ayurlog N J-R A S
e-ISSN: 2320-7379
April- June 2019 | Vol. 07th | Issue:2nd
National Journal of Research in Ayurved Science

Effect of ayurvedic treatment in diabetic nephropathy: a case study

Minal S. Vaidya[1], Swapil S. Parab*[2], Sanyogita S. Shinde[3]

1. HOD, professor & PG guide, email: drminalsvaidya@gmail.com
2. Final yr. PG Scholar
3. Final yr.PG Scholar

Kaychikitsa Department, Y.M.T Ayurvedic Hospital & College,
Kharghar, Navi-Mumbai, Maharashtra- 410210

*Corresponding author:
vd.swapnilparab@gmail.com

How to Cite this article:
Effect of ayurvedic treatment in diabetic nephropathy: a case study/ Minal S. Vaidya, Dr. Swapil S. Parab, Sanyogita S. Shinde / Ayurlog: National Journal of Research In Ayurved Science 2019; 3(2): pages: 01-05

Ethical approval:
Approved by the Institutional ethics committee

Conflict of Interest:
None declared

Sources of Funding: None

Date of Submission: 28/02/2019.
Date of Peer Review: 28/03/2019.
Date of Acceptance: 28/03/2019.
Date of Publishing: 01/04/2019.

Keywords:
Diabetic Neohropathy, Chronic Kidney Disease, *Vajeri Basti*, Renal Profie, *Vrikka Rog*.

Name of Publication
Dudhamal Publications
(OPC) Pvt. Ltd., Chembur, Mumbai, Maharashtra, India

Abstract:

AIM & BACKGROUND: Diabetic nephropathy (DN), also known as diabetic kidney disease, is the chronic loss of kidney function occurring in those with diabetes mellitus. Protein loss in the urine due to damage to the glomeruli may become massive, and cause a low serum albumin with resulting generalized body swelling and result in the *nephrotic* syndrome. Likewise, the estimated *glomerular* filtration rate (*eGFR*) may progressively fall from a normal of over 90 ml/min/1.73m^2 to less than 15, at which point the patient is said to have end-stage kidney (ESKD). It is usually slowly progressive over years.

CASE DESCRIPTION: A 70 year old female k/c/o DM for 10 years presenting with *anasarca*, puffiness of face, nausea, vomiting, weakness, hiccups, *oliguria* and itching all over body. Her renal and diabetic profile were deranged with huge proteinuria. Patient was on OHA (oral *hypoglycaemic* agent). According to *ayurved* she was diagnosed as case of *prameha upadrav janya kapha pradhan vrikka rog*. **OUTCOME:** After 11 days of *vajeri basti* and oral medication the patient showed significant relief in symptoms as well as reports.

CONCLUSION: Significant relief can be achieved in patients of diabetic nephropathy by applying classical ayurvedic principles. It's a single case study and can lead down road for further research.

INTRODUCTION:

Diabetic nephropathy (DN), also known as diabetic kidney disease, is the chronic loss of kidney function occurring in those with diabetes mellitus. Protein loss in the urine due to damage to the glomeruli may become massive, and cause a low serum albumin with resulting generalized body swelling and result in the nephrotic syndrome. Likewise, the estimated glomerular filtration rate(eGFR) may progressively fall from a normal of over 90 ml/min/1.73m^2 to less than 15, at which point the patient is said to have end-stage kidney (ESKD). It usually is slowly progressive over years. Affected individuals with end-stage kidney disease often require haemodialysis and eventually kidney transplantation to replace the failed kidney function. Diabetic nephropathy is associated with an increased risk of death in general, particularly from cardiovascular disease. The incidences of CKD in INDIA, which is densely populated country with low income, different food, cultural tradition and lifestyle habitat, is 7.85 million of its population and the prevalence rate is 0.78% [1]. Over 1 million people worldwide are alive on dialysis or with a functioning graft. Clinically diabetic nephropathy is characterised by progressive increase in proteinuria and decline in GFR. As per *ayurvedic* classics *upadravas* of *prameha* are nausea, vomiting, oedema, indigestion, hiccups, etc. these symptoms are seen as *upadrava* due to *kapha* and *pitta*. Though complications of *prameha* are well written in *samhitas* there is no clear mention of pathology that can clarify *dosha-dushya samrurchinna* involved in them. Considering nephropathy *vrikka rog* mentioned in *Bhaishjya Ratnavali* matches very well with signs and symptoms of diabetic nephropathy.

CASE DESCRIPTION:

A female patient of 70 years presented in out patient department of Y.M.T. Trust's Ayurvedic Hospital, on 12th November 2018, with complaints of anasarca, puffiness of face, nausea, vomiting, weakness, hiccups, oliguria and itching all over body from 2 months. she was known case of T2DM, she was o combination of gliclazide 80 mg and metformin 500 mg twice a day before meal. Despite above medications patient did not have good glycaemic control. Blood investigations showed sr. creatinine- 1.83 mg/dl, e-GFR - 40 ml/min, blood urea – 38, sr. sodium – 135 mEq/L, potassium – 4mEq/L, chlorides – 99 mEq/L, HbA1C – 9.1%, urine protein – present (+++).

DIAGNOSIS:

In view of modern sciences, it was a clearly case of Diabetic Nephropathy. According to *Ayurveda* the patient clearly shows symptoms of *prameha upadrava* such as vomiting (*chardi),* nausea(*hrillas*) weakness (*durbalya*). But precise diagnosis established was *Prameha upadrav kapha Pradhan vrikka rog* .

TREATMENT GIVEN:

Patient received orally - *chandraprabha vati* – 500 mg twice a day before food, *punarnavashtak kwath* – 40ml twice a day and panchakarma – *vajeri basti* - 120ml for 11 days.

All other allopathic treatment for diabetes were continued as before but patient did not take any treatment other than ayurvedic for nephropathy.

TREATMENT OUTCOME:

After starting *basti* treatment along with oral medication her symptoms like vomiting and nausea started to reduce over course of time. After 5 days of *basti* her anasarca begins to reduce and there was increase in appetite and urine output. On 7th day her weakness was reduced and there was more increase in appetite. On 11th day renal profile, urine routine and microscopic, eGFR were repeated and found that Sr. creatinie – 0.81 mEq/l, Blood urea – 21.7 mEq/L, Sr. sodium – 137 mEq/L, potassium -4 mEq/L, chlorides – 92 mEq/L, eGFR- 69

DISCUSSION:

Diabetic nephropathy is an important cause of morbidity and mortality in both type-1 and type-2 diabetes. It is now the most common cause of End-stage-renal failure in developed countries and account for between 20% and 50% of patients starting renal replacement therapy.

Risk factors:

- Poor *glycemic* control
- Long duration of diabetes
- Presence of other micro-vascular complications
- Ethnicity (Asian, pima Indians)
- Pre-existing hypertension
- Family history of diabetic nephropathy
- Family history of hypertension

About 30% of patients of type-1 diabetes have developed diabetic nephropathy 20 years after diagnosis. The pathophysiology is not fully understood and there are several postulated mechanisms by which hyperglycaemia causes the pathological changes seen in diabetic nephropathy. The central features are activation of the renin-angiotensin system, leading to both intrarenal and systemic effects, as well as direct toxic effects of prolonged hyperglycaemia leading to renal inflammation and fibrosis. The first changes coincide with the onset of microalbuminuria and include thickening of the glomerular basement membrane and accumulation of matrix material in the mesangium. Subsequently nodular deposits are characteristic and glomerulosclerosis worsens, as heavy proteinuria develops until glomeruli are progressively lost and renal function deteriorates.

To facilitate the assessment of CKD severity, the National Kidney Foundation developed criteria as apart of its Kidney Disease Outcome Quality Initiative,[2]

- Stage 1: Normal eGFR > 90 mL/min per 1.73m^2 and persistant albuminuria.
- Stage 2: eGFR between 60 to 89 mL/min per 1.73m^2
- Stage 3: eGFR between 30 to 59 mL/min per 1.73m^2
- Stage 4: eGFR between 15 to 29 mL/min per 1.73m^2
- Stage 5: eGFR < 15 mL/min per 1.73m^2 or End Stage Renal Disease.

Considering nephropathy, *Vrukka Roga* mentioned in *'Bhaishajyaratnavali'* matches very well with sign and symptoms of diabetic nephropathy. So, pathology of Diabetic nephropathy from *ayurveda's* point of view can be considered according to *Vrukka Roga* mentioned in *Bhaishajyaratnavali*. If symptoms of *upadrava* of *prameha* and *vrukka roga* are considered the patient

can be diagnosed as case of'*prameha upadravajanya kapha pradhan vrukkaroga*'. Acharyas have advised to use combination of herbal medicines which have functions such as *mutral, deepen, pachan, raktaprasadak, virechak and rasayana*.[3]

Patient received *chandraprabha vati*[4] which reduces *kapha, pitta, dhatushaithilya* (laxity*), kleda,* well known for its action on *mutrendriya (basti)* Hence, it acts as *rasayana* for *mutravaha srotasa* and have *pramehaghna* property. She also received *punarnawashtak*[5] *kwath*; which has *punarnava*, acts as *shothaghna*. *Punarnava* being *Rasayan* help to rejuvenate the nephron cells and plays reno-protective action (In experiments with Boerhviaduffusa there has been diuresis accompanied by increased excretion of sodium, 1972; *Mudgal Planta,* 1975 and In *Nephrotic* Syndrome,). As mentioned in *Sharangdhara Samhita*. *Haritaki* is *shrestha* in *Anulomana*. Being *Anulomana* it acts as *sroto-vishodhana* also by being *kashaya rasa* and *ruksha gun* helps in *kelda shoshana*. *Nimba* being *ruksha* and *laghu*, absorbs *kleda* being *tikta* and *ruksha* and thus purifies blood which in turns eliminates *kleda*. *Daruharidra* absorbs *kleda, meda* due to its *katu-tikta* and *ruksha* qualities. It also has liver stimulant and *pittasaraka* property due to its *tikta rasa* which helps to regulate the *pitta dosha, agnideepan* as mentioned in *Sharagdhara Samhita Kutaki* is *pradhana dravya* in *Bhedana*. Being this property *kutaki* plays major role in *srotoshodhana*. It helps to expels vitiated *kapha, kleda* and accumulated fluid in body through *purisha*. according to *BhavprakashaSamhita*; *Patol* also plays major role in *srotoshodhana* being it is *sukhakar Virechana*. *Guduchi* causes stimulation of *dhatvagnis* by its *tikta rasa* and nutrition of *dhatus* by its *madhura vipaka*. By its *tikta-katu-kashaya* and *ruksha guna,* it eliminates *kleda* associated with *dhatu*. It also causes stimulation of *medagni* by its *tikta-katu rasa* and *ushna veerya* so *guduchi* acts as reno-protective as *vrukka* is *mul-sthana* of *medovahasrotas*. *Shunthi* is *kaphaghna* being *katu, ushna* and *laghu*. As mentioned in *Sharangdhara Samhita shunti* possesses *Grahi* property. Being *grahi shunthi* absorbs *kleda* and *kapha*. *Gomutra* being *katu-tikta-kashaya* it causes *kleda shoshana*. This collectively helps in *kleda nirhana*. Also increase the bioavailability of drug.

In the process of *Ahara parinaman kriya*, the *Chaturvidha annapana* undergoes the *pachana* by the action of *pachaka pitta*, enters to the *pakwashaya* where it gets divided in to the *Drava bhaga* and the *Ghana bhaga*. The *Ghana bhaga* is the *purisha* and the *Drava bhaga* is the *malakhya kleda* and is carried to the *Basti* from the *pakwashaya* for excretion. Now because of *Kapha prakopa* and *angimandya* there is excessive product of *mala roop kleda*. According to *Ayurveda Mootra* is produced in *Pakwashaya*. In patients of CRF this *Mootra Nirmiti Prakriya* is hampered. Hence, in *Vajeri basti*[6] where we use decoction of *Pakwashaya* of goat so that with the support of '*Samanya Vishesha Siddhant'* we provide similar factors to patient's *Pakwashaya* which will help to regularize the urine production.

CONCLUSION:

As the number of diabetics is growing in India as well as worldwide, number of patients suffering from nephropathy will

also rise. Hence it is high time to improvise our treatment plans and help to answer complicated situations such as diabetic nephropathy. It is an observation in a single case and more studies in this direction would help in establishing ayurvedic treatment in this condition. significant relief can be achieved in patients of nephropathy by applying principles of diagnosis and treatment of *prameha* and *vrukkaroga*. It's a single case study and can lay down road ahead for further reserch.

References:

1. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4446915/
2. API Textbook of Medicine 1st vol. 8th edition reprint 2009 page no.733
3. Bhaishjya ratnavli edited by ambikadatta shastri, chaukhamba prakashan,vrikkaroga chikitsa prakaran93,page no-1193
4. Bhaishjya ratnavli edited by ambikadatta shastri, chaukhamba prakashan,kshudraroga chikitsa prakaran60, page no-961
5. Bhaishjya ratnavli edited by ambikadatta shastri, chaukhamba prakashan,udar roga chikitsa prakaran40,page no-756
6. Article published on IJIRR; Vol-1,issue,12,pp 187-190,dec.2014
7. Sharangadhar Samhita Vd.bhramanad tripathi , chaukhamba Sanskrit sansthan,Varanasi reprint 2008;madham khanda 2-1,2
8. Charak Samhita edited by Dr. brahmanand Tripathi Year 2007 edition Chaukhamba Surbharti Prakashan , Varanasi.
9. Sushrut Samhita edited by Kaviraj Ambikadutta Shastri Year 2007 edition Chaukhamba Surbharti Prakashan , Varanasi.
10. Ashtang Hrudaya Samhita edited by Dr. Brahmanand Tripathi Year 2006 edition Chaukhamba Surbharti Prakashan , Varanasi.
11. API Textbook of Medicine 8th edition reprint 2009
12. Harrison's Principles Of Internal Medicine 18th edition august 2011 publisher- Tata Mcgraw – Hill Education.

End of article