Clinical Research

A clinical comparative study of the management of chronic renal failure with *Punarnavadi* compound

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Abstract

India like any other country is facing a silent epidemic of chronic renal failure (CRF)- a facet of the health transition associated with industrialization partly fuelled by increase in sedentary lifestyle, low birth weight and malnutrition. Increasing figures by many folds seen is posing a difficult situation to overcome with respect to economy and health of the working and earning population of the nation. There is an urgent need to explore, highlight new interventions and modify modifiable risk factors as a basis for treatment strategies to prevent the development and progression of CRF. The present study was taken up to evaluate the role of trial formulation tab. Punarnavadi compound in the management of chronic renal failure. This was an open clinical comparative study in controlled circumstances wherein 67 patients were studied for two months in three groups- Group A (allopathic control), Group B (ayurvedic control) and Group C (ayurvedic test). It was a multi-centric study; patients were registered from Anandababa charitable dialysis centre, Jamnagar, Kayachikitsa O.P.D. of I.P.G.T. and R.A. Jamnagar and P.D. Patel Ayurveda hospital, Nadiad. Results were assessed on 15 parameters using Students (paired) 't' test. Group A patients showed comparatively better results in eight parametersweight, platelet count, serum urea, serum uric acid, serum sodium, potassium, chloride and total proteins. Parameter Hemoglobin% showed better results in Group B patients and in Group C patients comparatively better results in six parameters viz.- quality of life (breathlessness, weakness, general functional capacity), total count, serum creatinine and serum calcium - were observed. Throughout the study, trial drug tab. Punarnavadi compound did not show any adverse drug reaction. The results of this study will help in developing a cheap and safe treatment for the management of CRF.

Key words: Chronic kidney disease, chronic renal failure, *Punarnavadi* compound, ayurvedic management.

Introduction

India like any other developing country is facing a silent epidemic of chronic renal failure (CRF-a) facet of the health transition associated with industrialization partly fuelled by increase in sedentary lifestyle, low birth weight and malnutrition. India has very little infrastructural renal care facilities with few centers that too being placed at major cities only. Indian Government spends very less on health each year and patients are supposed to attend its primary health centers. Patients do not attend because in doing so they lose a day's wages. A normal person in India cannot afford this high costing treatment that too for an incurable disease- CRF.

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If still can afford it, these costs have to be borne for life time. This puts an unbearable load on the patient and his/ her family. This is the most important reason why only 2-3% of *Kidney* failure patients in India get treated. Rest prefers an early death so to decrease the financial burden on their kith and kins.^[1] Over the next decade, the number of patients with end-stage renal disease requiring renal replacement therapy is sure to increase by many folds world wide posing a difficult situation to overcome with respect to economy and health of the working and earning population of the nation. There is an urgent need to explore, highlight and modify modifiable risk factors as a basis for treatment strategies to prevent the development and progression of CRF.

Aims and objectives

To study the role of Ayurvedic medicine *Punarnavadi* compound in the management of chronic renal failure in controlled clinical trial.

Materials and Methods

Plan of Study

Presence of chronic *Kidney* disease was established based on the presence of *Kidney* damage and level of *Kidney* function through glomerular filtration rate (GFR), irrespective of the cause of renal failure, according to the K/DOQI guidelines.^[2]

Diagnosed patients of chronic renal failure were registered from OPD of Kayachikitsa department of I.P.G.T. and R.A., Jamnagar, Anandababa Dialysis Centre, Jamnagar and P. D. Patel *Ayurveda* Hospital, Nadiad.

Inclusion criteria

- Diagnosed patients of CRF as per K/DOQI guidelines of any etiology
- Clinically stable patients of stage 1 to 5 as per K/DOQI guidelines

Exclusion criteria

- Stage: patients of any stage requiring dialysis
- Age: below 10 years and above 80 years
- Others: HIV-associated nephropathy and transplant allograph failure

Study design

It was an open study in controlled circumstances in the following manner:

Groups: Patients were divided into three groups *viz*. Group A, Group B and Group C.

Group A: Twenty-eight patients were registered in this group and observed without interfering with their routine allopathic management.

Group B: Fourteen patients were kept on a combination of standard Ayurveda intervention comprising of-

Gokshuradi guggulu	3 tab TD
Varunadi kwatha	40 ml BD
Rasayana churna	3 gm BD
Tab. Uricare	2 TD

Group C: Twenty-five patients of this group received same intervention as in Group B in addition to Tab. *Punarnavadi* compound 4 gm/day in two divided doses with water.

Duration: eight weeks in all groups.

Pathyapathya

Patients of all the groups were advised to follow the causative disease specific diet schedule as mentioned in K/DOQI guidelines.

Criteria for assessment

Investigative parameters

Following pathological and biochemical investigations were carried out in the present study for assessment purpose:

- urine microscopic, albumin, sugar
- hemoglobin %, total count, differential count, platelet count.
- blood sugar- fasting blood sugar/post prandial blood sugar.
- serum creatinine, blood urea, serum uric acid
- serum total proteins
- serum electrolytes Calcium (Ca), Pottasium (K), Sodium(Na), Chloride (Cl)

Criteria for assessment of quality of life

Patients were examined monthly for the assessment of quality of life [Table 1]; data was collected at both starting and the end of duration of the study, collected data was subjected for statistical analysis to reveal the results of the study.

In Group B (ayurvedic control), pre-recorded patient case data were utilized, wherein minimal lab investigations like Hb%, serum creatinine and blood urea only were carried out previously. Same data was used for statistical procedures.

Preparation of Punarnavadi compound

Ingredients of the test drug *Punarnavadi* compound [Table 2] were authenticated botanically through Mr.Vaccharjani (Research officer (Botany), CCRAS unit, AhMedabad) before it was processed in the pharmacy of Gujarat Ayurveda University, Jamnagar. Decoction was prepared from drugs numbered 1 to 6 and after filtration *Shilajit* was added and further boiled in water bath up to semisolid stage was reached. Then powder of drug numbered 7 and 8 were added, mixed well and tablets of 500 mg weight were punched out. Obtained tablets were labeled and stored in a dry air tight container.

In this study apart from tab. *Punarnavadi* compound, other formulations like *Gokshuradi guggulu*, *Varunadi kwatha*, *Rasayana churna* (equal quantity of *Guduchi*, *Gokshura*, *Amalaki*) and Tab. Uricare were used. *Gokshuradi guggulu*, *Varunadi kwatha* and *Rasayana churna* were procured from Sundar Pharmacy attached to J. S. *Ayurveda* College, Nadiad and tab. Uricare having the contents- extracts of *Crataeva nurvala* (80 mg), *Boerhavia diffusa* (80 mg), *Tinospora cordifolia* (80 mg), *Shuddha Shilajit* (80 mg) and *Shuddha Guggulu* (80 mg) was supplied from Patlad Mahal Arogya mandal Pharmacy, Nadiad, Gujarat.

Table 1: Criteria for assessment of	quality of life	е
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Symptom	Scoring
Breathlessness	
Dyspnoea after heavy work but relived soon and upto tolerance	0
Dyspnoea after little work but relived later and upto tolerance	1
Dyspnoea after little work but relived later and beyond tolerance	2
Dyspnoea in resting condition	3
Weakness	
No weakness	0
Slight weakness	1
Feeling weak but can perform daily routines	2
Feeling weak, it is difficult to perform daily routines	3
General functional capacity	
Ability to carry on all usual routines	0
Adequate for normal activity-despite of comfort	1
Limited-only to little	2
Incapacitated-largely or wholly bed ridden	3

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Drugs	Latin name	Part used	Proportion
Punarnava	Boerhavia diffusa	Root	1 part
Gokshura	Tribulus terrestris	Seed	1 part
Varuna	Crataeva nurvala	Bark	1 part
Shigru	Moringa olifera	Bark	1 part
Kusha	Desmostachya bipinnata	Root	2 parts
Kandekshu	Saccharum officinalum	Root	1 part
Guduchi	Tinospora cordifolia	Stem	1 part
Shatavari	Asparagus racemosus	Root	1 part
Shilajit	Asphaltum panjabiunum	-	1 part

Table 2: Ingredients of Punarnavadi compound

Observations

Totally 67 patients in three groups completed the duration of the study, observations made among those patients are as follows:

In this study with respect to history of diabetes, minimum of 2 (3%) patients had a history of more than 25 years, maximum of 9 (13.4%) patients between 10 and 15 years, 4 (6%) patients less than 5 years and 6 (9%) patients between 16 and 20 years. Minimum of 6 (9%) patients had the history of hypertension of more than 11 years; maximum of 16 (23.9%) patients had a history of less than 5 years and 13 (19.4%) patients between 6 and 10 years. Among the different causes, analgesic-induced CRF was reported in 1 (1.5%) patient, BPH in 2 (3%), polycystic *Kidney* disease in 3 (4.5%), renal calculi in 3 (4.5%). Addiction to tobacco among the patients of this study- 20 (29.9%) used to chew and 17 (25.4%) patients were smokers [Figure 1].

Results

Effect on weight- Comparatively significant weight decrease was observed in all the three groups. In Group A weight decreased by 0.27%, in Group B by 1.05% and in Group C 0.79% [Table 3].

Effect on quality of life: In terms of breathlessness, weakness and general functional capacity, quality of life was assessed. Statistically non-significant 15% relief was observed in breathlessness and 8% relief in weakness; however, the treatment did not affect the parameter general functional capacity in Group A patients. Statistically non-significant 50% relief was observed in breathlessness and 18.76% relief in general functional capacity; however, the treatment did not improve the weakness parameter in Group B patients. Comparatively decrease in severity was seen in all three parameters- 80% in breathlessness, 12.5% in weakness and 35.71% in general functional capacity. However, only decrease in breathlessness was statistically significant among Group C patients [Figure 2].

Effect on hematological parameters: In Group A, 5.6% decrease in hemoglobin percentage, 6.73% decrease in total count and 0.44% decrease in platelet count. Treatment marginally decreased hemoglobin content by 1.87% but is statistically nonsignificant in Group B. After treatment, hemoglobin decreased by 4.32%, total count by 11.82%, and platelets by 4.07%. Amongst



Figure 1: Different observations of the study

Table 3: Effect on body weight (in kilograms)	in
Group A, B and C	

Parameter	n	Mean difference ± S.E.M	% change	t	Ρ
Group A	28	0.18 ± 0.32	0.27↓	0.553	-
Group B	14	0.64 ± 0.258	1.05↓	2.498	-
Group C	25	0.48 ± 0.33	0.79↓	1.437	-

statistically significant decrease was seen in hemoglobin and total counts was observed in case of Group C patients [Figure 3].

Effect on bio-chemical parameters: Decrease of 29.39% in urea, 0.43% in total proteins was observed, decrease in all these parameters was statistically non-significant except that of urea. Increase of 3.34% in uric acid, 0.99% in creatinine was observed in Group A. Increase of 7.06% in urea and 8.41% in serum creatinine was observed after the treatment; however, the observed increase was statistically non-significant in Group B patients. Uric acid increased by 24.4% and total proteins by 1.98%. Among these, statistically significant increase was seen in uric acid only. Administration of test drugs did not alter the serum creatinine level [Figure 4].

Effect on serum electrolytes: Levels of serum calcium, potassium, chloride and sodium decreased after treatment by 0.71%, 1.71%, 0.39% and 0.63%, respectively. However, the observed decrease was statistically non-significant in Group A. Statistically non-significant decrease in serum calcium by 2.79%, non-significant increase in serum potassium by 0.09%, serum chloride by 1.77% and serum sodium by 0.8% was observed in Group C [Figure 5].

Effect on average urine albumin and urine sugar in Group A and Group C patients: In Group A, before treatment, average of albumin was 2.5+ and sugar 2.1+, after treatment average of albumin and sugar were 2.6+ and 0.7+ respectively. In Group C, before treatment average of albumin and sugar were 1.9+ and 0.2+, after treatment average of the same were 1.3+ and 0.5+ respectively [Table 4].

Overall results of the study

i.

To assess overall effect, results obtained by Paired 't' test are used and following steps are followed-

After treatment, values obtained from all the parameters (except RBS as all patients were not diabetics and did not have raised RBS value before test) are tabled systematically;

- ii. Best value among the three groups for each parameter is counted as one point;
- iii. At the end of the table, points are totaled for each group individually; and
- iv. Treatment modality of the group obtaining highest number is considered as best among the three groups. The results are shown in Table 5.

Decrease in weight is observed in all the three Groups; comparatively in Group A less decrease in weight is observeda better value of this study. Group C shows superior results in all the three parameters concerned to quality of life, in the blood parameters. Group B has less decrease in Hb% - a fine value in comparison to other two groups, superior decrease in total count is seen in Group C, healthier value of increase in platelet count and decrease in blood urea level is observed in patients of Group A. Serum uric acid is increased in Group A and C, comparatively Group A shows less increase - a good result - serum creatinine has increased in Group A and B, comparatively a fine result is seen in Group C where value has neither increased nor decreased, total proteins has increased in Group C in comparison to Group A, among electrolytes more decrease in serum calcium is in Group C - a good value - good values of serum potassium, serum chloride and serum sodium are seen in Group A wherein they are deceased simultaneously have raised in Group C.

Overall, in this study set-up among 15 different parameters Group A is showing good results in eight parameters, Group B in one parameter and Group C in six parameters.

Discussion

In this study population, 46 patients were not having diabetes



Figure 2: Effect on quality of life in Group A, B and C patients



Figure 4: Effect on serum- biochemical parameters in Group A, B and C patients

and rest 21 patients had diabetes with variable duration. Two patients had a history of diabetes of more than 25 years, 6 patients of 16 to 20 years, 9 patients of 10 to 15 years, 4 patients had less than 5 years history. Out of 67 patients, 32 patients were not hypertensives, more than 11 years history was present in 06 patients, 6 to 10 years in 13, less than 5 years in 16 patients. More than 50% of the study group reported for presence of hypertension indicating the severity of the disease in general population.

Good percentage of the patients of the study population had diabetes; same is true in general population with shortly India becoming the capital of diabetes. Diabetes and hypertension are the important causes for renal failure and their incidence is raising world wide.^[5-6] Irrespective of the underlying mechanisms, elevated levels of blood glucose ignite a vicious cycle of metabolic disturbances within the intracellular and extracellular environment that, if left unchecked, lead to a broad array of complications in macro-vessel and micro-vessel structures. A particular target in this setting is the *Kidney*.^[7]

Table 4:	Before and after trial, average of urine
albumin	and sugar of Group A and Group C
natients	

patiente				
Urine		Gro	ups	
microscopy		4	()
parameter	BT	AT	BT	AT
	average	average	average	average
Albumin	2.5+	2.6+	1.9+	1.3+
Sugar	2.1+	0.7+	0.2+	0.5+



Figure 3: Effect on hematological parameters in $\operatorname{Group} A,B$ and C patient



Figure 5: Effect on serum- electrolytes in Group A and C patients

Parameter	Group A	Group B	Group C
	(Allopathic control)	(Ayurvedic control)	(Ayurvedic test)
	(%) of change	(%) of change	(%) of change
Weight (kilograms)	0.27↓	01.05↓	0.79↓
Quality of life			
Breathlessness	15.38↓	50.00↓	80.00↓
Weakness	07.69↓	00.00	12.50↓
General functional capacity	00.00	18.76↓	35.71↓
Blood and serum parameters			
Hb (gm %)	05.60↓	01.87↓	04.32↓
Total count (/c.mm)	06.73↓	-	11.82↓
Platelets (/c.mm)	00.44↑	-	04.07↓
Urea (mg %)	29.39↓	07.06↑	15.34↓
Uric acid (mg %)	03.34↑	-	24.4↑
Creatinine (mg %)	00.99↑	08.14↑	00.00
Total proteins (gm/dl)	00.43↓	-	01.98↑
Calcium (mg/dl)	00.71↓	-	02.79↓
Potassium (mmol/l)	01.71↓	-	00.09↑
Chloride (mmol/l)	00.39↓	-	01.77↑
Sodium (mmol/l)	00.63↓	-	00.80↑
Overall assessment			
Allopathic control	08	-	-
Ayurvedic control	-	01	-
Ayurvedic test	-	-	06

Hypertension has particular relevance to patients with chronic *Kidney* disease. In them, elevated blood pressure plays a dual role: it is both the result of parenchymal damage within the *Kidney* and a provocator of further deterioration in *Kidney* function.^[8]

Many diseases may produce renal damage and lead to chronic renal failure. In the study population, analgesic induced renal failure was noted in one patient, BPH in two, poly-cystic Kidney disease in three and renal calculi in three patients. Analgesics and non-steroidal anti-inflammatory drugs are among the most widely and frequently used drugs. Many studies^[9,10] have suggested an association between chronic ingestion of analgesics and Kidney disease. It is characterized by Kidney papillary necrosis and chronic interstitial nephritis that leads to insidious onset of progressive Kidney failure. Incidence of this variety nephropathy is usually seen in elderly in whom musculoskeletal aches are more common. BPH is a post renal cause for renal failure. Mechanical obstruction of hypertrophied prostate mechanically obstructs urine flow by constricting the urethral hollow. This initiates retrograde flow of urine causing hydronephrosis and later renal failure. Such conditions are possible in elderly with delayed diagnosis of hypertrophied prostate. PCKD, an autosomal dominant polycystic Kidney disease, is a common inherited disorder, characterized by the formation of fluid-filled cysts in both kidneys that leads to progressive renal failure. The factors that affect disease progression in patients with autosomal dominant polycystic Kidney disease are unclear.[11] Boys and girls have equal chance of inheriting the disease.^[12] Calculi cause obstruction to urine flow either in ureter or at the level of urethra. Calculi formed inside the kidneys at times destroy the nephron mass locally if large enough can induce renal failure. Incidences are common in regions where people drink hard water.

In this study, 30 patients were not addicted to tobacco in any of its form. Twenty patients used to chew and 17 patients smoked among the total of 67 patients. Smoking increases the risk of micro-albuminuria; shortens the interval between onset of diabetes and the start of albuminuria or proteinuria; accelerates the rate of progression from micro-albuminuria to persistent proteinuria; and pathologically promotes the progression of diabetic nephropathy to ESRD.^[13] Tobacco chewers may be associated with albuminuria and abnormal renal function in non diabetic and non hypertensive people in future.^[14]

As observed, among 67 patients of chronic renal failure 19 patients had sound sleep and a maximum of 48 patients had disturbed sleep. Poor sleep is common in renal failure patients, exact reason is not known. Quality of sleep decreases in the early stages of CKD and does not appear to be associated with the subsequent degree of renal failure. It is hypothesized that the causes of decreased quality of sleep in renal failure patients are not specific to chronic renal illness and probably involve psychiatric disorders, such as depressed mood.^[15]

In both Group A and C, albumin and sugar were present before and after test. Explanation says that greater proteinuria indicates a more severe glomerulopathy, and this accounts for the faster GFR decline.^[16] Recently, it has become clear that proteinuria, particularly when heavy and nonselective, can be nephron-toxic through a variety of mechanisms.^[17-19] There is strong evidence that proteinuria is both a marker for and a mechanism of *Kidney* disease progression. In Group B, comparatively decrease in urine albumin level was seen indicating a better prognosis.

In patients of Group A and C during the study period epithelial cells, RBCs, pus cells, WBCs and granular casts, hyaline casts were present in urine microscopy. Increased number of epithelial cells may indicate nephrosis or amyloids, RBC presence is indicating glomerular bleeding and presence of WBC is indicating the presence of infection in urinary tract. Among all, WBCs were present in more number of patients indicating the active infection present in the kidneys. Most patients were of diabetes, presence of granular casts, hyaline casts is indicating the same. Granular casts are formed from cell debris and are seen in pyelonephritis, acute tubular necrosis.^[20] Hyaline casts presence indicates damage to capillary membrane of glomerulii, which allows proteins to leak.^[21]

Results of the clinical study obtained are discussed under different parameters of assessment accordingly:

Weight- In the present study, patients did not show any signs of free fluid and in all the three groups weight decreased after treatment; however, this decrease was non-significant statistically. This is the actual body weight which has decreased. Likely reasons are the strict diet restrictions, decreased appetite due to disease proper and up to certain extent the psychological stress. Continued abstinence makes the patient *Kshaama*, next *Ksheena* lastly *Krisha* with actual decrease in *Shareera Bala* (Chakrapani, Ca. Sa.Chi.11/55).

Quality of life- Breathlessness, weakness and general functional capacity:

"One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is caring for the patient."- Frances Weld Peabody, (1881 to 1927).^[22] In this study, comparatively better values for the parameter quality of life are seen in Group B and C. This might be due to the strong anti-oxidant and free radical scavenging activities i.e. the *Rasayana* effect of the Ayurvedic drugs (Ca.Sa.Chi.1/1/7) like increase in Ayu (increases quantity and quality of life), increases *Deha Indriya Bala* (strengthens physical body and sense organs) used in these two groups. Even on oral questioning, patients were heard saying they experience a sense of well being after consuming the test drug *Punarnavadi* compound.

HB% and platelet count- Anemia is one of the major complications in CRF. Decreased erythropoietin production results primarily from destruction of renal parenchyma. Due to "uremic" toxin, red cell survival is shortened with a mild to moderate decrease in red cell life span. Bone marrow space fibrosis occurs resulting in decreased erythropoiesis. Decreased platelet count increases the bleeding tendency further decreasing blood volume and its cells. Hb% decreased in all the patients, whereas platelet count increased in Group A patients only. This might be due to the erythropoietin injections they were injected with. As a life saving measure, these injections could be used in patients of CRF on Ayurvedic managements. Bone marrow is Sarakta Meda (Su.Sa.Sha.4/12-16) as commented by Vaidya Ranjith Rai Desai and produces red blood cells. Sarakta Meda in particular can be considered as a Dushya for the complication anemia as is evident in CRF pathology.

Serum urea- Human Kidney excretes nitrogenous wastes in the

form of urea through urine- an evolutionary adaptation. Hypofunctioning of *Kidney* increases its concentration in blood. Urea level comparatively decreased more in Group A, it might be because of the strong diuretics administered. This activity even though present in *Punarnavadi* compound and rest of the Ayurvedic formulations comparatively is less strong, hence might have lead to increase in urea.

Uric acid- It is the end product of purine metabolism. Urate is excreted via two routes- 1/3 secreted into the gut and 2/3 through renal excretion. Hyper-uricemia can result from high intake of purine-rich foods, high fructose intake and impaired excretion by the kidneys. Since patients were on strict diet, increase in the level of uric acid is due to the decreased functioning of kidneys. Group A shows less increase in comparison to Group C, again it might be due to the strong diuretics used in that group.

Serum creatinine- It is determined both by *Kidney* function and muscle mass; patients with low serum creatinine and low muscle mass may have significant impairment of *Kidney* function. Conversely patients with high muscle mass may have serum creatinine above normal without impairment of *Kidney* function. Comparatively, Group B has shown more increase in creatinine level and simultaneously has shown more decrease in weight. This decrease in weight might have increased the creatinine level. Cause for decrease could be decreased nourishment.

Serum total proteins- Plasma protein changes indicate the inflammation or tissue damage. Comparative increase in total proteins in Group C shows the on going inflammatory process even after completion of treatment duration, indicating the decreased efficacy of Group C medications in controlling inflammatory process.

Serum electrolytes- Calcium, potassium, chloride and sodium. Group A has shown decrease in all the four serum electrolytes in comparison to Group C wherein only serum calcium has decreased. This might be due to the better diuretic action of allopathic drugs.

Overall, in this study set-up among 15 different parameters, Group A is showing good results in eight parameters, Group B in one parameter and Group C in six parameters.

On putting together the results and observations, an attempt to understand the probable mode of action of trial drug tablet *Punarnavadi* compound is being made as follows:

Action of any drug is explained on the basis of *Rasa*, *Guna*, *Veerya*, *Vipaka* and *Prabhava*. As evident from the texts, most of the drugs have *Madhura* and *Tikta Rasa* and action of these two *Rasa* are to pacify *Pitta Dosha*. *Laghu*, *Ushna*, *Snighda* are the predominant *Guna* present, amalgamation of all these *Guna* acquires a *Tridoshahara* property. *Veerya* of some drugs is *Sheeta* whereas of rest are *Ushna*, in combination this acts as *Tridoshahara*. Most of the drugs have *Madhura Vipaka*, this is again known to decrease *Pitta Dosha*.

In total *Punarnavadi* compound is predominantly packed with *Pitta Shamaka Rasa*, *Tri-doshahara Guna*, *Tri-doshahara Veerya*, *Pitta Shamaka Vipaka*. This combination makes it a potent *Tridoshahara* mainly targeting *Pitta Dosha*. On the other hand, chronic renal failure has *Pitta* dominant *Tridosha* in its pathology, hence probably it can be postulated that *Punarnavadi* compound shows its action by controlling *Pitta* and *Vata* followed by rest of the *Dosha* avoiding them from further aggravation thus slowing the pace of disease. *Tikta Rasa* present in *Punarnavadi* compound brings *Niraamata* and increases *Jatharagni*, another useful function recommended in renal failure wherein Agni Mandya is invariably present. *Tikta Rasa* and *Rooksha Guna* help in drying of *Ama*, bring out a clear *Rasa Dhatu* and clear the Srotus, further helping in good circulation making way for proper *Dhatu* nourishment. *Tikta Rasa* is chosen in the *Vyadhis* involving *Rakta Dhatu* here in renal failure *Rakta* is the earliest *Dhatu* to be affected.

Punanava, Guduchi, Gokshura, Shatavari, Shilajit used come under the list of Rasayana drug. Especially Punarnava, Gokshura, Shilajit are recommended exclusively in the disorders of Mootravaha Samsthana. These drugs should be accepted as Naimittika Rasayana for Kidney and other organs of Mootravah Srotas. These are drugs by their virtue impart the Rasayana properties which was evident in clinical study wherein patients felt increase in their Jatharagni, quantity and quality of sleep, sense of well being, increase in functional capacity, along with decrease in disease features up to certain extent. Modern researches have shown ample evidence that Ayurveda Rasayana drugs bear the property of anti-oxidant and work as free radical scavengers. Even plenty of research works in the field of phyto-chemistry have exposed the rich anti-oxidant activity and free radical scavenging properties present in these drugs.

Clinical study showed very good results in the groups administered with Ayurvedic formulations in comparison to Allopathic management. Again the group administered with *Punarnavadi* compound has shown a very good result by improving the quality of life of patients. Lastly, gathering data from different observations, *Punarnavadi* compound can be concluded as a drug having a very potent anti-oxidant property against uremic toxicity in patients of chronic renal failure along with mild nephro-protective activity without any adverse drug reactions.

Conclusion

- Punarnavadi compound showed promising results in parameters pertaining to quality of life.
- On blood and bio-chemical parameters, *Punarnavadi* compound showed marginally better results.
- At any corner of the study, *Punarnavadi* compound did not show any adverse effects, so it is safe for human use.
- Further long duration studies are needed to observe exact drug action.

References

- 1. KherV.End stage renal disease in developing countries. Kidney Int 2002;62:350-62.
- National Kidney Foundation. K/DOQI clinical practice guidelines for chronic Kidney disease: Evaluation, classification, and stratification. Am J Kidney Dis 2002;39:S1-266.
- Wendt T, Tanji N, Guo J, Hudson BI, Bierhaus A, Ramasamy R, et al. Glucose, glycation, and RAGE: Implications for amplification of cellular dysfunction in diabetic nephropathy. J Am Soc Nephrol 2003;14:1383-95.
- El-Atat FA, Stas SN, McFarlane SI, Sowers JR. The relationship between hyperinsulinemia, hypertension and progressive renal disease. J Am Soc Nephrol 2004;15:2816-27.
- U. S. Renal Data System. USRDS 2003 15th Annual Data Report: Atlas of endstage renal disease in the United States. Bethesda (MD): National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. 2003.
- Steinberg HO, Chaker H, Leaming R, Johnson A, Brechtel G, Baron AD. Obesity/ insulin resistance is associated with endothelial dysfunction. Implications for the syndrome of insulin resistance. J Clin Invest 1996;97:2601-10.
- Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: Analysis of worldwide data. Lancet 2005;365:217-23.
- Evidence Report: Appropriate Patient Preparation for Renal Replacement Therapy, Prepared for the Renal Physicians Association, Duke Evidence-based Practice Center, Center for Clinical Health Policy Research, 2200 W. Main Street, Suite 220, Apr 11, 2002.
- BennettWM, DeBroe ME.Analgesic nephropathy a preventable renal disease. N Engl J Med 1989;320:1269.
- Pintér I, Mátyus J, Czégány Z, Harsányi J, Homoki M, Kassai M, et al. Analgesic nephropathy in Hungary: The HANS study. Nephrol Dial Transplant 2004;19:840-3.
- Peters DJ, Breuning MH. Autosomal dominant polycystic Kidney disease: Modification of disease progression. Lancet 2001;358:1439-44.
- Available from: http://www.pkdcure.org and http://www.kidney.org/atoz/ pdf/polycystic.pdf Kansas: Polycystic Kidney Disease Foundation; National Kidney Foundation, 30 East 33rd Street. New York: NY 10016, (800); 2003. p. 622-9010
- Hansen HP, Rossing K, Jacobsen P, Jensen BR, Parving HH. The acute effect of smoking on systemic haemodynamics, Kidney and endothelial functions in insulin-dependent diabetic patients with microalbuminuria. Scand J Clin Lab Invest 1996;56:393-9.
- Shah H. Incidence of Micro-Albuminuria in tobacco chewers. Indian J Clin Biochem 2005;20:189-91.
- Iliescu EA, Yeates KE, Holland DC. Quality of sleep in patients with chronic Kidney disease. Nephrol Dial Transplant 2004;19:95-9.
- Keane WF. Proteinuria: Its clinical importance and role in progressive renal disease. Am J Kidney Dis 2000;35:S97-105.
- Hebert LA, Wilmer WA, Falkenhain ME, Ladson-Wofford SE, Nahman NS Jr, Rovin BH. Renoprotection: One or many therapies? Kidney Int 2001;59:1211-26.
- Taal MW, Brenner BM. Renoprotective benefits of RAS inhibition: From ACEI to angiotensin II antagonists. Kidney Int 2000;57:1803-17.
- 19. Zoja C, Morigi M, Remuzzi G. Proteinuria and phenotypic change of proximal tubular cells. J Am Soc Nephrol 2003;14:S36-41.
- Gupta LC, Chauhan RD, Gupta A. Interpretation of Common Investigations. In: Gupta LC, ed. 5th ed. New Delhi: Jaypee Publications 2006. p. 35.
- Kim Ah. Macleod's Clinical Examination. In: Graham D, editor. 11th edition. Toronto: Elsevier Churchill Livingstone Publication; 2005. p. 194.
- 22. Peabody FW The care of the patient. JAMA 1927;88:877-82.

हिंदी सारांश

वृक्क की जीर्ण कार्य अक्षमता में पुनर्नवादि योग के प्रभाव का तुलनात्मक चिकित्सकीय अध्ययन

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विभिन्न कारणों से भारत में वृक्क की जीर्ण कार्य अक्षमता (क्रोनिक रिनल फेल्युअर) के रूग्णों की संख्या बढती जा रही है। इस व्याधि के प्रतिबंध एवं चिकित्सा में लाभदायी उपलब्धियाँ जरूरी हैं। प्रस्तुत चिकित्सकीय अध्ययन में पुनर्नवादी योग वटी का क्रोनिक रिनल फेल्युअर में प्रभाव देखा गया। कुल ६७ रूग्णों को पंजीकृत करके तीन वर्गसमूहों में बाँटा गया। समूह ''अ''में आधुनिक चिकित्सा, समूह ''ब'' में पारम्परिक आयुर्वेदिक चिकित्सा एवं समूह ''क'' में आयुर्वेदीय पारम्परिक चिकित्सा के साथ आयुर्वेदीय औषधी पुनर्नवादि योग वटी दी गयी। परिणामस्वरुप समूह ''अ'' में तुलनात्मक रुप से शारीरिक भार, प्लेटलेट का उंट, सिरम यूरिआ, यूरिक एसिड, सोडिअम-पोटॅशिअम क्लोराइड एवं टोटल प्रोटिन्स् पर अच्छे परिणाम मिले। हिमोग्लोबिन की मात्रा में समूह ''ब''की चिकित्सा से वृद्धि हुई। तथा समूह ''क'' की चिकित्सा से जीवनमान सुधार, टोटल काउंट, सिरम क्रिएटीनिन, सिरम केल्सियम, (श्वासकष्टता, दौर्बल्य, सामान्य कार्यक्षमता) में अच्छा सुधार पाया गया। इस चिकित्सा क्रम में पुनर्नवादी योग वटी से कोई भी दुष्परिणाम नहीं देखे गये।