ISSN2515-8260 Volume 09,Issue 01,2022

Clinical profile of patients of chronic kidney disease undergoing regular twice weekly maintenance hemodialysis

[1]Dr.Raghu Nandan,[2]Dr.Anil Kumar,[3]Dr.Vinay Durgad

[1]Assistant Professor, Department of General Medicine, Vydehi Institute of Medical Sciences and Research Centre, Bangalore, Karnataka, India
[2,3]Assistant Professor, Department of General Medicine, East Point College of Medical Sciences and Research Centre, Bangalore, Karnataka, India

Corresponding Author: Dr.Vinay Durgad

Abstract
With increasing life expectancy and prevalence of life style diseases, United States (US) has seen a 30% increase in prevalence of chronic kidney disease (CKD) in the last decade. Unfortunately, from India there is no longitudinal study and limited data on the prevalence of CKD. Chronic Kidney Disease was diagnosed when at least one of the evidence of chronicity like documented renal failure for >3 months, bilateral small kidneys, histological evidence of chronicity or GFR < 30 ml/min for 3 or more months was present.A pre informed written consent was obtained from every case before inclusion in the study. The clinical and demographic profile was recorded in a specially designed proforma. On ultrasonogram bilateral kidneys of group A, 12 patients had bilateral contracted kidneys which correspond to 60% of the study patients indicative of ESRD. 6 patients had normal or slightly enlarged kidney size suggestive of diabetic nephropathy (30%). Findings of ultrasonogram bilateral kidneys in group B revealed 11 patients had bilateral contracted kidneys which correspond to 55% of the study participants indicative of ESRD. 5 patients had normal or slightly enlarged kidney size (25%) and 1 patient was having bilateral enlarged kidneys with multiple cysts of varying sizes suggestive of autosomal dominant polycystic kidney disease (5%).

Keywords: Chronic kidney disease, autosomal dominant polycystic kidney disease, clinical profile

Introduction

With increasing life expectancy and prevalence of life style diseases, United States (US) has seen a 30% increase in prevalence of chronic kidney disease (CKD) in the last decade. Unfortunately, from India there is no longitudinal study and limited data on the prevalence of CKD.

An Indian population-based study determined the crude and age-adjusted ESRD incidence rates at 151 and 232 per million population, respectively[1].If validated in other parts of this region, it would mean that about 220,000-275,000 new patients need RRT every year in this part of the world. It is estimated that there are about 55,000 patients on dialysis in India and the dialysis population is growing at the rate of 10-20% annually.

In western countries, diabetes and hypertension account for over 2/3rd of the cases of CKD[2].In India too, diabetes and hypertension today account for 40-60% cases of CKD[3].As per recent Indian Council of Medical Research data, prevalence of diabetes in Indian adult population has risen to 7.1%, (varying from 5.8% in Jharkhand to 13.5% in Chandigarh) and in urban population (over the age of 40 years) the prevalence is as high as 28%[4, 5].Likewise the reported prevalence of hypertension in the adult population today is 17% (14.8% from rural and 21.4% from urban belt). A similar prevalence of 17.4% has been reported by Panesar *et al*. (in the age group of 20-59 years) even from slum-resettlement colony of

Delhi[6, 7].With rising prevalence of these diseases in India, prevalence of CKD is expected to rise and obviously this is the key target population to address.

A study from a rural belt of Karnataka showed that the study population had a mean age of 39.88 ± 15.87 years with 3.82% prevalence of diabetes and 33.62% of hypertension. and 6.3% prevalence of CKD stage 3. Modi and Jha[8] reported an age-adjusted incidence of end-stage kidney disease (ESKD) as 229/million population. This is more than double of what has been believed (100/million) over a long time. Agarwal *et al*.[9]studied south Delhi urban population and reported stage 3 prevalence of 0.785%. Singh *et al*.[10] studied urban and semi urban population of Delhi. They had 31.2% hypertensives and 7.3% diabetics in the screened population. 4.2% population was found to be suffering from stage 3 CKD.

In a recently published (2013) Screening and Early Evaluation of Kidney Disease [SEEK] study[11] the mean age of the population was 45.22 ± 15.2 years.They found the prevalence of CKD as 17.2% with stage 1, 2, 3, 4, 5 as 7%, 4.3%, 4.3%, 0.8% and 0.8% respectively. 43.1% of their cohort had hypertension and 18.8% had diabetes.

Methodology

A prospective study was done on forty adult patients of chronic kidney disease undergoing regular twice weekly maintenance hemodialysis for four hours per session as outpatient or as inpatient in Nephrology Department.

Chronic Kidney Disease was diagnosed when at least one of the evidence of chronicity like documented renal failure for >3 months, bilateral small kidneys, histological evidence of chronicity or GFR < 30 ml/min for 3 or more months was present.

A pre informed written consent was obtained from every case before inclusion in the study. The clinical and demographic profile was recorded in a specially designed proforma. The basic biochemical investigations of each case was recorded at 0, 1, 2 and 3 months.

103 Patients of CKD who were undergoing regular maintenance hemodialysis in nephrology department in this institute were screened. All these patients were receiving injection Recombinant Human Erythropoietin (rHuEPO) 4000 I.U. subcutaneously twice weekly and injectable iron in a dose of 100 mg/week following dialysis session. The patients were observed for one month and rise in hemoglobin values were seen for each patient. Out of these 103 patients, 61 were found to have inadequate rise in Hb (< 1g/dl rise in Hb in one month). The dose of erythropoietin was increased to 6000 I.U. S/C twice weekly and response was seen after one month. 8 patients showed good response to increased dosage of erythropoietin and were excluded from the study. One patient developed lower G.I. bleeding due to haemorrhoids and was also excluded. So, final group of 52 patients were labelled as erythropoietin resistant. These 52 patients were randomly divided into two groups A and B. Group A included 26 patients and Group B included 26 patients. Group A was givenpegylatederythropoietin(0.6 mcg/kg bodyweight, s/c) onceintwoweeksandi.viron 100 mg/week, afterthehemodialysissession, for 3 months. Group B wasgivendarbepoetinalfa(0.45 mcg/kg body weight, s/c) onceweeklyandi.viron100 mg/weekafterthehemodialysissessionfor3months.

Out of these 52 patients, 12 patients couldn't complete the study. 5 patients (2 in group A and 3 in group B) left the study in between due to some unknown causes and 7 patients (4 in group A and 3 in group B) expired during the study. So finally 40 patients (20 in each group) completed the study.

Results

The study included 20 patients with age range of 23-70 years in group A and 32-80 years in group B. The mean age, weight, height and BMI in group A were 46.4±14.65 years, 57.05± 9.84 kg 162.95±8.99 cm and 21.46±2.32 kg/m^2, respectively. In group B mean age, weight, height and BMI were 46.3±14.98 years, 60.05±6.76 kg, 166.6±7.34 cm and 21.62±1.93 kg/m^2, respectively. The parameters were comparable in the two groups and the difference was not statistically significant (p >0.05).

Table 1: Baseline profile of patients

Sr. No.	Parameters	Group A	Group B	P value
1	Age (in years)	46.4±14.65	46.3±14.98	0.333
2	Weight (in kg)	57.05±9.84	60.05±6.76	0.817
3	Height (in cm)	162.95±8.99	166.6±7.34	0.368
4	Body Mass Index (kg/m^2)	21.46±2.32	21.62±1.93	0.274

The gender analysis revealed males predominating in both the groups i.e. 12 (60%) in group A and 14 (70%) in group B. Females were 8 (40%) in group A and 6 (30%) in group B.

Table 2: Gender of patients

Sex	Group A		Group B	
	Number	% of patients	Number	% of patients
Male	12	60%	14	70%
Female	8	40%	6	30%

Table 3: Baseline clinical parameters

Clinical parameters	Group A		Group B	
	Number	% of patients	Number	% of patients
Pallor	20	100%	20	100%
Breathlessness	16	80%	15	75%
Pedal Edema	14	70%	13	65%
Jugular Venous Pressure	8	40%	7	35%
Clubbing	0	0%	0	0%
Nausea and vomiting	9	45%	11	65%
Seizures	4	20%	5	25%
Cyanosis	1	5%	3	15%

Clinical parameters at baseline revealed 14 patients had pedal edema and 8 patients had raised jugular venous pressure in group A, whereas in group B, 13 patients had pedal edema and 7 patients had raised jugular venous pressure. All patients in both groups were having pallor, but none of the them had clubbing. In group A, 16 patients were having breathlessness and in group B, 15 patients were having breathlessness.

In the group A patients, electrocardiographic findings of left ventricular hypertrophy and strain pattern were seen in 12 patients suggestive of hypertension out of which 4 patients had Q wave and ST-T wave changes/bundle branch block suggestive of ischaemic heart disease. 3 patients had tall tented 'T' waves suggestive of hyperkalemia. In the group B participants, electrocardiographic findings of left ventricular hypertrophy and strain pattern were seen in 12 patients out of which 3 patients had Q wave and ST-T wave changes/bundle branch block. 4 patients had tall tented 'T' waves. 5 patients ingroup A and6 patientsin groupBhad normal electrocardiogram and 4 patients in each group had prolonged QT_c interval suggestive of low calcium levels.

Table 4: Electrocardiographic findings

		Group A		Group B	
Sr. No.	ECG Parameters	No. of patients	% of patients	No. of patients	% of patients
1.	Left ventricular hypertrophy or 'strain' pattern	12	60%	12	60%
2.	Tall tented 'T' waves	3	15%	4	20%
3.	QT_c Prolongation	4	20%	4	20%
4.	Q wave and ST-T wave changes orbundle branch	4	20%	3	15%

	block suggestive of ischemia				
5.	Normal Graph	5	25%	6	30%

Chest X-ray findings in group A revealed cardiomegaly in 11 patients (55%), non-homogenous opacity in left upper zone in 2 patients suggestive of old healed koch's lesion (10%) andnormal chest skiagrams in 5 patients (25%). Chest X-ray findings in group B revealed cardiomegaly in 12 patients (60%), non-homogenous opacity in left upper zone in 4 patients (20%) andnormal chest skiagrams in 6 patients (30%). Pleural effusion in 4 patients (20%) and pulmonary edema in 3 patients (15%) was present in each group.

Table 5: Chest X-Ray findings

Sr. No.	Parameters	Group A		Group B	
		No. of patients	% of patients	No. of patients	% of patients
1.	Cardiomegaly	11	55%	12	60%
2.	Pleural Effusion	4	20%	4	20%
3.	Pulmonary Edema	3	15%	3	15%
4.	Non homogenous opacity in upper zone	2	10%	1	5%
5.	Normal chest X-Ray	5	25%	6	30%

On ultrasonogram bilateral kidneys of group A, 12 patients had bilateral contracted kidneys which correspond to 60% of the study patients indicative of ESRD. 6 patients had normal or slightly enlarged kidney size suggestive of diabetic nephropathy (30%). Findings of ultrasonogram bilateral kidneys in group B revealed 11 patients had bilateral contracted kidneys which correspond to 55% of the study participants indicative of ESRD. 5 patients had normal or slightly enlarged kidney size (25%) and 1 patient was having bilateral enlarged kidneys with multiple cysts of varying sizes suggestive of autosomal dominant polycystic kidney disease (5%). 3 patients in group A (15%)and4patientsingroupB (20%) had renal calculi with or without hydronephrotic changes suggestive of obstructive uropathy. All the patients were having bilateral grade-II or grade-III increase in echogenicity suggestive of chronic renal parenchymal disease.

Table 6: Ultrasonographic findings of bilateral kidneys

		Group A		Group B	
Sr. No.	Bilateral kidney size	No. of patients	% of patients	No. of patients	% of patients
1.	Bilateral contracted kidneys	12	60%	11	55%
2.	Bilateral normal or slightly enlarged kidneys	6	30%	5	25%
3.	Renal Calculi with or without hydronephrosis	3	15%	4	20%
4.	Polycystic kidneys	0	0%	1	5%

Table 7: Etiology of CKD

		Group A		Group B	
Sr. No.	Cause of Chronic Kidney Disease	No. of patients	% of total patients	No. of patients	% of total patients
1.	Hypertensive nephropathy	7	35%	7	35%
2.	Diabetic nephropathy	6	30%	5	25%
3.	Chronic glomerulonephritis	3	15%	3	15%
4.	Chronic pyelonephritis	1	5%	1	5%
5.	Autosomal dominant polycystic kidney disease	0	0%	1	5%
6.	Obstructive uropathy	3	15%	3	15%

The etiology of CKDshowed hypertension to be the most common cause of CKD (7 patients in group A and 7 in group B) followed by diabetic nephropathy (6 and 5 patients in each

group respectively), chronic glomerulonephritis (3 in group A and 3 in group B) and chronic pyelonephritis (1 in group A and 1 in group B). 3 patient in each group had obstructive uropathy. Autosomal dominant polycystic kidney disease was present in 1 patient of group B.

Discussion

CRP is considered the prototypical acute-phase reactant in man. CRP was discovered by Tillet and Francis, named for its capacity to precipitate the somatic C-polysaccharide of Streptococcus pneumoniae andwas the first acute-phase protein to be described and is an exquisitely sensitive systemic marker of inflammation and tissue damage.

CRP belongs to the pentraxin family of calcium dependent ligand-binding plasma proteins, the other member of which in humans is serum amyloid P component (SAP). CRP gene is located on chromosome 1q21-q23. Plasma CRP is produced by hepatocytes although other sites of local CRP synthesis have been suggested. CRP rises above normal within 6 hours and peaks at 48 hours. The plasma half-life of CRP is about 19 hours and is constant under all conditions of health and disease, so that the sole determinant of circulating CRP concentration is the synthesis rate, which thus directly reflects the intensity of the pathological process stimulating CRP production. There are two different tests of CRP. The standard test measures a much wider range of CRP levels but it is less sensitive in lower ranges. The highly sensitive CRP (Hs-CRP) test can more accurately detect lower concentration of protein (it is more sensitive) which makes it more useful than CRP test in predicting a healthy person's risk for cardiovascular diseases. In most disease, the circulating value of CRP reflects ongoing inflammation and/or tissue damage much more accurately than other laboratory parameters of the acute phase response. The CRP concentration is thus a very useful nonspecific biochemical marker of inflammation, measurement of which contributes importantly to screening for organic disease, monitoring of the response to treatment of inflammation and infection and detection of intercurrent infection[12].

Elevated CRP levels have been described in a significant proportion of end-stage-renal disease patients on hemodialysis or peritoneal dialysis. About one-third of patients with chronic renal failure have serum CRP concentration > 10 mg/l. In healthy men, high CRP level has been identified as a risk factor for cardiovascular disease. As occurs in the general population, prospective studies point to a correlation between CRP plasma levels and overall cardio-vascular mortality also in end-stage-renal disease patients[13].

Tumor necrosis factor (TNF-α) is an adipokines involved in systemic inflammation and is a member of a group of cytokines that stimulate the acute phase reaction. It is produced chiefly by activated macrophages, although it can be produced by many other cell types such as CD4+ lymphocytes, NK cells and neurons.The primary role of TNF is in the regulation of immune cells. TNF, being an endogenous pyrogen, is able to induce fever, apoptotic cell death, cachexia, inflammation and inhibits tumorigenesisand viral replication and respond to sepsis via IL1 & IL6 producing cells.A local increase in concentration of TNF causes the cardinal signs of inflammation to occur: heat, swelling, redness, pain and loss of function. Whereas high concentrations of TNF induce shock-like symptoms, the prolonged exposure to low concentrations of TNF can result in cachexia, a wasting syndrome. The pathological activities of TNF-α have attracted much attention.High levels of TNF-α correlate with increased risk of mortality.TNF-αparticipates in both inflammatory disorders of inflammatory and non-inflammatory origin[14].

Conclusion

The etiology of CKDshowed hypertension to be the most common cause of CKD (7 patients in group A and 7 in group B) followed by diabetic nephropathy (6 and 5 patients in each group respectively), chronic glomerulonephritis (3 in group A and 3 in group B) and chronic pyelonephritis (1 in group A and 1 in group B).

References

1. Dr. AarushiKataria, Dr. Naveen Nandal and Dr. Ritika Malik, Shahnaz Husain -A Successful Indian Woman Entrepreneur, International Journal of Disaster Recovery and Business ContinuityVol.11, No. 2, (2020), pp. 88–93
2. Aarushi, Naveen Nandal, Parul Agrawal. AN EXPLORATORY RESEARCH IN PRODUCT INNOVATION IN AUTOMOBILE SECTOR. JCR. 2020; 7(2): 522-529. doi:10.31838/jcr.07.02.98
3. Kumar, S. (2022). Effective hedging strategy for us treasury bond portfolio using principal component analysis. Academy of Accounting and Financial Studies Journal, Vol. 26, no.2, pp. 1-17
4. Almahirah, M. S., S, V. N., Jahan, M., Sharma, S., & Kumar, S. (2021). Role of Market Microstructure in Maintaining Economic Development. Empirical Economics Letters, Vol.20, no.2, pp. 01-14
5. Modi GK, Jha V. The incidence of end-stage renal disease in India: a population-based study. Kidney Int. 2006;70:2131-3.
6. Snyder S, Pendergraph B. Detection and evaluation of chronic kidney disease. Am Fam Physician.2005;72:1723-32.
7. Rajapurkar MM, John GT, Kirpalani AL, Abraham G, Agarwal SK, Almeida AF, et al. What do we know about chronic kidney disease in India: First reportof the Indian CKD registry. BMC Nephrol.2012;13:10.
8. Raman R, Ganesan S, Pal SSet al. Prevalence and risk factors for diabetic retinopathy in rural India. BMJ Open Diabetes Res Care. 2014;2:e000-0005.
9. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R et al. Prevalence of diabetes and prediabetes (impaired fasting glucose and/orimpaired glucose tolerance) in urban and rural India: Phase I results of theIndian Council of Medical Research-India DIABetes (ICMR-INDIAB)study. Diabetologia.2011;54:3022-7.
10. Panesar S, Chaturvedi S, Saini NK. Prevalence and Predictors ofhypertension among residents aged 20-59 years of a slum resettlement colonyof Delhi, India. WHO South East Asia J Public Health.2013;2:83-7.
11. Bhadoria AS, Kasar PK, Toppo NA, Bhadoria P, Pradhan S, Kabirpanthi V. Prevalence of hypertension and associated cardiovascular risk factors in Central India. J Family Community Med. 2014;21:29-38.
12. Modi GK, Jha V. The incidence of end-stage renal disease in India: A population-based study. Kidney Int.2006;70:2131-3.
13. Agarwal SK, Dash SC, Irshad M, Raju S, Singh R, Pandey RM. Prevalence of chronic renal failure in adults in Delhi, India. Nephrol DialTransplant. 2005;20:1638-42.
14. Singh NP, Ingle GK, Saini VK, Jami A, Beniwal P, Lal M *et al*. Prevalence of low glomerular filtration rate, proteinuria and associated risk factors in North India using Cockcroft-Gault and Modification of Diet in Renal Disease equation: An observational, cross-sectional study. BMC Nephrol. 2009;10:4.
15. Singh AK, Farag YM, Mittal BV, Subramanian KK, Reddy SR, Acharya VN, *et al*. Epidemiology and risk factors of chronic kidney disease in India-results from the SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC Nephrol. 2013;14:114.
16. Remuzzi G, R.E: Hematologic consequences of renal failure. Brenner BM,Ed.The kidney. 5th ed. Philadelphia. 1995;2:170-85.
17. Parson L, Ekolo M. Aaemia in azotemia. Am J Med Sci. 1933;185:181-90.
18. Magner W. A textbook of the hematology, Philadelphia: P Blakisshan'sSon and Co; Kidney Int. 2002;38:395-9.