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AYURVEDIC MANAGEMENT OF DIABETIC KIDNEY DISEASE IN INSULIN DEPENDENT TYPE 2 DIABETES MELLITUS CASE - A CASE REPORT

Nidhi Verma[1], Mandip Goyal[2], Siddhesh Waje[3]

[1]Ph.D Scholar, [2]Associate Professor, [3]Ph.D Scholar,
Department of Kayachikitsa, IPGT and RA Gujarat Ayurved University Jamnagar, Gujarat, India

Email: smilynidhi25@gmail.com

ABSTRACT

Diabetic nephropathy is a disorder affecting the kidneys, in the form of damage to the glomerular basement membrane mesenglial cell proliferation and glomeruloschlerosis diabetic nephropathy is the 3rd leading cause of death, among end stage renal disease patients. It is characterized by excessive albumin secretion. A 60 years old man visited to outer patient department of Kaya Chikitsa IPGT and RA. Hospital, Gujarat Ayurved University, Jamnagar, with the complaint of swelling on both feet, burning sensation on soles, lethargy, pain on both calf muscle breathlessness and increase frequency of micturation, having history of Insulin dependent Type 2 diabetes mellitus (RBS-334mg/dl) and hypertension (160/80mmHg) from 16 years, albumin was found to be present in Urine. After Ayurvedic treatment of 40 days patient was discharged with clinical improvement along with improved kidney function and good controlled diabetes.

Keywords: Ayurveda, Diabetic kidney disease, *Madhumeha*

INTRODUCTION

Diabetic Kidney disease (DKD) is characterized by excessive urinary albumin excretion followed by loss of kidney function. According to urinary albumin excretion values DKD has been didactically categorized into 3 stages 1st stage is normoalbuminuria when urine albumin excretion is <30mg/24hr. 2nd stage is microalbuminuria when urine albumin excretion is between 30 to 299 mg/24hr. 3rd stage is macroalbuminuria when urine albumin excretion is ≥300 mg/24hrs.[1] Diabetic nephropathy is a disorder affecting the kidneys, in the form of damage to the glomerular basement membrane mesenglial cell proliferation and glomeruloschlerosis diabetic nephropathy is the 3rd leading cause of death, among end stage renal disease patients. Ranjit et all investigated prevalence and risk factors of diabetic nephropathy in an urban south Indian population. The prevalence of overt nephropathy was 2.2%. Microalbiminuria was present in 26.9% compared to the non diabetic kidney disease subjects. Kidney disease subjects had greater prevalence rate of microalbuminuria with retinopathy and overt nephropathy, logistic regression analysis has shown that HbA1C, smoking, duration of diabetes, blood pressure, were associated with microalbuminuria. Duration of diabetes and systolic B.P were associated with overt nephropathy.

This study revealed that elevate plasma creatinine had two to three fold increased rate of cardiovascular disease relative to subjects with macroalbuminuria only.[2] Increasing albumin excretion may lead to increased cardiovascular disease directly, or may marker of underling abnormality such as enhanced platelet aggregability, penetration of endothelium by athrogenic lipoprotein particles. Alder et al investigated on development and progression of nephropathy in type 2 diabetes. This study predicts that patients without micro-albuminuria at diagnosis of diabetes remain free of nephropathy for a median of approximately 19 years, it has been suggested that most patients reach at End Stage Renal Disease within 10 years after the onset of proteinuria.[3] With the help of Ayurveda one can prolonged this period even reverse the process to some extent as previous studies prove[4].

CASE REPORT-

A 60 yrs old, middle class, man visited to outer patient department of Kaya Chikitsa IPGT and RA. Hospital, Gujarat Ayurved University, Jamnagar. O.P.D. (Reg.No.-18028003) on 22/3/2018 with the complaint of swelling on both feet, burning sensation on both soles, lethargy, pain on both calf muscle breathlessness having history of insulin dependent Type 2 diabetes mellitus and hypertension from 16 years and diagnosed with liver parenchymal disease with mild spleenomegaly. General Examination revealed bilateral pedal edema pits on firm pressure cyanosis. Clubbing was absent, B.P was 160/80 mmHg, pulse rate was 81 regular, random blood sugar was 334mg/dl, respiratory rate was 20 per minute weight 65 Kg, height 160 cm BMI 25.4 Central obesity was found, lungs was clear, heart sound was normal, abdomen examination was soft, no palpable organomegaly was found. Superficial and deep reflex was normal, patient was of *Vata Kapha Prakruti*. Status *Agni* was normal *Mutrapravriti* was increased up to 10-12 times per day and 3 times per night, Mala Pravriti 1 time per day with normal consistency. *Jivha* was *Sama*, sleep was disturbed as breathlessness increased on lying down. No family history of diabetes and hypertension was found, patient was on insulin taking Human mixtard 32 units in the morning and 22 units in the evening. The patient was provisionally diagnosed as *Vyadhisankar* (*Madhumeha, Shotha, Shwas*) and admitted in I.P.D (Reg No-181366) on 22/3/2018 for Ayurvedic treatment .

Laboratory evaluation Haematological investigations- Hb concentration was 11.2gm%, TLC 3,600/cumm, Neutrophil-49%, Lymphocytes-30%, Eosinophils-7%, Monocyte-5%, Basophyls-0%, PCV-32.3%, ESR-40mm/hr, Total RBC-40.07mil/cumm, Platelet count-83000.-Biochemical investigations –Blood sugar F-119, PP-249, S.Cholesterol,-108, S.Trigleceride-89, S.HDL-40, S.LDL-30, S.VLDL-18, S.Urea-48, S.creatinine-2.1, Total bilrubin-1.2, Direct 0.6, SGOT-22, SGPT-26, S. Alkaline phosphatase-84, S.Albumin-2.8, S.globulin-3.0, A.G ratio-0.9.Physical examination of urine reveal pale yellow colour, turbid appearance and acidic reaction, Chemicaly Albumin was present and sugar absent, microscopic examination –plenty of puss cells was found. USG Abdomen shows shrunken left kidney changes of past nephritis.

Assessment- Based on medical history, records, and physical examination and lab results patients was assessed as chronic case of diabetes mellitus, with impaired renal and liver function which results in pedal edema, dyspnoea, along with myalgia and diabetic neuropathy**.**

Treatment plan- After assessment of patient care plan was *Deepana* (digestive system stimulant) *Pachana* (digestive), *Anulomana* (laxative) and *TiktaKashaya, Upshoshana* (bitter and astringent), *Dravyas* along with *Shothahara* properties. Goal of the treatment was to control blood sugar, symptomatic relieve, improve liver functions and kidney function. For this he was advised to take *Eranda Bhrishta Haritaki* 3gm+*Kutaki* 1gm at bed time, *Punarnavashtaka Kwatha* (*Punarnava, Nimba,*

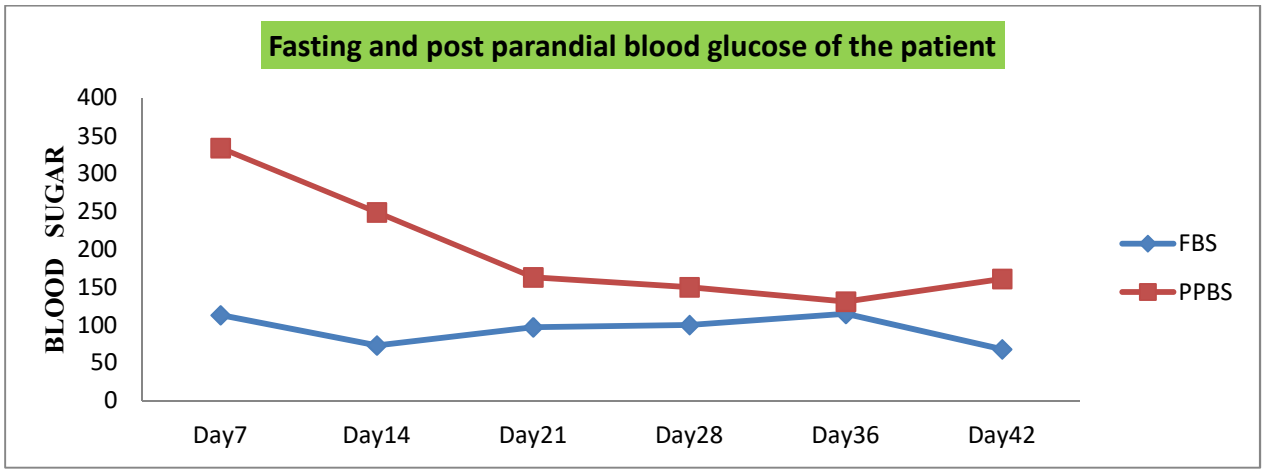
Patol, Kutaki, Shunthi, Guduchi, Devdaru, Haritaki) 10 ml + *Vijaysaradi Kwatha* (*Vijaysar, Haritaki, Amalki, Vibhitaki, Kiratatikta, Patolapatra, Katuki, Gokshura, Musta, Swetachandan, Daruharidra, Usira*) 10 ml before breakfast and dinner, and *Chandrprabha Vati* 2 tab (250mg) thrice in a day was administered He was kept on only *Mudaga Yush* and wheat chapaties, patient was discharged after substantial relief in pedal edema, dyspnea, lethargy and decreased frequency of micturation in 40 days.

Outcome: At the end of 7th day of medication there was marked reduction in pedal edema mild relief in dyspnoea was found which increases on lying down and sleep was disturbed due to the same, frequency of micturation was decreased up to 6-7 times/24 hrs, on 10th day patient noticed marked improvement in dyspnea also felt mild improvement in calf muscle pain and weakness, on 15th day patient told that he got relief in calf muscle pain but burning sensation on both soles still exists. Patient was discharged on 40th day with complete relief in pedal edema, marked improvement in dyspnea, lethargy and calf muscle pain along with mild improvement in burning sensation on both soles, frequency of micturation was decreased up to 4-5 times per day and 1 times at night fasting blood sugar was reduced to 68mg/dl and PPBS was reduced up to 162 mg/dl. Serum urea was reduced up to 42mg/dl S.cretenine was reduced upto 1.9 mg/dl. S.bilrubin was found to increased 0.9 to 1.4 but SGPT was reduced to 30 IU/L from 72 IU/L.

Table 1: Showing biochemical parameters before and after treatment.

Biochemical parameters	Before Treatment	After Treatment
S.creatinine	2.1	1.9
S.Urea	48	42
Total bilrubin	0.9	1.4
SGOT	30	18
SGPT	72	30
S.alkaline phosphatase	100	99

Fig 1: Showing blood Sugar fasting and Post parandial from baseline to after treatment



DISCUSSION

According to *Ayurveda*, nephropathy is a disease of *Mutravaha Srotasa* involving all the three *Doshas* are responsible *Kapha* is responsible for the *Srotosanga* i.e. blocking micro vessels and developing microangiopathy, and blockage can be removed

by the preparations having *Lekhana* (scraping) effect like *Chandraprabha Vati* which is known and commonly used medicine in the disease of *Mutravaha Srotas*. It is specially indicated in *Prameha, Mutarkrichha a*nd *Mutraghata.* Damage in tissue can be repaired and prevent by the use of Rasayanas like *Guduchi, Triphala* and *Gokshura* (contents of *Vijaysaradi Kwath*), along with this *Vijaysaradi Kwath* contains mainly *Tikta Kashaya Rasa* and have *Rukshana* properties *Tikta Rasa* has *Kleda-meda-vasa-majja-lasika Shoshana* properties[5] and is known to successfully used for the treatment of *Madhumeha, Punarnavashtaka* is indicated for the Treatment of *Shwasa* and *Yakrut Roga.* V.N Shah et al and some other researches proved *in vivo* and *in vitro* antioxidant and hepatoprotective effect of *Punarnasvashtaka Kwath*[6] Brijesh et al conducted a study to find role of oxidative stress on Diabetic nephropathy, this study was conducted in 4 groups in West India population control 235, type 2 DM 214, nephrotathy with diabetes (DN) 188 and nephropathy without diabetes 196. Oxidative stress markers such as malondialdehide (MDA), glutathionine (GSH), Superoxide dismutase and catalase were measured in all the groups. The higher serum levels of oxidative stress markers in diabetic patients with nephropathy suggest the possible role of oxidative stress.[7] So, keeping all these properties medicines are prescribed to the patient. Patient was kept on above medicine along with dietary restriction and advised to continue medicine after getting discharged from the hospital. Marked symptomatic improvement was found along with good control on diabetes (Fig.1) which is must for diabetic neuropathy and also decrease in serum urea was also noticed in Patient (Table.1)

CONCLUSION

It can be concluded that the cases of diabetic neuropathy can be successfully managed with the help of above regimen having antioxidant, immunomodulating, antidiabetic effect this can protect the kidney from further damage and reverse the damage to some extent, as this is the disease advanced stage and regular treatment which can also be prescribed on OPD basis. Present case report opens the way for the new treatment modalities in the cases of diabetic nephropathy however a further large scale trial may be required to understand the probable mode of action of such management in the cases of diabetic kidney disease.

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