

**CHRONIC KIDNEY DISEASE -A CASE STUDY IN AYURVEDIC SETTING****Ritu Yadav<sup>1</sup>, J.P. Singh<sup>2</sup>, Pankaj Sharma<sup>3</sup>, Vikas Nariyal<sup>4</sup>**<sup>1</sup>MD Scholar, <sup>2</sup>Associate Professor, <sup>3</sup>MD Scholar, <sup>4</sup>Phd Scholar,  
P.G. Department of Kayachikitsa, NIA, Jaipur, Rajasthan, India**ABSTRACT**

Chronic kidney diseases (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate(GFR). The epidemiologic transition has been fuelled by rapid economic development and globalization, leading to rapid urbanization, major lifestyle changes, and altered eating habits. This has been paralleled by a rapid spurt in the incidence and prevalence of non-communicable or so-called lifestyle diseases such as hypertension, diabetes, coronary artery disease, malignancies and chronic kidney disease (CKD). Here we are reporting a case of CKD of a 55 year old male patient. The direct description of the diseases is not available in Ayurvedic science, So we can compare the disease with Ayurvedic concepts only on the basis of general signs and symptoms. The possible understanding of the case in terms of Ayurveda and a therapeutic protocol with promising result has been discussed.

**Key words** - Chronic kidney diseases, glomerular filtration rate, end stage renal diseases.

**INTRODUCTION**

Chronic diseases have become a major public health problem. The direct description of the diseases is not available in Ayurvedic science, So we can compare the disease with Ayurvedic concepts only on the basis of general signs and symptoms. Chronic diseases are a leading cause of morbidity and mortality in India and other low and middle income countries. The chronic diseases account for 60% of all deaths worldwide. Eighty percentage of chronic disease deaths worldwide occur in low- and middle-income countries<sup>1</sup>. The global annual growth of number of ESRD patients is reported at 7% .<sup>2,3</sup>The incidence was suggested to be 100 per million population (pmp)<sup>4,5</sup> by single centre studies from tertiary care hospitals and from experience of opinion leaders. The prevalence of CKD was reported to be 0.79% in study from Delhi which screened 4972 adults. This study used a serum

creatinine cut off >1.8mg/dl to define CKD and hence underestimating the prevalence<sup>6</sup>. Another study by Mani et al in a South Indian village reported the prevalence of GFR < 15ml/min (CKD stage V) to be 0.09%.<sup>7</sup> Etiology of CKD in India is diabetic nephropathy (31.2%), undetermined (16.4%), chronic glomerulonephritis (13.8%), hypertension (12.8%), tubulointestinal diseases (7%), obstructive uropathy (3.4%), autosomal dominant polycystic kidney diseases (2.5%), renovascular diseases (0.8%), kidney transplant graft loss (0.3%), others (11.7%). Clinical and laboratory manifestation of Chronic kidney diseases include fluid, electrolyte and acid base disorders, disturbed potassium homeostasis, metabolic acidosis, disorders of calcium and phosphate metabolism, cardiovascular abnormality include ischaemic heart diseases, heart failure, hypertension and left ventricular failure and

pericardial diseases, haematological abnormalities include anemia, neuromuscular abnormalities, G.I.T and nutritional abnormalities, endocrine and metabolic disturbance etc.<sup>8</sup>

The initial approach for evaluation of patients with CKD include history and physical examination, laboratory investigations includes RFTs, serum concentration of calcium, phosphorus, PTH to evaluate metabolic bone diseases, Hb, iron, folic acid, foliate, 24 h urine evaluation, imaging studies and renal biopsy.<sup>9</sup> Treatment of CKD aimed at specific causes of CKD. For slowing the progress of CKD concern is given to protein restriction, reducing intraglomerular hypertension and proteinuria, control of blood sugar, managing the complications. Finally renal replacement therapy is option.

**Case Report-**A 55 year old male patient came to N.I.A. O.P.D. on 17-4-2014 with following chief complaints Breathlessness, Swelling in bilateral lower limb, Nausea, Indigestion since 6 months.

**Associated complaints-**Pain in small joints of hand and foot, Generalized weakness, abdominal discomfort.

### History of present illness

The patient was quite asymptomatic 1 year before. Gradually he developed pain in small joints of foot starting from toe (pricking type, associated with tenderness and swelling, more during night time) which later on involved all small joints of hands including wrist joint. His blood uric acid was found to be raised. He took treatment from modern consultant but did not get any improvement. Then after some time he gradually developed difficulty in breathing (firstly occur on exertion then orthopnea occur). Later on he developed swelling in B/L L/L (more during evening hours, pitting type). On further investigation he was

diagnosed as CHF and was treated accordingly. He got symptomatic relief but swelling on B/L L/L did not subside. Later on his Blood urea and Sr. creatinine level was found to be raised. He took modern treatment but the condition did not improve. So with the above complaints patient came to N.I.A for further treatment.

**Past history-**No h/o DM, HTN, TB, No any surgical history.

**Drug history-**Iron tablet, Carvedilol (3.125 mg) half tab BD, Digoxin (0.25mg) half tab 5 times/week, Tab. Calcium, Vit D3 1 tab BD. Patient was taking this treatment since 4 months. Dialysis 3 times in last 15 days .

**Family History-** No any relevant family history.

**Vitals at time of first visit to N.I.A.-** B.P. 120/70 mm of Hg, Pulse-82/min, Afebrile, R.R-18/min

**Physical examination -** General condition - fair, Pallor<sup>+</sup>, Icterus<sup>0</sup>, Cyanosis<sup>0</sup>, Clubbing<sup>0</sup>, Pedal Oedema with facial puffiness, Lymph node not palpable, Respiratory system- NAD, CVS-NAD, GIT-NAD, CNS-NAD

### Investigations Done

**Blood examination-** (on dated 17-04-2014)

**CBC-** Normal

**ESR-** Normal

#### RFT

Serum urea-315 mg /dl

Serum creatinine-13.2 mg/dl

**Uric acid -** 9.3 mg/dl

**USG:** Dated ( 24-04-14)

- Hepatosplenomegaly,

- B/L MRD,

- Mild ascitis

#### 2D-ECHO

- Global hypokinesia of left ventricle

- Mild AR/Mild MR

- Mild AS
- Moderate LV systolic function
- LVEF-35%

*ragnimandya* which further leads to *Dhat-vagnimandya* and formation of *Ama* at *Jathar* as well as *Dhathu* level.

## DIAGNOSIS

- The initial pathology of the disease started with involvement of multiple small joints. On lab investigation uric acid level found to be raised which later on deposited in multiple small joints causing joints inflamed and tender.
- Comparing such type of deposition on Ayurvedic parameter it is quite acceptable that there would be *Srotorodha* which make this deposition possible.
- As *Srotorodha* is not possible without *Kapha* and *Ama Vridhi* So there is possible *Ama dosha Utapatti* at multiple level. The possible cause behind that will be *Jatha-*

- So keeping the route cause in mind, the goals set for the treatment are  
-*Aam Doshapachan*  
-*Srotosodhana*  
-Improve quality of life

## AYURVEDIC TREATMENT GIVEN

1. *Bakayan Swarasa* 20 ml BD
2. *Jwarahara Kasaya* 40mlBD
3. *Ashavgandha churna* 3gm  
*Shatavari churna* 3 gm  
*Goksahru churna* 5 gm  
(***Ksheerpaka vidhi***) 40 ml BD
4. *Peepal twaka Kwatha* 40 ml BD
5. *Kaishor Guggulu* 2 tab BD

## RESULTS:

**Table No 1-Showing results on various parameters.**

Date	17-04-2014	30-04-2014	4-07-2014
Blood Urea (mg/dl)	315	205	200
Sr. Creatinine (mg/dl)	13.23	8.9	7.9
Sr.Uric Acid	9.3		7

## DISCUSSION

- The initial pathology of the disease started with involvement of multiple small joints. On lab investigation uric acid level found to be raised which later on deposited in multiple small joints causing joints inflamed and tender.
- Comparing such type of deposition on *Ayurvedic* parameter it is quite acceptable that there would be *Srotorodha* which make this deposition possible.
- As *Srotorodha* is not possible without *Kapha* and *Ama Vridhi* So there is possible *Ama dosha Utapatti* at multiple level. The possible cause behind that will be *Jatharagnimandya* which further leads to *Dhat-vagnimandya* and formation of *Ama* at *Jathar* as well as *Dhathu* level.

- So keeping the route cause in mind, the goals set for the treatment are  
-*Aam Doshapachan*  
-*Srotosodhana*  
-Improve quality of life

## Propable drug of action

### 1.*Bakayana Swaras*

The drug has nephroprotective<sup>10,11</sup> action. The drug having mainly *Tikta Rasa*<sup>12</sup> which also has the property of *Deepana*, *Pachana*, *Lekhana* and *Shodhana*.

### 2. *Jwarhara Kasaya*

It contain *giloy*, *daruharidra*, *triphala*, *tulsi* etc. Maximam dravya contains *tikta* and *katu Rasa* having the property of *Amapachan* and *Srotosodhana*.

### 3. *Ksheerpaka of Ashavghandha, Shatavari and Goksahru churna*

Leads to *utrottara Dhatu Pushti* and improve Rogi bala. *Gokshuradi churn*<sup>13</sup> acts on *Mutra-vaha Sansthan* having the property of *Srotosodhana* and *Mutravirechana*

### 4. *Peepal twak kwath* <sup>(14)</sup>

It is praised for treating even *Tridosha Vataraakta* by *Acharya Charaka*. Due to its *Tikta Kasaya Rasa* it has the property of *lekhana* and *Srotoshodhana*.

### 5. *Kaishora Guggulu*

It contains mainly *tikta dravya* which leads to *Ama Pachana* and *Srotovishodhana* and *Guggulu* also has its own properties of *Lekhana* and microcirculation.

## CONCLUSION

On the basis of above case study it can be concluded that *Bakayan Swaras, Jwarhara Kasaya, Ksheerpaka of Ashavghandha, Shatavari and Goksahru churna, Peepal twak kwath, Kaishora Guggulu* is quite effective in management of chronic renal failure.

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