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Original Research Article

Study of thyroid dysfunction and dyslipidemia in chronic kidney diseases

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ABSTRACT

Background: Though there are many studies on thyroid dysfunction and dyslipidemia in Chronic Kidney Disease (CKD), no study is conclusive. Aim of this study was to correlate abnormalities in thyroid function and lipid profile with the severity of renal failure and also to observe the difference of these abnormalities between patients on conservative management verses hemodialysis.
Methods: Hundred consecutive CKD cases admitted to Medicine Department were taken up for the study. They were divided into two groups as Group-A [on conservative management] and Group-B [on regular Hemodialysis (HD)]. Hundred healthy persons were taken as control in Group-C. After evaluation of thyroid function and lipid profile statistical analysis was done by students t-test, chi-square and regression analysis.
Results: Hundred CKD cases with 74% male (n=74) and 26% female (n=26) in a M: F ratio of 2.9:1 were found to be in different stages CKD (0, 2, 20, 28 and 50 in stage-1 to stage-5 respectively). In 50 cases of stage-5 CKD, 30 were on HD and 20 on conservative management. Diabetes Mellitus (DM) (40%) was the commonest etiology of CKD followed by Hypertension (HTN), obstructive uropathy, chronic glomerulonephritis (CGN) and polycystic kidney disease (PKD). Thyromegaly was not found in a single case. In all CKD cases (Group-A+B) TT3 (TT3) was significantly low (P =0.0011) when compared with control (Group-C) and no difference was found between Group-A and Group-B. Fall in TT3 worsened with increasing severity of CKD. Lipid profile study revealed Decreased High-Density Lipoprotein Cholesterol (HDLc) and increased Triglyceride (TG), Total Cholesterol (TC), Low Density Lipoprotein Cholesterol (LDLc), TC/HDLc and LDLc/HDLc in Group-A than Group-B but only TG and TC increase was statistically significant. The levels of TG and TC and TC/HDLc increased as the stage of CKD progressed and was statistically significant (P= 0.035).
Conclusions: There occurs a state of biochemical hypothyroidism without overt clinical hypothyroid state in CKD, the extent of which correlates with the severity of CKD. Increased cardiovascular complications occur due to accelerated atherosclerosis in CKD. This study confirmed that atherogenic lipid profile and thyroid dysfunction worsen with the progression of disease. Difference between patients on conservative management and HD was not found.

Keywords: Biochemical hypothyroid state, Dyslipidemia, Hemodialysis, Thyroid function Test

INTRODUCTION

Chronic Kidney Disease(CKD) is a silent Pandemic.[1] The number of people with impaired renal function is rapidly rising, especially in developing countries from Asia due to increase in concomitant diseases such as Type 2DM, HTN and analgesic abuse. The incidence of End Stage Renal Disease (ESRD) in India is around 229 per million population. And more than one lakh new patients enter renal replacement programmes annually in India. The prevalence of Chronic Renal Failure (CRF) in India is 0.8%.[2] DM has emerged as the main cause (30-40%) followed by HTN (14-22%), CGN (16-20%), Chronic Interstitial Nephritis (5.4-12.7%), Heredofamililal disease (8.4%) and Obstructive uropathy (2.9%). Progression of CKD is associated with increase in number of complications like anemia, peripheral neuritis, thyroid dysfunction, dyslipidemia and CVD.[3]

Though uremia in some instances causes clearly recognizable endocrinopathies, more commonly the endocrine dysfunctions remain as only laboratory abnormalities. CKD alters the thyroid status at biochemical levels, sometimes leading to overt clinical syndromes. With the introduction of radioimmunoassay and chemiluminescense assay methods for estimation of thyroid hormones, the thyroid status in CKD has been studied extensively by a host of workers. Most of the authorities have demonstrated biochemical evidence of hypothyroidism and a few have detected hyperthyroidism, even goiter and exophthalmos like Silverberg et al and carter et al.[4,5] Although uremia shares some of the clinical features of myxedema, overt clinical disturbances of thyroid function ordinarily does not occur Grantham et al and Spector et al.[6,7] In CKD Ramirez et al and Lim et al observed biochemical evidence of hypothyroidism but in contrast Spector et al reported clinical euthyroidism in CKD patients on HD.[7-9]

Various workers have attributed different causes for thyroid dysfunction however Victoria et al in their study of CKD patients before HD, during HD and after renal transplant comprehended the abnormalities at three different levels.[10] First there is a blunted Thyroid-stimulating Hormone (TSH) response to Thyrotropin-releasing Hormone (TRH) suggesting pituitary dysfunction, second there occurs intrathyroidal defects in hormonogenesis or hormonal secretion or both and last being impaired conversion of T4 to T3 in extra thyroidal tissues, resulting in selective and marked reduction in serum TT3 concentration.[9]

CKD leads to disturbances in the function of virtually every organ system of the body. However, it is well documented that CVDs are major cause of morbidity and mortality in patients with CKD, so the American Heart Association has recommended CKD (specially on dialysis) to be classified in the highest risk group for developing cardiovascular events.

CVD mortality is 10 to 30 times higher in CKD.[11] Accelerated atherosclerosis leads to increase in cardiovascular complications in patients with CKD. Several factors contribute for atherogenesis, most notable among which is alterations in lipoprotein metabolism involving all lipoprotein classes and it shows variations depending on the degree of renal impairment, etiology, presence of nephrotic syndrome and method of dialysis i.e. HD or peritonea! dialysis (PD).

Dyslipidemia seen in CKD is characterized by high TG and low HDLc levels, accumulation of remnant particles, predominance of low-density lipoprotein (LDL) particles, and increased lipoprotein a (Lp (a)).[12] Dyslipidemia has also been hypothesised to cause kidney damage and to play an important role in the progression of renal failure as well.[13] Dyslipidemia may damage glomerular capillary endothelial and mesangial cells as well as podocytes. Mesangial cells express receptors for LDLc and oxidized LDLc, which upon activation induce mesangial cell proliferation, increase mesangial matrix deposition, and enhance the production of chemokines such as macrophage chemo-attractantprotein-1 and cytokines such as interleukin-6. Macrophages infiltration release cytokines causing damage to the endothelial cells, mesangial cells and podocytes leading to progressive renal damage.

Thyroid dysfunction added to dyslipidemia in CKD may further increase CVD risk. Hence early diagnosis of thyroid dysfunction and dyslipidemia by regular screening, and treatment slows the progression of CKD, in addition to reduction of CVD risk.[13] Thyroid dysfunction causes significant changes in kidney function and kidney diseases can be associated with thyroid disorders. Thus, CKD and thyroid dysfunction mutually influence each other. Kidney Disease Outcomes Quality Initiative (KDOQI) working group suggested all CKD patients to be evaluated for dyslipidemia because of high risk for CVD1. In view of lack of data for thyroid dysfunction and dyslipidemia in CKD in this part of India the present study was undertaken.

The aim of the study was to determine thyroid dysfunctions and dyslipidemia in CKD patients and to establish correlation between severity of renal disease with these two metabolic parameters. The aim was also to find differences if any in thyroid dysfunction and dyslipidemia in CKD patients in conservative management and those on regular HD.

METHODS

Hundred consecutively admitted CKD patients to the PG Department of medicine of SCB Medical College Hospital, Cuttack were enrolled for the study. The inclusion criteria for CKD was that of National Kidney Foundation-Kidney Disease Outcome Quality Initiative (NKF-KDOQI) guidelines 1. for CKD which is presented below:

- Kidney damage for >3 months, as defined by structuralor functional abnormalities of the kidney, with or without decreased GFR, manifest by either:
- Pathological abnormalities or
- Marker of kidney damage, including abnormalities in the composition of the blood or urine, or abnormalities in the imaging tests
- GFR <60ml/min/1.73m^2 for >3months, with or without kidney damage. Kidney damage is evidenced by
  -Proteinuria >300mg/day
  -Pathological abnormality found in histopathological study or
  -Renal USG showing bilateral contracted kidney <9cm with thinned parenchyma and reduced corticomedullary differentiation.

Informed consent was obtained from each patient. The study had Institute Ethical Committee (IEC) approval.

Patients having past history of thyroid dysfunction, patients on thyroid hormone supplementation or antithyroid drugs, patients on lipid lowering agents and patients on drugs having potential to alter thyroid function or lipid profile like Rifampicim, Salicylates, Amiodarone, Propranalol, sulfonamides, Acetazolamide, Aminoglutethimides, Phenylbutazone, Iodides, Phenytoin, Lenalidomide, Alemtuzumab, Sunitinib, Lithium, IFN-a, Statins and Fibric Acid derivatives were excluded from the study.

The patients were divided into two groups as Group-A (n=70) (on conservative management) and Group-B (n=30) on HD. Group-A was further divided into various stages of CKD basing on Kidneys Disease Improving Global Outcome (KDIGO) staging presented below.

Group-B comprised of Stage-5 CKD patients as per recommendations. Another hundred healthy persons were taken as control after age and sex standardization and were kept in Group-C.

Table 1: Kidney disease improving global outcome (KDIGO) staging of chronic kidney disease (U9).

Stages	Criteria
1	GFR >90 with evidence of renal damage
2	GFR 60-89 with evidence of renal damage
3a	GFR 45-59
3b	GFR 30-44
4	GFR 15-29
5	GFR < 15 or patient is on dialysis

Detailed history and results of thorough clinical examination of patients were recorded. Estimation of urea by Urease Glutamate dehydrogenase method, creatinine by Alkaline Picrate method and 24hour urinary protein estimation was done by Esbach's Albuminometer (Mason and Swash 1980). Microscopic examination of urine for cells, casts, and crystals was done in centrifuged urine.

Thyroid function test of TT4, TT3, Free T4 (FT4), Free T3 (FT3), TSH and Thyroxin-Binding Globulin (TBG) was done by Chemi-Luminescence–Immuno-Assay (CLIA) TC and TG in the plasma were measured enzymatically and the cholesterol in the supernatant was measured after precipitation of Apo-B containing lipoproteins to determine HDL-C. LDL Cholesterol was measured by Friedwald formula

[LDL-C = Tc – [HDL– C + TG/5]]

VLDL-C was estimated by dividing plasma TG by 5 reflecting the ratio of cholesterol to triglyceride in VLDL particles.

The data were statistically analysed using SPSS version 22. For continuous variables, the data were presented as the mean $\pm$SD or the median within range and means were compared using one-way analysis variance. For categorical variables the data were presented as counts and percentages and the differences were analysed using Chi-Square test

RESULTS

Hundred CKD cases admitted to Medicine Department of SCB Medical College Hospital, Cuttack consisting of 74 (74%) male patients and 26 (26%) female patients with male to female ratio of 2.9:1 formed the study group. Out of 100 patients no one was in stage 1, while 2, 20, 28 and 50 patients were in stage 2, stage 3, stage 4, and stage 5 CKD respectively as presented in Table 1.

DM was the commonest etiology accounting for 40% cases of CKD with only DM present in 24% cases and in rest 16% cases DM was associated with HTN.

Next in order was HTN alone in 10%, SLE in 10%, obstructive uropathy in 10%, CGN in 4%, PKD in 2% and the etiology could not be found in 24% of the cases.

Out of 50 cases in stage 5 CKD, 20 cases were on conservative management and 30 cases were on regular HD.

Thyromegaly was not found in a single case. Thyroid function study revealed only TT3 mean values to be significantly low (P =0.0011) in CKD cases (Group-A+ B) and when compared with control (Group-C) and was significantly low (P= 0.004).

When TT3 values of Group-A (on conservative management) was compared with Group C (healthy subjects) and that of Group-B was compared with Group-C it was also significantly low (P value = 0.004 and P = 0.0071 respectively) as presented in Table-2 and Table-3.

Table 2: Thyroid function tests: group-A vs group-C.

	Total T_4 (µg/dl)	Total T_3 (ng/dl)	Free T_4 (ng/dl)	Free T_3 (pg/ml)	TSH (µu/ml)	TBG (µg/dl)
Group-A (n=70) Conservative treatment	7.772±2.547	1.193±0.423	1.146±0.423	2.898±1.074	7.38±11.66	19±1.029
Group-C (n=100) Control	7.825±1.42	1.54±0.39	1.256±0.268	0.05±0.39	2.487±1.5	19+0.917
P value	0.474	0.004	0.299	0.5475	0.0684	>0.99

Table 3: Thyroid function tests: Group-B vs Group-C.

	Total T_4 (µg/dl)	Total T_3 (ng/dl)	Free T_4 (ng/dl)	Free T_3 (pg/ml)	TSH (µu/ml)	TGB (µg/dl)
Group-B Hemodialysis (N=30)	7.62±1.457	1.196±0.292	1.245±0. 23	3.101±0.425	3.505±1.133	19±1.069
Group-C Control (N=100)	7.825±1.42	1.54±0.391	1.256±0.268	3.05±0.393	2.487±1.50	19+0.917
P Value	0.5537	0.0071	0.8993	0.716	0.354	0.889

Table 4: Thyroid function tests: Group-A vs Group-B.

	Total T_4 (µg/dl)	Total T_3 (ng/ml)	Free T_4 (ng/dl)	Free T_3 (pg/ml)	TSH (µu/ml)	TGB (µg/dl)
Group-A Conservative (N=70)	7.772±2.547	1.193±0.423	1.146±0. 423	2.898±1.074	7.38±11.66	19±1.029
Group-B Hemodialysis (N=30)	7.62±1.475	1.196±0.292	1.245±0.23	3.101±0.425	3.505±1.133	19+1.069
P Value	0.8304	0.988	0.399	0.4854	0.2077	>0.99

Statistically significant difference in thyroid function between Group-A and Group-B was not found in our study as Presented in Table 4. With increasing severity of CKD from Stage 2 to Stage 5 only fall in TT3 was found to be statistically significant (P = 0.01) when compared with healthy controls (Group-C) as presented in Table 5.

Table 5: Correlation of thyroid function tests with stages of CKD on conservative management.

Thyroid function tests	Stage-2 (N=2)	Stage-3 (N=20)	Stage-4 (N=28)	Stage-5 on conservative management (N=20)
Total T_4 (µg/dl)	9.4	9.32±1.486	7.0885±2.739	7.048±2.701
Total T_3 (ng/dl)	2.34	1.361±0.221	1.1085±0.362	1.029±0.46
Free T_4 (ng/ml)	1.46	1.367±0.282	1.037±0.415	1.1065±0.515
Free T_3 (pg/ml)	3.66	3.47±0.855	2.515±0.962	2.807±1.287
TSH (µiu/ml)	1.25	3.889±5.795	11.34±16.37	5.9±6.94
TGB(mg/l)	20	19.4±1.074	19.07±1.141	18.5±0.707

Difference in thyroid function of male CKD comparing with female CKD patients was statistically significant in TT4 (P=0.0258) and TT3 (P=0.0421). When thyroid function of 40 diabetics out of 100 CKD cases was compared with control group low TT3 was only significant (P =0.0005). Lipid Profile study revealed HDLc to be higher in female CKD cases than males which was statistically significant (P=0.025). The mean values of TG, TC, LDLc, VLDLc, TC/HDL and LDLc/HDLcwere higher in diabetic CKD cases than non-diabetic CKD cases and all were statistically significant (P<0.05). HDL-C was found to be lower in diabetic CKD cases than non-diabetic CKD cases and was statistically significant (P=0.0343). The mean values of TG, TC, LDLc, VLDLc, TC/HDL, LDLc/HDLc were higher and mean value of HDLc was lower in CKD patient on conservative management (Group-A) as compared to those on HD (Group-B) and all were statistically

significant (P < 0.05) except LDLc, HDLc and LDL-c/HDL-c as presented in Table 6.

Table 6: Comparison of lipid profile between CKD Patients on conservative treatment (Group-A) vs on hemodialysis (Group-B).

	Triglyceride (MG%)	Total cholesterol(MG%)	LDL-C (MG%)	HDL-C (MG%)	VLDL-C (MG%)	TC/ HDL-C	LDL-C/ HDL-C
On conservative treatment(N=70)	260.23±53.45	212.45±22.76	121.35±22.34	33.98±5.14	52.35±11.65	6.11±0.36	3.55±0.36
On hemo dialysis (N=30)	239.45±53.37	193.32±25.53	118.54±22.67	36.54±5.68	48.38±12.57	5.57±0.65	3.36±0.67
P Value	0.0433	0.0311	0.0421	0.0343	0.039	0.047	0.038

The level of TG and TC increased as the stage of CKD Progressed and was statistically significant in both with P=0.001 and P=0.035 respectively as presented in Table-7 and Table-8. The mean value HDLc decreased as the stage of CKD progressed in our study as presented in Table-9. The mean value of TC/HDLc also increased as the stage of CKD progressed and was statistically significant (P= 0.035) as presented in Table 10.

Table 7: Serum Triglyceride levels (mg%) among patients in different stages of CKD.

CKD stage	Triglyceride (mg%)
1	-
2	219.00±4.5
3	223.88±31.94
4	232.91±58.90
5	272.79±50.65
Mean value	250.91±56.15

Table 8: Serum cholesterol levels (mg%) among patients in different stages of CKD.

CKD stage	Cholesterol (mg%)
1	-
2	192.00±22.00
3	203.09±26.08
4	206.25±23.92
5	210.43±25.99
Mean value	205.33±27.02

The means value of LDLc and LDLc/HDLc increased as CKD stage progressed but in stage 3 both were higher than that of Stage 4 and Stage 5 and this finding was not statistically significant.

DISCUSSION

Of late globally increasing trend of CKD has put the health care facilities around the world under tremendous strain. Increase CVD morbidity and mortality in CKD is caused by dyslipidemia. Thyroid dysfunction in CKD is another area of concern. Keeping in mind the paucity of studies in this aspect the present study was conducted to evaluate dyslipidemia and thyroid dysfunction in CKD patients and to correlate both with the severity of disease.

Dyslipidemia and thyroid dysfunction between CKD patients on conservative management and HD were also compared to observe differences.

Table 9: Serum HDLc levels (mg%) among patients in different stages of CKD.

CKD stage	HDL-C (mg%)
1	-
2	35.5±3.50
3	35.25±3.06
4	35.22±3.92
5	34.45±4.28
Mean value	34.86±3.99

Table 10: Serum total cholesterol/HDLc levels in different stages of CKD.

CKD stage	Total cholesterol/HDL-C
1	-
2	4.34±0.49
3	5.81±0.85
4	5.85±0.48
5	6.19±1.02
Mean value	5.95±0.96

100 cases of CKD diagnosed as per NKF-KDOQI guidelines clinically and confirmed by biochemical tests and imaging studies were taken for the study. Out of 100 patients studied, none was in stage 1 CKD, while 2, 20, 28 and 50 patients were in stage-2, stage-3 and stage-4 and stage-5 respectively.

The mean age of total patients enrolled in this study was 53.94±13.38yrs. The mean value of blood urea in study patients was 87.611±25.10 mg% and the mean value of serum creatinine was 6.57±2.69 mg%. Male patients in the series were 74 (74%) and females were 26 (26%) with a M: F ratio of 2.9:1.

DM was the commonest etiology accounting for 40% cases of CKD out of which 24% were having DM alone and 16% were associated with HTN. Next in order were HTN (10%), SLE (10%) and Obstructive uropathy (10%), CGN (4%) and PKD (2%) and the cause was unknown in 24% of the cases.

The cases presented with wide range of symptoms. The commonest was anorexia (84%) followed by lethargy (70%) and oliguria (44%). No specific symptoms of hypo or hyperthyroidism was present in our study. Most common physical sign was pallor (100%) and HTN (84%). Some overlapping physical signs found in both CKD and hypothyroidism like facial puffiness, pedal edema, coarse skin was present in some of the cases. No patient had thyromegaly. Absence of goiter in our study is consistent with the observation made by Mehta et al but is in contrast to Ramirez et al, Silverberg et al, Lym VS et al and Victoria et al all of whom found goiter in a significant percentage of patients.[4,8-10,14]

When the thyroid function of 100 CKD cases (Group-A+B) were compared with the control (Group-C), it was found that TT4, TT3, FT4 and FT3 values were lower and only TT3 was significantly low (P=0.0011). This result is concordant with the studies of Victoria et al, Silverberg et al, Carter et al, Grantham et al.[4,5,6,10,15] The mean TSH of all CKD patients (Group A+B) was higher than that of controls (Group C) but was not statistically significant (P=0.0994).

TT4, TT3, FT4, and FT3 values of Group-A were lower in comparison with that of Group-C but only TT3 was significantly low (P=O.004) which is consistent with the study of Mehta et al, Victoria et al, Silverberg et al.[4,10,14] Though mean value of TSH in Group-A was higher than that of Group-C but it was not statistically significant (P=0.0684).When thyroid function of 30 CKD cases on regular HD (Group-B) was compared with the healthy subjects (Group-C), only TT3 was found to be low (P=0.0071) which is consistent with the studies of Mehta et al; Victoria et al, Silverberg et al.[4,10,14] High TSH of Group-B comparing with Group-C was not statistically significant (P=O.354).

Statistically significant difference in thyroid function between Group-A and Group-B patients was not found (P>0.05). With increasing severity of renal dysfunction, the fall in TT3 was only statistically significant in our study (P=0.01). Lipid profile observation in our study showed increase in mean values of TG, TC, LDLc, VLDLc, TC/HDLc and LDLc/HDLc and decrease in HDL-C in CKD cases (Group-A+B) when compared with to control (Group-C) and were statistically significant except for LDLc, HDLc, LDLc/HDLc (p<0.05).

The mean values of TG, TC, LDLc, VLDLc, TC/HDLc and LDLc/HDLc were higher and the mean value of HDLc was lower in CKD patients in Group-A than Group-B. Among these all were statistically significant except LDLc, HDLc and LDLc /HDLc (p<0.05)

The level of TG increased as the stage of disease progressed and was statistically highly significant (p value=0.001). This is in concordance with the study conducted by Kaysen et al.[15] TC levels also increased as the stage of CKD progressed, similar to findings of Kasiske BL et al study.[16] LDLc levels increased as the stage of CKD progressed except for stage 3 where it was higher than stage 4 and stage 5, but this was not statistically significant (p=O.15) and is in concordance with the study by Kasiske et al.[16]

Increased TC/HDLc ratio indicative of atherogenic risk in ESRD patients was found to be high and increased as the stage of disease progressed and was statistically significant(p=0035) concordant with studies of Cheung et al and Avram et al.[17,18] The mean value of LDLc/HDLc was also high which is concordant to the studies of Cheung et al and Massy et al.[17.19] This ratio increased as the stage of disease progressed except for stage 3 where it was higher than stage 4 and stage 5, and this was not statistically significant.

CONCLUSION

Review of literature reveals that despite extensive study of thyroid function and dyslipidemia in CKD patients the results are still inconclusive. Most of the studies infer that biochemical hypothyroidism occurs in a high percentage of cases, but overt clinical manifestation does not occur. Increased cardiovascular complications in CKD is due to accelerated atherosclerosis by dyslipidemia.

The present study also revealed low T3 despite a near normal T4 and it worsens with the progression of CKD but metabolic status remains normal without clinical hypothyroid state. No difference in thyroid status was observed between dialysis and nondialysis patients. Dyslipidemia in CKD worsened as patients progresssed to severe stages with significant increase in TG, TC, VLDLc and TC/HDLc confirming presence of atherogenic lipid profile needing early intervention to prevent cardiovascular complications.

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