treatment if the condition gets worse
If damage to the blood vessels in the kidneys continues, kidney failure eventually develops. When that occurs, it is likely that you will need dialysis treatment (renal replacement therapy)—an artificial method of filtering the blood—or a kidney transplant to survive. For more information, see the topic Chronic Kidney Disease.

Diabetic nephropathy can get worse during pregnancy and can affect the growth and development of the fetus. If your nephropathy is not severe, your kidney function may return to its pre-pregnancy level after the baby is born. If you have severe nephropathy, pregnancy may lead to permanent worsening of your kidney function. If you have nephropathy and are pregnant or are planning to become pregnant, talk with your doctor about which medicines you can take. You may not be able to take some medicines (for example, Angiotensin-converting enzyme [ACE] inhibitors, such as captopril, lisinopril, ramipril, or enalapril) during pregnancy, because they may harm your developing baby.

Prevention
Prevention is the best way to avoid kidney damage from diabetic nephropathy.

Keep your blood sugar levels within a target range. Manage your blood sugar by eating a balanced diet, taking your medicines (insulin or oral medicines), and getting regular exercise. The American Diabetes Association recommends that you keep your blood sugar levels at:

- Less than 180 mg/dL 1 to 2 hours after meals.
- Less than 180 mg/dL 1 to 2 hours after meals.
- Less than 180 mg/dL 1 to 2 hours after meals.
- Less than 180 mg/dL 1 to 2 hours after meals.

Your doctor will want you to check your blood sugar several times each day. For more information, see: Diabetes: Checking Your Blood Sugar.

Have yearly testing for protein in your urine. If you have type 1 diabetes, begin urine tests for protein after you have had diabetes for 5 years. Children with type 1 diabetes should begin yearly urine protein screening when they are 10 years of age and have had diabetes for 5 years. If you have type 2 diabetes, begin screening at the time diabetes is diagnosed.

Keep your blood pressure at less than 130/80 mm Hg with medicine, diet, and exercise. Learn to check your blood pressure at home. For more information, see: Chronic Kidney Disease: Changing Your Diet.

High Blood Pressure: Checking Your Blood Pressure at Home.

Stay at a healthy weight. This can help you prevent other diseases, such as high blood pressure and heart disease. For more information, see the topic Weight Management.

Follow the nutrition guidelines for hypertension (including the Dietary Approaches to Stop Hypertension, or DASH, diet). For more information, see: High Blood Pressure: Using the DASH Diet.

Do not smoke or use other tobacco products. For more information, see the topic Quitting Smoking.

If you already have diabetic nephropathy, you may be able to slow the progression of kidney damage by:

- Avoiding dehydration by promptly treating other conditions such as diarrhea, vomiting, or fever that cause it. Be especially careful during hot weather or when you exercise.

- Reducing your risk of heart disease. Lifestyle changes such as eating a low-fat diet, quitting smoking, and getting regular exercise can help reduce your overall risk of developing heart disease and stroke. For more information, see the topics Healthy Eating, Fitness, and Quitting Smoking.

- Treating other conditions that may block the normal flow of urine out of the kidneys, such as kidney stones, an enlarged prostate, or bladder problems.

- Not using medicines that may be harmful to your kidneys, especially non-steroidal anti-inflammatory drugs (NSAIDs).

- Be sure that your doctor knows about all prescription, non-prescription, and herbal medicines you are taking.

- Avoiding X-ray tests that require IV contrast material, such as angiograms, intravenous pyelography (IVP), and some CT scans. IV contrast can cause further kidney damage. If you do need to have these types of tests, make sure your doctor knows that you have diabetic nephropathy.

- Avoiding situations where you risk losing large amounts of blood, such as unnecessary surgeries. Do not donate blood or plasma.

- Lowering your blood pressure, because high blood pressure can make kidney damage even worse.

- Checking with your doctor to find out if it is safe for you to drink alcohol. If you do drink alcohol, have no more than 1 drink a day. Limiting alcohol can lower your blood pressure and lower your risk of kidney damage.[11][12]

Conclusion
It is aimed to record medicinal folk-lure for curing nephropathy that exist in threatening stage. In India ayurvedic referred system of medicines several, herbal drugs and are prescribed for reducing renal damage and to avoid kidney related complication. These can be immense value in combating renal damage. In this paper, we have attempted to use our best endeavors of indigenous herbs to alternative medicine of renal damage. Family Euphorbiaceae, Leguminosae are mostly used as burning urination. A plant used to eradicate kidney stone formation is given in Table.

Annual screening for microalbuminuria will allow the identification of patients with nephropathy at a point very
Early in its course. Improving glycemic control, aggressive antihypertensive treatment, and the use of ACE inhibitors or ARBs will slow the rate of progression of nephropathy. In addition, protein restriction and other treatment modalities such as phosphate lowering may have benefits in selected patients.

As diabetic nephropathy progresses, blood pressure usually rises, making it necessary to add more medicine to control blood pressure and keep it less than 130/80 mm Hg. Patients at risk for acute uric acid nephropathy can be given allopurinol or rasburicase prior to treatment with cytotoxic drugs. Prevention is the best way to avoid kidney damage from diabetic nephropathy.

Keep your blood sugar levels within a target range. Manage your blood sugar by eating a balanced diet, taking your medicines (insulin or oral medicines), and getting regular exercise.

References

9) Rudnick M, VALOR presentation at ASN 05, 2005.
13) Rudnick M, VALOR presentation at ASN 05, 2005.