

# WWW.IJAPC.COM



e ISSN 2350 0204

# **VOLUME 12 ISSUE 1 2020**

GREENTREE GROUP PUBLISHERS (GGP)



CASE STUDY

www.ijapc.com e-ISSN 2350-0204

# Ayurvedic Management of Chronic Renal Failure (CRF) - A Case Report.

Vivekaradhya M<sup>1\*</sup>, Prakash Paltye<sup>2</sup> and Madhu Ranjan<sup>3</sup>

<sup>1-3</sup>Department of Panchakarma, Ashwini Ayurvedic Medical College and Hospital, Tumkur, Karnataka, India

## ABSTRACT

Chronic Renal Failure (CRF) is a syndrome characterized by progressive and irreversible deterioration of renal function due to slow destruction of renal parenchyma eventually terminating in death when significant numbers of nephrons have been destroyed. In CRF the choice of available treatment in the conventional system of medicine includes hemodialysis, nutritional supplement and renal transplantation, which is economically burden to the CRF patients in India.

In this regard *Ayurveda* provides an effective management of the disease by incorporating Ayurvedic drugs, dietary and lifestyle interventions. CRF can be understood as a *Mutravaha Sroto Vikara* as per the principles of *Ayurveda*. Here we are reporting a case of CRF of a 35 year old female patient who was managed successfully with Ayurvedic formulations like Gokshuradi Guggulu, Chandraprabha Vati, Punarnavasam, Neeri KFT. The prognosis was found to be effective and safe.

# **KEYWORDS**

Chronic Renal Failure (CRF), Hemodialysis, Mutravaha-Sroto Vikara, Gokshuradi Guggulu, Neeri KFT.



# **INTRODUCTION**

Chronic Renal Failure (CRF) is a syndrome characterized by progressive and irreversible deterioration of renal function due slow destruction of renal to parenchyma eventually terminating in death when significant numbers of nephrons have been destroyed<sup>1</sup>. Chronic Kidney Disease affected 753 million people globally in 2016, 417 million females and 336 million males<sup>2</sup>. The three most common causes of CRF are Diabetes Hypertension, Mellitus. and Glomerulonephritis<sup>3</sup>. Often, it is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure or diabetes and those with a blood relative with renal disorders.

This initially manifests only as a biochemical abnormality. CRF is considered when Glomerular Filtration Rate (GFR) falls below 30ml/min. In early CRF, the patient is often asymptomatic. Renal failure may present as a raised blood urea and creatinine often accompanied by Hypertension, Proteinuria, Hyperkalemia, Hypocalcemia, Hyperuricaemia or anemia<sup>4</sup>. Clinical symptoms include metabolic acidosis (Kussmauls breathing), decreased urine production, nausea,

anorexia, pedal edema, vomiting, muscular twitching  $etc^5$ .

In CRF the choice of available treatment in the conventional system of medicine includes Hemodialysis<sup>6</sup>, nutritional supplement which is not satisfactory and ultimate goal is to Renal Transplantation, which is economically burden to the CRF patients in India<sup>7</sup>. In this regard Ayurveda provides an effective management of the disease by incorporating Ayurvedic drugs, dietary and lifestyle inventions. CRF can be understood as a *Mutravaha Sroto Vikara* as per the principles of Ayurveda. All the tridoshas and all the dushyas are involved in the disease. Morbid changes in the *srotas* due to accumulation of vitiated *doshas* may cause sangha (Blockage) which can lead to karyahaani of mutravaha srotas (decreased renal functions).

Involvement of *dushyas* can be understood by the analysis of clinical signs and symptoms. Fluid and electrolyte imbalance, cardiovascular complications can be taken as Rasadusti. Anemia, bleeding tendency, nausea can be caused by Raktadusti. Myopathy due to mamsadusti, dislipidemia involvement of due to medas. osteodystrophy due to asthidusti, neuroparthy due to *majja* and *snayu* involvement. Sexual dysfunction, loss of libido indicated Sukradusti. Mutra, sweda and udaka are commonly affected in CRF.



Based on the *dosha*, *dushya*, and *adhistana* along with the *rogabala* and *rogibala* ayurvedic management can be planned in order to increase the quality of life of the patient and to postpone or avoid the dialysis and renal transplant. Here we are reporting a case of CRF of a 35 year old female patient who was managed successfully with Ayurvedic Treatment along with dietary and lifestyle interventions.

#### CASE REPORT

A female patient aged 35 years came to Panchakarma OPD in Ashwini Ayurvedic Medical College and Hospital, Tumkur, Karnataka with the complaints of bilateral pedal edema, puffiness of face, shortness of breathing, decreased urine output, nausea, anorexia, muscle twitching since 6 months. She was a k/c/o HTN since 5 years. She had consulted Fortis Hospital Bengaluru, where she consulted a nephrologist and during routine investigations her serum creatinine was very high along with electrolyte imbalance. She was diagnosed with CRF and was advised with diuretics and other medications. In the next follow up she was advised to undergo dialysis. She underwent dialysis for 2times but didn't find relief from her complaints. Then she decided to take Ayurvedic treatment.

Chief complaints: Bilateral pedal edema, puffiness of face, shortness of breathing,

decreased urine output, anorexia, nausea, muscle twitching since 6 months. Physical Examination General condition: Ill looking Blood Pressure: 160/100 mmhg Pulse: 98 bpm Temp: 98.4 F **Built: Moderate** Weight: 62 Kg Height: 5 feet 4 inches Respiratory Rate: 22 cycles/min Pallor: + +Icterus: Absent Clubbing: Absent Cyanosis: Absent Lymph nodes: Not palpable Edema: b/l edema. pedal pitting perioribital, face.

#### **Personal History**

Appetite: Reduced

- Bowel: Constipated
- Micturition: reduced frequency
- Sleep: Disturbed
- Diet: Mixed
- Habits: No addictions

#### **Menstrual History**

Normal & Regular

No abnormal bleeding or discharge.

#### **Systemic Examination**

CNS: Well oriented to place, person and time. Intact Higher mental functions. No sensory deficits.



CVS: S1 S2 heard, HR: 98bpm, No murmurs heard.

RS: b/l symmetrical air entry. b/l basal mild crepitation heard.

P/A: soft, non-tender, no organomegaly.

Investigations done during first visit: (18/12/2017)

Hb: 9.8 gm/dl (decreased) TLC: 6,800 cells/cumm DC: N-49%, L-41%, E-6%, M-4%, B-0%

ESR: 45 mm/hr (raised) Platelet Count: 3.09 lakh/cumm RBC count: 3.86 millions/cumm

P.C.V: 38% (decreased)

M.C.V: 86 fl

M.C.H: 28 pg

M.C.H.C: 32%

RBS: 132.0 mg/dl

Serum Creatinine: 8.1 mg/dl (raised)

Serum Electrolytes:

 Table 1 Treatment plan for every follow up

Na: 141.2 mEq/L (reduced) K: 3.91 mEq/L Cl: 105.5 mEq/L Urine Examination: Urine Sugar: Nil Urine Albumin: Present + Pus cells: 2-3 cells/hpf Epithelial cells: 3-4 cells/hpf RBCs: Absent.

**Treatment History:** Has taken a course of Antibiotics, Diuretics. On regular medications for Hypertension. Underwent Dialysis for 2 times.

## METHODOLOGY

#### **Treatment Given:**

After thorough physical and systemic examination along with laboratory investigations, the treatment was planned. The treatment given is detailed in Table 1.

First Vi	sit: 04/12/2017		
Sl. No	Formulation	Dose (For 15 days)	Anupana
1	Syp Neeri KFT	10ml-0-10ml b/f	Ushna Jala
2	Gokshuradi Guggulu	2-0-2 a/f	Ushna Jala
3	Punarnavasam	20ml-0-30ml a/f	Ushna Jala
4	Cap Nefpro	0-2-0 a/f	Ushna Jala
Second	Visit: 19/12/2017		
Sl. No	Formulation	Dose (For 30 days)	Anupana
1	Syp Neeri KFT	10ml-0-10ml b/f	Ushna Jala
2	Gokshuradi Guggulu	2-0-2 a/f	Ushna Jala
3	Punarnavasam	15ml-0-15ml a/f	Ushna Jala
4	Tab Chandraprabha Vati	0-2-0 a/f	Ushna Jala
Third V	isit: 03/02/2018		
Sl. No	Formulation	Dose (For 30 days)	Anupana
1	Syp Neeri KFT	10ml-0-10ml b/f	Ushna Jala
2	Gokshuradi Guggulu	2-0-2 a/f	Ushna Jala
3	Punarnavasam	15ml-0-15ml a/f	Ushna Jala
4	Tab Chandraprabha Vati	0-2-0 a/f	Ushna Jala
5	Tab Abhra loha	1-0-1 a/f	Ushna Jala

2020 Greentree Group Publishers © IJAPC www.ijapc.com 43



Fourth V	Fourth Visit: 22/03/2018					
Sl. No	Formulation	Dose (For 30 days)	Anupana			
1	Syp Neeri KFT	10ml-0-10ml b/f	Ushna Jala			
2	Gokshuradi Guggulu	1-0-1 a/f	Ushna Jala			
3	Punarnavasam	20ml-0-20ml a/f	Ushna Jala			
4	Tab Chandraprabha Vati	0-2-0 a/f	Ushna Jala			
5	Tab Abhra loha	1-0-1 a/f	Ushna Jala			
Fifth Vis	sit: 21/06/2018					
Sl. No	Formulation	Dose (For 30 days)	Anupana			
1	Syp Neeri KFT	10ml-0-0 b/f	Ushna Jala			
2	Gokshuradi Guggulu	1-0-1 a/f	Ushna Jala			
3	Punarnavasam	0-0-20ml a/f	Ushna Jala			
4	Tab Chandraprabha Vati	0-2-0 a/f	Ushna Jala			
5	Tab Abhra loha	1-0-1 a/f	Ushna Jala			

#### Diet

Patient was advised to take light food, fresh coriander juice, and restricted water intake according to the 24hour urine output. Patient was advised to restrict salty, spicy, heavy and oily food items, protein rich food (Pulses). Patient was advised to avoid day sleep and suppression of natural urges.

## RESULTS

There was gradual improvement in the patient's general conditions and symptoms during the course of treatment and she was exempted from dialysis. The assessment of the signs and symptoms before, during and after treatment is tabulated in Table No 2.

**Table 2** Assessment of signs and symptoms before and after treatment.

Signs and Symptoms	First Visit 04/12/2017	Second Visit 19/12/2017	Third Visit 03/02/2018	Fourth Visit 22/03/2018	Fifth Visit 06/03/2019
Decreased urine production	+ +	+ +	+	+	-
Pedal edema	++	+ +	+	-	-
Puffiness of face	++	+ +	+	-	-
Shortness of breath	+ +	+	+	-	-
Anorexia	+ +	+	-	-	-
Nausea	+	-	-	-	-
Muscle twitching	++	+	-	-	-

The Laboratory findings are listed in Table

No 3.

**Table 3** Assessment of laboratory findings before and after treatment.

Laboratory	25/11/2017	18/12/2017	03/01/2018	02/02/2018	19/06/2018	25/01/2019
Test						
Hb %		9.8 g/dl	10.2 g/dl	10.7 g/dl	12.4 g/dl	
Serum	9.40 mg/dl	8.1 mg/dl	5.2 mg/dl	3.3 mg/dl	2.50 mg/dl	1.5 mg/dl
Creatinine						
Serum	130 mmol/L	141.2				
Sodium		mmol/L				
Serum	3.63 mmol/L	3.9 mmol/L			3.69 mmol/L	
Potassium						



Serum	88 mmol/L	105.5			
Chloride		mmol/L			
ESR		45 mm/hr	30 mm/hr	28 mm/hr	
Urine	Absent	Absent	Absent	Absent	Absent
Sugar					
Urine	Present +	Present +	Present +	Traces	Absent
Albumin					
Urine RBC	10-15	Absent	Absent	Absent	
	cells/hpf				
Urine	3-5 cells/hpf	2-3 cells/hpf	2-3	4-5 cells/hpf	
Epithelial	-	-	cells/hpf	_	
cells			-		

The serum creatinine levels before and after

Figure No 2 respectively.

treatment is shown in Figure No 1 and

NTNA		Diagnostics	TAL
PATIENT NAME : P LALITHA	CLIENT DATE:	H83PT	,
	CLIENT PATIENT ID :	UHID:757472	
	D: 25/11/2015	DATE OF BIRTH :	
ACCED 25/11/2017 COLL RECEIVE DRAWN : CUENT NAME : FHSL BG ROAD - IPD CUENT NAME :	D: 25/11/2017 06:30	REPORTED : 25/11/2017 08:50	
DRANE : FHSL BG ROAD - IPD	REFERRING DOCT	TOR : DR. RAJANNA SREEDHARA	
		DR. RAJANNA SREEDHARA	
CLENT NAME : FHSL BG ROAD IT D CLENT NAME : CLINICAL INFORMATION : CLINICAL 104818			
PID: 204818 PID: 204818 NS 1 IPD-L6 WARD NS 1 PD-L6 Preliminary			
IPD-L6 With Test Report Status Preliminary	Results	<b>Biological Reference Interval</b>	Units
Test		41.4	
	BIO CHEMISTRY	2	
SERUM	DECULT DEND		
CREATININE, SERUM	RESULT PENDI		
CREATININE CREATININE	9.40	0.50 - 0.90	mg/dL
<sub>CREATININE</sub> ELECTROLYTES (NA/K/CL), SERUM			
	130	Low 136 - 145	mmol/L
SODIUM METHOD : ISE INDIRECT			
CCTLIM	3.63	3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT		No. of the second secon	
CHLORIDE	88	Low 98 - 107	mmol/L
common disease, metabolic acidosis, acute starvation, der	yuration, and with rapid K miusi	I in Addison's disease, hypopituitarism, liver disease. Hyp Hyperkalemia may be seen in end-stage renal failure, hr on.Chloride is increased in dehydration, renal tubular ad a and loss of sodium bicarbonate, diabetes insipidus, ad	renocortical hyper
ommolie dicease, metabolic acidosis, acute starvation, der	ssociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium bicarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion	renocortical hyper
communi disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
common' disease, metabolic acidosis, acute starvatin, den addison's disease, metabolic acidosis, acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a metabolic acidosis, acute renal failure, metabolic alkalosis, congestive heart fa alt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium bicarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion	renocortical hyper
communi disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
communi disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
communi disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
common disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
common disease, metabolic acidosis, acute sarvatini, den addison's disolis), acute renal failure, metabolic acidosis a metabolic acidosis), acute renal failure, metabolic acidosis a alicylate intoxication and with excessive infusion of isotonic salt-losing nephritis, metabolic alkalosis, congestive heart fa	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuis disease, metabolic acidosis, actured interaction of a second se	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
or movies disease, metabolic acidosis, actures, metabolicacidosis, disbolicacidosis), acute renal failure, metabolicacidosis metabolicacidosis), acute renal failure, metabolicacidosis metabolicacidosis, acutera failures, congestive heart fa please visit www.srh Dr.Manjula S J, MD	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
promotions' disease, metabolic acidosis, acture subtraction data metabolic acidosis), acute renal failure, metabolicacido of isotoni alticolar interaction and with excessive influsion of isotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni plate interaction and with excessive influsion of sotoni plate interaction and with excessive influsion of sotoni plate interaction and with excessive influence of sotoni plate interaction and with excessive influence of sotoni plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic act	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
or movies disease, metabolic acidosis, actures, metabolicacidosis, disbolicacidosis), acute renal failure, metabolicacidosis metabolicacidosis), acute renal failure, metabolicacidosis metabolicacidosis, acutera failures, congestive heart fa please visit www.srh Dr.Manjula S J, MD	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
promotions' disease, metabolic acidosis, acture subtraction data metabolic acidosis), acute renal failure, metabolicacido of isotoni alticolar interaction and with excessive influsion of isotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni alticolar interaction and with excessive influsion of sotoni plate interaction and with excessive influsion of sotoni plate interaction and with excessive influsion of sotoni plate interaction and with excessive influence of sotoni plate interaction and with excessive influence of sotoni plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic action of sotonic plate interaction of sotonic action of sotonic act	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, sisease, metabolic acidosis, actue standaudin, adiatabile acidosis, actue real failure, metabolica acidosis), acute real failure, metabolica of social metabolica acidosis), acute real failure, metabolica of social metabolica ideologis, acute real failure, metabolica of social alt-losing nephritis, metabolic alkalosis, congestive heart fa Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, sisease, metabolic acidosis, actue standaudin, adiatabile acidosis, actue real failure, metabolica acidosis), acute real failure, metabolica of social metabolica acidosis), acute real failure, metabolica of social metabolica ideologis, acute real failure, metabolica of social alt-losing nephritis, metabolic alkalosis, congestive heart fa Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
orminuir, disease, metabolic acidosis, actue standaudin, adiato acidi acidosis, actue and relative and acido acidosis, actue and failure, metabolica adiatosis, congestive infusion of isotoni alticlosing nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper
ommunic disease, metabolic acidosis, actue statuatadui, disease dispolic acidosis), actue renal failure, metabolino of social neinolate intoxication and with excessive infusion of social altiboling nephritis, metabolic alkalosis, congestive heart fr Please visit www.srh Dr.Manjula S J, MD Senior pathologist and Lab	issociated with prolonged diarrhe c saline or extremely high dietan ailure, Addisonian crisis, certain	and loss of sodium blcarbonate, diabetes insipidus, ac a and loss of sodium blcarbonate, diabetes insipidus, ac y intake of salt.Chloride is decreased in overhydration, c types of metabolic acidosis, persistent gastric secretion oct+**	renocortical hyper

Figure 1 Showing the Serum creatinine levels before treatment.

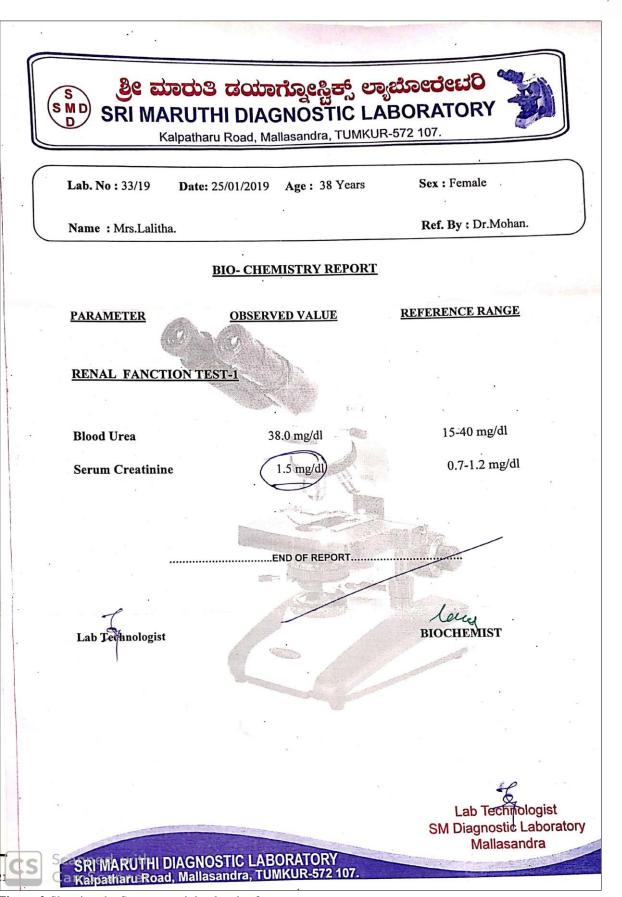
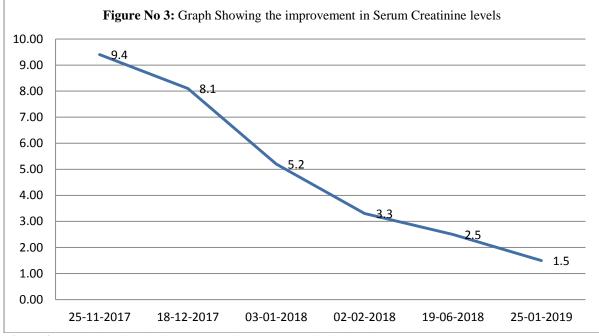


Figure 2 Showing the Serum creatinine levels after treatment



The improvement in Serum Creatinine levels is depicted in the form graph on Figure No 3.



#### Figure 3 Graph Showing the improvement in Serum Creatinine levels

## DISCUSSION

CRF is a progressive loss of renal functions over a period of months or years. CRF can be considered as a *Mutravahasroto vikara* based on its *lakshana, doshas,* and *dushyas* involved. In Ayurveda there is no direct relation and description of this disease in ancient texts. But increase in creatinine levels can be considered as indicator of Mutravahastrotas Dusti. As the Vruka is made from Rakta and Meda so in this disease decrease filtration rate has been occurred due to accumulation of Meda and Rakta Dusti which is carried out by Vata Dosha<sup>8</sup>. Here in this case the patient initially took conventional treatment and did not find satisfactory improvements. And we observed that given Ayurvedic medicines considerably reduced the signs and symptoms of the patient. There was marked improvement in the serum creatinine levels, electrolytes and the Hb levels. This was probably due the reno-protective and nephron-genetic effect of the drugs like Punarnava<sup>9,10</sup> and Gokshura, which was the major part of this treatment plan.

*Gokshuradi guggulu*<sup>11</sup>, the chief ingredient *Gokshura* has diuretic action which is beneficial in CRF to decrease the oliguria. Rasayana properties of Gokshura and *Chandraprabha Vati* help to prevent and



repair the tissue damage of Mutravahasrotas by their antioxidative properties. Guggulu which has the Rasayana and Lekhana<sup>12</sup> property will remove the blockage of the Mutravaha srotas

*Punarnavasam*<sup>13</sup>: Punarnava has fibrinolytic activity, smooth muscle relaxant property<sup>14</sup>. Helps in improving the glomerular filtration rate also removing waste out of body which damages the 
 Table 4 Ingredients of Gokshuradi Guggulu

kidney. Neeri KFT Syrup of AIMIL pharma also contains Punarnava and Gokshura. All these drugs together will remove the blockage, enhances the quality and repairs the damage of renal parenchyma thereby increasing the functioning capacity by increasing the GFR. The ingredients of Gokshuradi Guggulu, Punarnavasam, Neeri KFT<sup>15</sup> are listed in Table No 4, 5, 6 respectively.

Sl No.	Sanskrit Name	<b>Botanical Name</b>	Quantity
1	Shudda Guggulu	Commiphora mukul	336 Grams
Kashaya	Dravyas		
2	Gokshura	Tribulus terrestris	1.34 Kgs
3	Jala	Water	8.06 litres
Churna I	Dravyas		
4	Shunti	Zingiber offinalis	48 grams
5	Maricha	Piper nigrum	48 grams
6	Pippali	Piper longum	48 grams
7	Haritaki	Terminallia chebula	48 grams
8	Vibitaki	Terminallia belerica	48 grams
9	Amalaki	Embila officinalis	48 grams
10	Musta	Cyperus rotundus	48 grams
Table 5 Ir	gredients of Punarnavasam	V1	0
SI No.	Sanskrit Name	Botanical Name	Quantity
1	Pippali	Piper longum	16 grams
2	Shunti	Zingiber offinalis	16grams
3	Maricha	Piper nigrum	16 grams
2 3 4 5	Darvi	Berberis aristata	16 grams
5	Haritaki	Terminallia chebula	16 grams
6	Vibitaki	Terminallia belerica	16 grams
7	Amalaki	Embila officinalis	16 grams
8	Vasaka	Adhatoda vasica	16 grams
9	Erandamoola	Ricinis communis	16 grams
10	Katuki	Picrorhiza kurroa	16 grams
11	Punarnava	Boerhavia diffusa	16 grams
12	Gokshura	Tribulus terrestris	16 grams
13	Kantakari	Solanum xanthocarpum	16 grams
14	Bruhati	Solanum indicum	16 grams
15	Gajapippali	Scindapsus officinalis	16 grams
16	Shushka moolaka	Raphanus sativus	16 grams
17	Duralabha	Fagonia cretica	16 grams
18	Nimba	Azadirachta indica	16 grams
19	Guduchi	Tinospora cordifolia	16 grams



20	Patola	Trichosanthes dioica	16 grams
20	Dhataki	Woodfordia fruticosa	256 grams
21	Madhu	Honey	800 grams
23	Draksha	Vitis vinifera	320 grams
24	Sharkara	Sugar	1.6 kgs
25	Jala	Water	8.1 litres
<b>Fable 6</b> In	gredients of Neeri KFT Syrup for	each 10ml	
Sl No.	Sanskrit Name	Botanical Name	Quantity
Aqueous	extracts derived from:		
1	Punarnava	Boerhaavia diffusa	1000 mg
2	Panchtrin Mool	Classical Ay. Preparation	1000 mg
3	Kasni	Cichorium intybus	600mg
4	Kakamachi	Solanum nigrum	500mg
5	Guduchi	Tinospora cordifolia	500 mg
6	Kamala nala	Nelumbo nucifera	400 mg
7	Palasha	Butea monosperma	300 mg
8	Gokshura	Tribulus terrestris	300 mg
9	Kamala	Nelumbo nucifera	200 mg
10	Shirisha	Albizzia lebbeck	200 mg
11	Rakthachandana	Pterocarpus santalinus	200 mg
12	Haridra	Curcuma longa	200 mg
13	Shigru	Moringa oleifera	200 mg
14	Ushira	Vetiveria zizanioides	150 mg
15	Ananthamoola	Hemidesmus indicus	150 mg
16	Dhanyaka	Coriandrum sativum	100 mg
17	Varuna	Crataeva nurvala	100 mg
18	Tanduliyaka	Amaranthus spinosus	100 mg
19	Amlavetasa	Rheum emodi	100 mg
20	Eravu	Cucumis utilissimus	100 mg
21	Eranda Karkati(Pappaya)	Carica papaya	50 mg
Infusion:			-
22	Kankola	Piper cubeba	100 mg
Juice of:		L	5
23	Ananas (Pineapple)	Ananas comosus	0.5 ml
24	Alabhu	Lagenaria siceraria	0.5 ml
25	Dhanyaka	Coriandrum sativum	0.5 ml
26	Amalaki	Embila officinalis	0.5 ml
Powder:			
27	Shwetha Parpati	Classical Ay. Preparation	100mg
28	Sugar free syp base	- 1	Q.S
29	Excipients		Q.S
	1		~

The Hb levels were improved due to the use of *Abhraloha*. *Abhraloha* also acts as cardioprotective and cardiotonic. Thus we can say that the given Ayurvedic drugs are effective in the management of CRF and completely safe. With such therapeutic management we can avoid hemodialysis, further damage of renal parenchyma and renal transplantation.

## CONCLUSION

In this case study there was marked improvement in the renal function and



general condition of the patient by administration of the above said ayurvedic formulations. Improvement in the serum creatinine. serum electrolytes, Hb percentage, Urine albumin levels were observed during the study. So from this we can conclude that the above said treatment is effective, cost-effective and safe in the management of CRF and can further reduce the requirement of dialysis and renal transplantation. It provides lead to further such large sample studies based on scientific parameters.



## **REFERENCES**

1. Harsh Mohan. (2010). Textbook of Pathology, 6<sup>th</sup> edition. Jaypee Publications, Newdelhi; p654.

2. Bikbow B, Pericon, Remuzzi G (23<sup>rd</sup> May 2018), Disparties in chronic kidney diseases prevalence among males & females in 195 countries: Analysis of the global burden of disease 2016 study. Nephron, 139(4), 313-318. PMID29791905 3. www.en.m.wikipedia.org/wiki/chronic\_ kidney\_disease (Last accessed on 03/11/2019).

4. Stanley Davidson. (2010), Davidson's principles and practice of medicine, 21<sup>st</sup> edition. Churchill livin stone publications. p 433.

5. Harsh Mohan. (2010). Textbook of Pathology, 6<sup>th</sup> edition. Jaypee Publications, Newdelhi; p655.

6. Stanley Davidson. (2010), Davidson's principles and practice of medicine, 21<sup>st</sup> edition. Churchill livin stone publications. p 435.

7. Umesh Khanna. (2009 Jan), The Economics of Dialysis in India. Indian K Nephrol, 19(1): 1-4. PMID20352002

8. Ananthram Sharma. (2012), Sushrutha Samhitha, Vol 2 (Sharirasthana 4<sup>th</sup> chapter Shloka no 31). Chaukhambha Sanskrit Sansthan, Varanasi, p-55. 9. Singh, R.G. et al. (2010), Evaluation of antiprotenuric & renoprotective effect of punarnava (Boerhavia diffusa Linn) in diabetic nephropathy. J Res Edu Indian medicine; 16(1-2): 45-48.

10. Prasanth GS et al. (Apr 2010), A clinical comparative study of the management of chronic renal failure with punarnavadi compound. Ayu; 31(2): 185-92.

11. Srikanta Murthy K R. (2003), A
Treatise on Ayurveda Sharanghadara
Samhitha, (Madhyamakhanda 7/84-87).
Chaukhambha Orientalia Varanasi, p 109.
12. Sharma A P, Sharma G. (1979),
Kaiyadeva Nighantu (Aushada varga
1418), Chaukhambha Orientalia Varanasi,
p14-15.

 Muralidhar Mishra. (1945), Bhaishajya Ratnavali 3<sup>rd</sup> edition, Shotha Prakarana. Nethakumar Press, Lucknow. P176.

14. Pranati Nayak, M Thirunavoukkarasu. (2016 Mar). A review of the plant Boerhaavia diffusa: its chemistry, pharmacology and therapeutical potential. JPHYTO 5(2):83-92.

15. https://www/aimilpharmaceuticals.com /product/neeri-kft. (Last Accessed on 04/11/2019).