A Comparative Cross-Sectional Study on Clinical and Laboratory Profile of Chronic Kidney Disease in Diabetic and Non-Diabetic Patients at a Tertiary Care Teaching Hospital, India

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ABSTRACT

BACKGROUND

Diabetic nephropathy is the major cause for chronic kidney disease (CKD) in India, but there is plethora of non-diabetic causes of CKD. This study was conducted to analyse the aetiological profile of CKD, compare demographic details, clinical characteristics, laboratory parameters between diabetic and non-diabetic causes of CKD.

METHODS

This is a comparative cross-sectional study conducted in a tertiary centre at Maduranthagam, Tamil Nadu, on 250 subjects. The study population included all renal failure cases diagnosed in the study setting during the period December 2017 - December 2019. CKD grade is assessed as per National Kidney Foundation (NKF / KDOQI) staging system. The quantitative variables were analysed by mean, and standard deviation. Categorical variables were analysed by frequency and proportion.

RESULTS

250 patients were included in the analysis; 49.20 % were diabetics with a mean age of 62.81 ± 10.44 years, and 50.80 % were non-diabetics with a mean age of 59.24 ± 10.46 years. Among the non-diabetics, 88.98 % had hypertension and 51.22 % among diabetics had hypertension. 55 subjects had both diabetes and hypertension. In the diabetes group, 39.84 % patients had trace proteinuria, 9.76 % had proteinuria +, 4.88 % had proteinuria ++ and 45.53 % participants had proteinuria +++. Among non-diabetics, 51.97 % had trace proteinuria and 40.94 % had proteinuria +++. In both groups, majority of patients had grade 5 renal failure with 57.72 % among diabetics and 56.69 % among non-diabetics.

CONCLUSIONS

The clinical and laboratory profile was significantly different among the two groups. In diabetic CKD, intensified risk factor control of blood glucose and HbA1c was needed, while in non-diabetic CKD, better blood pressure control measures was needed.

KEYWORDS

Chronic Kidney Disease, Aetiological Profile, Diabetes Mellitus, Laboratory Parameters, Diabetic Nephropathy, Hypertensive Nephropathy

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BACKGROUND

Chronic kidney disease carries a great burden to health and economic status of the population. CKD is considered as an independent risk factor for cardiovascular illness.[1] CKD is diagnosed based on the signs of renal damages such as: serum creatinine, albumin: creatinine ratio (ACR) and poor renal function when threshold of glomerular filtration rate (GFR) projected from serum creatinine is very low.[1] CKD is grouped into 5 stages:" stage 1 (kidney damage with normal or increased GFR, 90), stage 2 (kidney damage with mildly decreased GFR, 60 - 89), stage 3 (moderately decreased GFR, 30 - 59), stage 4 (severely decreased GFR, 15 - 29), and stage 5 (kidney failure, GFR < 15)" [all GFR in mL / min / 1.73 m2].[2]

According to Global Burden of Disease (GBD), CKD is ranked as 17, among the causes of mortality globally. However, in majority of countries, CKD ranks among the top 5 causes of mortality. As per the GBD 2015, chronic kidney disease ranks as the 8th leading cause of mortality in India.[3] CKD is grouped based on aetiology, albuminuria category and GFR category. CKD is defined as any abnormality of kidney structure or function (GFR < 60 ml / min / 1.73 m^2), present for > 3 months.[4] The aetiology of CKD is usually based on with or without underlying systemic diseases and site of known or assumed pathologic irregularities (glomerular, tubule-interstitial, vascular or cystic and congenital diseases).[5] Sathyan S et al.[6] found that chronic glomerulonephritis (51 %) and diabetic nephropathy (22 %) were common causes of CKD. Another study by Alam AM et al[7] found hypertensive nephropathy (60.4 %) to be most frequent cause for CKD followed by chronic glomerulonephritis and diabetic nephropathy.

The development and progression of CKD involves risk factors such as decreased nephron count at birth, nephron loss due to ageing, acute and chronic exposure to noxious substance or illness such as type 2 diabetes and obesity.[8] CKD is greatly associated with hypertension and diabetes in developed countries. But in developing countries the main cause of CKD is communicable diseases, toxins from environment and other causes are unknown.[9-11]

Diabetic nephropathy is one of the chief aetiologies of chronic renal failure in India,[14] but there is plethora of non-diabetic causes of CKD, which are less understood and less studied among Indian population. Thus, it is imperative for clinicians to have an understanding of the common contributing aetiologies for CKD pertaining to a particular region. There is paucity of data regarding spectrum of chronic renal failure in South India and the current study compares the demographic, clinical and laboratory parameters of chronic renal failure cases among diabetics and non-diabetics presenting at a rural tertiary care teaching hospital.

Objectives

1. To analyse the aetiological profile of chronic renal failure cases presenting to a rural tertiary care teaching hospital located in Maduranthagam, Tamilnadu.
2. To compare the demographic, clinical and laboratory parameters between diabetic and non-diabetic causes of chronic renal failure among the study population.

METHODS

This is a comparative cross-sectional study conducted in the Department of General Medicine, tertiary care teaching hospital, located in Maduranthakam, Tamilnadu. The study population included all the renal failure cases diagnosed in the study setting in 2 years from December 2017 - December 2019. The sample size was calculated by assuming the highest proportion of any particular aetiology of CKD as 51 %, with 95 % confidence level and 5 % absolute precision.

The demographic details, anthropometric data, presence of co-morbidities, family history of CRF, presenting signs, laboratory parameters consisting of complete blood picture, liver profile, urine protein were obtained from the hospital records from December 2017 to December 2019. A total of 250 subjects were included in the final analysis.

The data was collected retrospectively. The patients were already diagnosed cases of diabetes or hypertensive nephropathy based on imaging, and many had subsequently developed diabetes in hypertensive nephropathy group.

CRF was grouped based on glomerular filtration rate, as suggested by the US-based National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF-K / DOQI), and adopted by the Kidney Disease Improving Global Outcomes (KDIGO) as well as the National Service Framework (NSF) for Renal Services and Kidney Disease and National Institute of Health and Clinical Excellence (NICE).[14] Chronic renal failure and grade of renal failure is assessed as per National Kidney Foundation (NKF / KDOQI) staging system. GFR is estimated as per abbreviated MDRD (Modification of Diet in Renal Disease) formula.

Statistical Analysis

Proteinuria, renal failure and severity of nephropathy were considered as primary outcome variables. Study group (diabetes vs. non-diabetes) was considered as primary explanatory variable. Demographic variables, presenting complaints, co-morbidities, family history CKD and lab investigations were other study relevant variables. Mean and standard deviation was carried out for quantitative variables and categorical variables were analysed for proportion and frequency. Median and interquartile range (IQR) was analysed for non-normal distributed variables. For normal distribution Shapiro-Wilk test was done and a P-value of < 0.05 was considered significant. Independent t test was used to compare between 2 groups, chi square test was used for categorical outcomes. W + A P-value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.[15]

RESULTS

A total of 250 subjects were included in the final analysis. 55 subjects had both diabetes and hypertension in the present study. The mean age was high in diabetic people (62.81 ± 10.44) compared to non-diabetic people. The mean difference between two groups was statistically significant (P-value 0.007). (Table 2). The difference in chest discomfort between the groups was significant with a P-value of 0.034. The difference in oliguria between the study groups was found to be significant with a P-value of 0.003. The difference in pruritis between the groups was significant with a P-value of < 0.001. The difference in pallor, between the groups was significant with a P-value of 0.012. (Table 3).

Study Groups	Frequency	Percentages
Diabetes	123	49.20 %
Non-diabetes	127	50.80 %

Table 1. Descriptive Analysis of Study Groups in the Study Population (N = 250)

Demographic Variables		Study Groups: Diabetes (N = 123) (Mean ± SD)	Study Groups: Non-Diabetes (N = 127) (Mean ± SD)	P-Value
Age in years		62.81 ± 10.44	59.24 ± 10.46	0.007§
Gender	Male	83 (67.48 %)	82 (64.57 %)	0.627*
	Female	40 (32.52 %)	45 (35.43 %)	
Occupation	Employed	83 (67.48 %)	96 (75.59 %)	0.155*
	Unemployed	40 (32.52 %)	31 (24.41 %)	
Family History of CKD	Yes	0 (0 %)	5 (3.94 %)	0.06**
	No	123 (100 %)	122 (96.06 %)	

Table 2. Comparison of Mean of Demographic Variables between the Study Groups (N = 250)

chi- square test *, Fisher's exact test- **, independent t test- §

Presenting Complaints		Study Groups: Diabetes (N = 123)	Study Groups: Non-Diabetes (N = 127)	Chi Square	P Value
Difficulty in breathing	Yes	95 (77.24 %)	94 (74.02 %)	0.351	0.553*
	No	28 (22.76 %)	33 (25.98 %)		
Chest discomfort	Yes	26 (21.14 %)	42 (33.07 %)	4.493	0.034*
	No	97 (78.86 %)	85 (66.93 %)		
Generalised oedema / volume overload	Yes	23 (18.7 %)	17 (13.39 %)	1.313	0.252*
	No	100 (81.3 %)	110 (86.61 %)		
Oliguria	Yes	11 (8.94 %)	1 (0.79 %)	9.095	0.003**
	No	112 (91.06 %)	126 (99.21 %)		
Haematuria	Yes	0 (0 %)	1 (0.79 %)	NA	1**
	No	123 (100 %)	126 (99.21 %)		
Neurological symptoms	Yes	4 (3.25 %)	1 (0.79 %)	1.936	0.208**
	No	119 (96.75 %)	126 (99.21 %)		
Pruritis	Yes	3 (2.44 %)	26 (20.47 %)	19.816	< 0.001**
	No	120 (97.56 %)	101 (79.53 %)		
Pallor	Yes	38 (30.89 %)	22 (17.32 %)	6.310	0.012*
	No	85 (69.11 %)	105 (82.68 %)		
Icterus	Yes	8 (6.5 %)	7 (5.51 %)	0.109	0.741*
	No	115 (93.5 %)	120 (94.49 %)		
Pedal oedema	Yes	83 (69.75 %)	86 (67.72 %)	0.118	0.731*
	No	36 (30.25 %)	41 (32.28 %)		
Facial puffiness	Yes	23 (18.7 %)	18 (14.17 %)	0.934	0.334*
	No	100 (81.3 %)	109 (85.83 %)		
Anasarca	Yes	0 (0 %)	1 (0.79 %)	NA	1**
	No	123 (100 %)	126 (99.21 %)		

Table 3. Comparison of Presenting Complaints and Signs between the Study Groups (N = 250)

chi- square test *, Fisher's exact test **

The mean difference for systolic blood pressure SBP between the study group was statistically significant (P-value < 0.001). The mean difference for HB between the study group was statistically significant (P-value < 0.001). The mean difference for S. Bilirubin indirect between the study group was statistically significant (P-value 0.001). The mean difference of S. Bilirubin direct between the study group was statistically significant (value 0.042). The mean difference for SGOT between the study group was statistically significant (P-value 0.030) (Table 4)

	Parameter	Study Groups: Diabetes (N = 123) (Mean ± SD)	Study Groups: Non-Diabetes (N = 127) (Mean ± SD)	P Value
Examinations	Height (in cm) (N = 250)	160.07 ± 6.36	161.06 ± 7.03	0.241§
	Weight (in Kg) (N = 250)	62.28 ± 6.78	63.12 ± 8.86	0.405§
	SBP (mm of Hg) (N = 250)	143.11 ± 13.92	149.61 ± 11.78	< 0.001§
	DBP (mm of Hg) (N = 250)	86.26 ± 7.83	87.42 ± 6.77	0.212§
	Hypertension	63 (51.22 %)	113 (88.98 %)	< 0.001*
Investigation	Hb (N = 250)	10.18 ± 1.89	11.47 ± 2.34	< 0.001§
	TC (N = 250)	9439.02 ± 3397.41	9544.09 ± 2293.42	0.774§
	DC_ p (N = 250)	60.42 ± 10.17	61.44 ± 10.06	0.427§
	DC_L (N = 250)	26.7 ± 8.53	27.15 ± 7.15	0.651§
	DC_ E (N = 245)	4.29 ± 2.5	3.76 ± 2.11	0.078§
	DC_M (N = 248)	7.89 ± 7.01	8.21 ± 6.64	0.707§
	Blood urea (N = 250)	72.82 ± 22.95	75.76 ± 26.14	0.347§
	Serum creatinine (N = 250)	4.16 ± 1.83	4.3 ± 1.6	0.528§
	S. Bilirubin total (N = 250)	0.74 ± 0.5	0.74 ± 0.41	0.981§
	S. Bilirubin indirect (N = 250)	0.22 ± 0.07	0.25 ± 0.1	0.001§
	S. Bilirubin direct (N = 250)	0.43 ± 0.16	0.39 ± 0.17	0.042§
	SGOT (N = 250)	24.94 ± 22.23	32.03 ± 28.66	0.030§
	SGPT (N = 250)	27.26 ± 20.49	31.76 ± 24.05	0.113§
	GGT (N = 250)	32.85 ± 7.14	33.6 ± 6.45	0.382§
	S. Albumin (N = 250)	3.63 ± 0.43	3.61 ± 0.45	0.715§
	A/C ratio (N = 250)	300.63 ± 126.36	299 ± 122.95	0.918§

Table 4. Comparison of Mean of Examination and Investigation between the Study Groups

independent t test- §, chi- square test *

Parameters		Study Groups: Diabetes (N = 123)	Study Groups: Non-Diabetes (N = 127)	P Value
Proteinuria	Trace	49 (39.84 %)	66 (51.97 %)	0.07**
	+	12 (9.76 %)	4 (3.15 %)	
	++	6 (4.88 %)	4 (3.15 %)	
	+++	56 (45.53 %)	52 (40.94 %)	
	0	0 (0 %)	1 (0.79 %)	
Grade of Renal Failure at Diagnosis	2	5 (4.07 %)	11 (8.66 %)	0.62**
	3	8 (6.5 %)	5 (3.94 %)	
	3A	2 (1.63 %)	3 (2.36 %)	
	3B	7 (5.69 %)	5 (3.94 %)	
	4	28 (22.76 %)	31 (24.41 %)	
	5	71 (57.72 %)	72 (56.69 %)	
	6	1 (0.81 %)	0 (0 %)	

Table 5. Comparison of Proteinuria and Renal Failure between the Study Groups (N = 250)

Fisher's exact test- **

		Study Groups: Diabetes (N = 123)	Study Groups: Non-Diabetes (N = 127)	P Value
Etiological Factors	CGN	87 (70.73 %)	14 (11.02 %)	< 0.001*
	Obstructive uropathy	21 (17.07 %)	12 (9.45 %)	0.07*
	Tubulointerstitial disease	10 (8.13 %)	8 (6.29 %)	0.575*
	ADPKD	8 (6.5 %)	3 (2.36 %)	0.131**
	Miscellaneous	18 (14.63 %)	2 (1.57 %)	< 0.001**
	Unknown	3 (2.44 %)	3 (2.36 %)	1**
Severity of nephropathy	Diabetic nephropathy	105 (88.24 %)	12 (9.45 %)	< 0.001*
	Non-diabetic nephropathy CGN	3 (2.44 %)	2 (1.57 %)	0.680**
	Non-diabetic nephropathy drug induced	3 (2.44 %)	0 (0 %)	0.117**
	Non-diabetic nephropathy Hypertensive nephropathy	68 (55.28 %)	102 (80.31 %)	< 0.001*

Table 6. Comparison of Aetiological Factors Between the Study Groups

chi- square test- *, Fisher's exact test- **

In diabetes group, 49 (39.84 %) people had trace, 12 (9.76 %) participants had proteinuria +, 6 (4.88 %) participants had proteinuria ++ and 56 (45.53 %) participants had proteinuria +++. In diabetes group, patients with grade of renal failure at diagnosis, majority of participants 71 (57.72 %) had grade 5 and 28 (22.76 %) participants had grade 4. (Table 5)

The difference in diabetic nephropathy between the groups was significant (P < 0.001). The difference in non-diabetic nephropathy, hypertensive nephropathy between the groups was significant (P - < 0.001). The difference in CGN between the groups was significant (P-value < 0.001). The difference in miscellaneous between the groups was significant (P-value < 0.001) (Table 6).

DISCUSSION

CKD is of diverse aetiology like diabetic nephropathy, hypertensive nephrosclerosis, glomerulonephritis, chronic interstitial nephritis, obstructive uropathy, renovascular, and genetic mediated. A comprehensive understanding of the prevalence of CKD and its risk factors are therefore, necessary in different people from different areas.[16] This is a retrospective comparative study to analyse the aetiological profile of chronic renal failure cases and to compare the demographic, clinical and laboratory parameters between diabetic and non-diabetic causes. Regarding aetiology of CKD in our study, among the diabetic group, major cause of CKD was diabetic nephropathy while it was hypertensive nephropathy in the non-diabetic group. Majority of the patients had grade 5 renal failure with 57.72 % among diabetics and 56.69 % among non-diabetics.

A total of 250 subjects were included in the final analysis of whom 49.20 % participants had diabetes and 50.80 % participants were non-diabetic with the mean age of diabetes group being 62.81 ± 10.44 and that of non-diabetics was 59.24 ± 10.46. The difference in the proportion of gender and occupation between groups was statistically not significant. In their study population Zhang JJ et al had 18.14 % in CKD with diabetes and non-diabetics with CKD 81.86 %,[17] with a majority of non-diabetics. The mean age and male to female ratio was greater in CKD with diabetes and CKD patients with diabetes mellitus (DM) were generally less well-educated in their study.[17]

Among the presenting complaints, oliguria was noted in 8.94 % of the diabetics and it was noted only 0.79 % among the non-diabetics. Chest discomfort was noted more among the non-diabetic group with 33.07 %. Pruritus was also noted to be present among non-diabetic group at 20.47 % whereas only 2.44 % complained of pruritus among the diabetics. The proportion of presenting complaints of difficulty in breathing, generalised oedema, haematuria and neurological symptoms were similar among both groups.

Both groups had similar anthropometrics in terms of height and weight. With the percentage of hypertensives being more in the non-diabetic group, the mean SBP (mm of Hg) also was higher at 149.61 ± 11.78 as compared to those in the diabetes group, which was 143.11 ± 13.92. SBP was greater in CKD subjects with DM than in those without DM on Zhang JJ et al study.[17] Pallor was noted more so among the diabetics. The difference in icterus, pedal oedema and facial puffiness between the study groups is found to be insignificant. Among the laboratory parameters, the mean HB (g / dl) in people with diabetes group was 10.18 ± 1.89 and it was 11.47 ± 2.34 in people with non-diabetes group. In the study by Zhang JJ et al[17], haemoglobin (g / dL) was 11.86 ± 2.27 among the diabetic group and 14.62 ± 1.34 among non-diabetics. Non-diabetics had comparatively better haemoglobin in both the studies. White blood cells (WBC) and differential count were found to be similar among both groups. Blood urea, serum creatinine, serum bilirubin total was also same among both groups. Mean serum bilirubin indirect in diabetes group was 0.22 ± 0.07 and it was 0.25 ± 0.1 in people with non-diabetes group. The mean serum bilirubin direct was high in diabetes group at 0.43 ± 0.16 and it was 0.39 ± 0.17 in people with non-diabetes group. Mean SGOT was higher among the diabetic group. The mean difference between the proportion of mean serum albumin and mean albumin-creatinine ratio was not significant between two groups. Serum albumin levels were lower in CKD patients with DM than in those without DM in the study by Zhang JJ et al.[17]

In diabetes group, 39.84 % people had trace proteinuria, 9.76 % had proteinuria +, 4.88 % had proteinuria ++ and 45.53 % participants had proteinuria +++. Among non-diabetics 51.97 % had trace proteinuria and 40.94 % had proteinuria +++. In both groups majority of patients had grade 5 renal failure with 57.72 % among diabetics and 56.69 % among non-diabetics.

Among the diabetic group, a whooping majority of 88.24 % had diabetic nephropathy. Hypertensive nephropathy was noted in 80.31 % of the non-diabetic group. This is because majority of the participants in the non-diabetic group were hypertensive. A study by Chaudhari ST et al[18] showed a prevalence of 32 % with diabetic nephropathy followed by hypertension in 20 % and chronic glomerulonephritis in 10 % with most frequent cause of CKD. In another study diabetic nephropathy (31.2 %) and hypertensive nephropathy (12.8 %) were frequent causes for CKD.[19] A study by Parsi MM et al[20] found that diabetes and hypertension to be most frequent cause for CKD. Vejakama et al noted in their study that CKD progressed at more rapid rate in diabetic patients with kidney failure when compared to non-diabetics. The diabetic patients showed CKD progression in about 5 to 8 years lesser for varying GFR category.[12] A meta-analysis by Fox CS et al[13] showed the risk of death was 1.2 to 1.9 times greater in diabetes compared to non-diabetes across the ranges of eGFR and albumin-to-creatinine ratio. From the fixed reference points of ACR and eGFR in non-diabetics and diabetics, the hazards ratio of death outcomes as per low eGFR and greater ACR was similar between non-diabetics and diabetics.

CONCLUSIONS

The clinical and laboratory profile was significantly different among the two groups. In diabetic CKD, intensified risk factor control of blood glucose and HbA1c is needed, while

in non-diabetic CKD, better blood pressure control measures are needed. Similarly, among hypertension patients, better blood pressure control measures would help in controlling the progression of chronic renal failure.

Limitations

No laboratory tests for HbA1c were done and no data on glycaemic control was taken. Large-scale studies should be done for a clear profile of chronic renal failure among diabetics and non-diabetics.

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REFERENCES

[1] Hill NR, Fatoba ST, Oke JL, et al. Global prevalence of chronic kidney disease–a systematic review and meta-analysis. PLoS One 2016;11(7):e0158765.
[2] National Clinical Guideline Centre (UK). Chronic kidney disease (partial update): early identification and management of chronic kidney disease in adults in primary and secondary care. London: National Institute for Health and Care Excellence (UK), July 2014.
[3] GBD 2015 Mortality and Causes of Death Collaborators. Global, regional and national life expectancy, all-cause mortality and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet 2016;388(10053):1459-1544.
[4] National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Dis 2002;39(2 Suppl 1):S1-S266.
[5] Levey AS, Coresh J, Bolton K, et al. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Diseases 2002;39(2 Suppl 1):S1-S266.
[6] Sathyan S, George S, Vijayan P, et al. Clinical and epidemiological profile of chronic kidney disease patients in a tertiary care referral centre in South India. Int J Community Medicine and Public Health 2016;3(12):3487-3492.
[7] Alam AM, Anissuzzaman S, Jahan N, et al. Clinical and aetiological profile of chronic kidney disease patients on hemodialysis in a tertiary care hospital in northern part of Bangladesh. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 2018;17(8):42-45.
[8] Romagnani P, Remuzzi G, Glassock R, et al. Chronic kidney disease. Nat Rev Dis Primers 2017;3(1):17088.
[9] Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease. Global dimension and perspectives. Lancet 2013;382(9888):260-272.
[10] Couser WG, Remuzzi G, Mendis S, et al. The contribution of chronic kidney disease to the global burden of major non-communicable diseases. Kidney Int 2011;80(12):1258-1270.
[11] Stanifer JW, Jing B, Tolan S, et al. The epidemiology of chronic kidney disease in Sub-Saharan Africa: a systematic review and meta-analysis. Lancet Global Health 2014;2(3):e174-e181.
[12] Vejakama P, Ingsathit A, Attia J, et al. Epidemiological study of chronic kidney disease progression: a large-scale population-based cohort study. Medicine (Baltimore) 2015;94(4):e475.
[13] Fox CS, Matsushita K, Woodward M, et al. Associations of kidney disease measures with mortality and end-stage renal disease in individuals with and without diabetes: a meta-analysis. Lancet 2012;380(9854):1662-1673.
[14] Viswanathan V. Type 2 diabetes and diabetic nephropathy in India - magnitude of the problem. Nephrol Dial Transpl 1999;14(12):2805-2807.
[15] IBM Corp. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp. Released 2013.
[16] Rajasekar P, Sameeraja V, Poornima B. Aetiological spectrum of chronic kidney disease in young: a single center study from South India. J Integr Nephrol Androl 2015;2(2):55-60.
[17] Zhang JJ, Yang L, Huang JW, et al. Characteristics and comparison between diabetes mellitus and non-diabetes mellitus among chronic kidney disease patients: a cross-sectional study of the Chinese Cohort Study of Chronic Kidney Disease (C-STRIDE). Oncotarget 2017;8(63):106324-106332.
[18] Chaudhari ST, Sadavarte AV, Chafekar DS. Clinical profile of end stage renal disease in patients undergoing hemodialysis. MVP J Med Sci 2017;4(1):8-13.
[19] Jha V. Current status of end-stage renal disease care in India and Pakistan. Kidney Int Suppl 2013;3(2):157-160.
[20] Parsi MM, Kanni Y, Malhotra V. Etiology and clinico-social profile of chronic kidney disease cases admitted to a dialysis unit in a rural tertiary care hospital. Sch J App Med Sci 2015;3(6A):2183-2189.