Thyroid Profile in Chronic Kidney Disease Patients- A Study in Rims Hospital

L.Romesh Sharma¹, Mossang K², Salam Ranabir³

¹Assistant professor, ²Post graduate trainee, ³Associate professor Department of Medicine, Regional Institute of Medical Sciences, Imphal, Manipur, India. Corresponding Author: Salam Ranabir

Abstract: Chronic kidney disease is an irreversible deterioration of renal function. The serum level and the function of most of the hormones are altered due to various mechanism including synthesis, transport, accumulation of inhibitors, target organ responsiveness and renal clearance. Our study aims to evaluate the thyroid profile in patients of chronic kidney disease. It is a cross-sectional study carried out at Department of Medicine, Regional institute of Medical Sciences, Imphal. Duration of study was from September 2014 to February 2016. Study Population includes 100 patients of CKD above 21 years of age. The commonest cause of CKD was found to be diabetes (35%).21% patients had TSH level above the normal range (>5 µIU/ml), 19% patients had low T3(<1.3 ng/ml) and 16% had low Thyroxin (T_4) level. The prevalence of Hypothyroidism was found to be higher in patients with reduced GFR.

Date of Submission: 11-12-2019

Date of Acceptance: 26-12-2019

I. Introduction

Chronic kidney disease (CKD) is an irreversible deterioration of renal function, which results from diminished effective functioning of renal tissue. Ensuing impairment of excretory, metabolic and endocrine function of the kidney leads to the development of clinical syndrome of uremia.¹Prevalence of CKD in India is estimated at 7572 per million and end stage kidney disease at 757 per million populations.²Cardiovascular disease is a major cause of morbidity and mortality among patients with CKD.^{3,4,5} Majority of patients die from cardiovascular system complications. In CKD, the serum level and the function of most of the hormones are altered because of several interplaying mechanisms. The mechanism may be synthesis, transport, accumulation of inhibitors, abnormality of target organ responsiveness and impaired renal clearance.⁶ Dialysis affects thyroid hormone metabolism minimally.⁷ Though regular dialysis is done in these patients, most of these uremic hormone abnormalities are not reversed, and some of them may even get worse.^{8,9}Uraemia influences the function and size of the thyroid.¹⁰ Uraemic patients have an increased thyroid volume compared with subject with normal renal function and a higher prevalence of goitre, mainly in women.^{10,11,12} Also, thyroid nodules and thyroid carcinoma are more common in uraemic patients than in the general population.¹³Most patients with end stage renal disease have decreased plasma level of free triiodothyronine (T3), which reflect diminished conversion of thyroxineT4 toT3 in the periphery.^{14,15} Chronic metabolic acidosis associated with the CKD may contribute in this effect.¹⁶ This abnormality is not associated with increased conversion to metabolically inactive reverse T3(rT3), since the plasma rT3 levels are typically normal. Although free and total T4 concentration may be normal or slightly reduced, sometimes freeT4 may be high due to the effect of heparin used in anticoagulation during haemodialysis (HD), which inhibit T4 binding to its binding proteins.¹⁷The plasma concentration of thyroid stimulating hormone (TSH) is usually normal in CKD. However, the TSH response to exogenous thyrotrophic-releasing hormone (TRH) is often blunted and delayed, with a prolonged time required to return to baseline level.^{18,19} Reduced renal clearances may contribute to this delayed recovery, since TSH and TRH are normally cleared by kidney. The kidney normally contributes to the clearance of iodide from the body. With advancing renal failure iodide excretion is diminished leading sequentially to an elevated plasma inorganic iodide concentration and an initial increment in the thyroidal iodide uptake. The ensuing marked increase in the intrathyroidal iodide pool results in diminished uptake of radiolabeled iodide by the thyroid in uraemic patients. Increases in total body inorganic iodide can potentially block thyroid hormone production (the Wolff-Chaikoff effect). These changes in iodide metabolism may account for reports of a slightly higher frequency of goitre and hypothyroidism in patient with CKD.^{20,21}A study had reported reversion of thyroid function to normal after successful renal transplantation.²²This study was designed to evaluate the thyroid profile in CKD patient in RIMS Hospital, Imphal and to correlate the findings with different parameters.

II. Material and Methods

It is a Cross-sectional study carried out at Department of Medicine, Regional institute of Medical Sciences, Imphal. Duration of Study was from September 2014 to February 2016. Study Population includes patients with CKD.

Inclusion criteria:

Patients of CKD above 21 years.Diagnosic criteria for CKD includes a) Clinical signs and symptoms of uraemia, b)The presence of CKD is established based on the presence of kidney damage and the level of kidney function (GFR) and c) Ultrasonographic evidence of bilateral shrunken kidney/ loss of corticomedullary differentiation.

Exclusion criteria:

- 1. Patients with history of alcohol consumption
- 2. Patients with thyroid and liver disease
- 3. Patients with Nephrotic syndrome
- 4. Patients on medication likely to affect thyroid function
- 5. Pregnancy.

Sample size:

A minimum of 100 CKD patients admitted in the Medicine ward, RIMS within the period of September 2014 to February 2016 were included in the study.

Methodology:

All the selected patients were subjected to detailed history and complete physical examination and data were noted in a pre-designed proforma.Blood sample was drawn (10ml) for assessing baseline electrolytes, hemogram, Liver function tests, Renal function tests and Thyroid function test. Haemogram was assessed by spectrophotometric method available in Department of pathology RIMS. Renal function test and liver function test was assessed by Roche auto-analyzer module P40 available in Department of Biochemistry, RIMS.All the laboratory methods was standardized with the coefficient of variation (CV) for all of the biochemical parameters varying from 1-5 %.Ultrasonography of kidney was performed in department of Radiodiagnosis. The presence of CKD was established based on presence of kidney damage and level of kidney function (GFR). Markers of kidney damage included abnormalities in the composition of blood (elevated blood urea, serum creatinine) or urine abnormalities. Ultrasonogram showing Bilateral shrunken Kidneys(<8.5cm) with loss of corticomedullary differentiation was taken as indicative of chronic renal failure. Estimation of Total Thyroxine(T4) and Total triiodothyronine was carried out ELISA test as described by Hopton MR et al²⁴. Hypothyroidism was diagnosed by TSH >10 μ IU/ml and low level of T4 and T3.Subclinical hypothyroid was diagnosed by normal level of T4 and T3 along with TSH >5 μ IU/ml.

Statistical analysis of all the data was done by using SPSS 20 software. Chi square test, Pearson's correlation coefficient and T test were used wherever applicable. The level P < 0.05 was considered as the cutoff value or significance. Informed consent was taken from the patient included in the study and the study was approved by Institutional Ethics committee.

III. Result

A total of 100 patients of CKD irrespective of cause, duration and stages were taken up for the study. The most common cause of CKD was Diabetic Nephropathy and Chronic Glomerulonephritis followed by Obstructive Uropathy(Table 1). The mean age of the patients ranged from 21 to 77 years (mean age 50.38 \pm 14.91 years). There were 62(62%) males and 38(38%) females. Maximum number of patients 29 (29%) were in the age group of 51 – 60 years. The total duration of disease ranged from 1 to 20 years (mean=4.85 \pm 3.51 years). Most patients (34%) had disease duration ranging between 4 to 5 years. The body mass index ranged from 13.04kg/m² to 30.50kg/m² with a mean of 21.68 \pm 3.34kg/m². Most of the patients (70%) are in normal BMI range.

Table I. Causes of CKD	
Underlying Disease	Number of patients
Diabetic Nephropathy	35(35%)
Chronic glomerulonephritis	35(35%)
Obstructive uropathy	23(23%)
Lupus Nephritis	6(6%)
ADPKD(Autosomal dominant polycystic kidney disease)	1(1%)

Table 1. Causes of CKD

Thyroid stimulating hormone (TSH). TSH level ranged from 1-23 μ IU/ml (mean 4.11±3.5 μ IU/ml). Maximum patients 79 (79%) had TSH level in the normal range (0.25-5 μ IU/ml)and 21(21%) patients had TSH level above normal range (>5 μ IU/ml) (Figure 1).





Tri-iodothyronine(T_3):Tri-iodothyronine level ranged from 0.5-3.1ng/ml (mean 1.63± 0.63). Maximum patients 81 (81%)had T3 level in normal range (1.3-3.1 ng/ml) and 19 (19%)patients had low T3(<1.3 ng/ml) (Figure 2).



Figure 2

Thyroxine (**T4**):Thyroxine(T_4) level ranged from 39.60 -158 µg/ml (mean 95.05±29.1). Most of the study patients(84) had Thyroxine (T_4) level in the normal reference range(66-181 µg/ml) and 16 patients had low T4(<66 µg/ml) (Figure 3).





The relation of mean serum TSH, T3 and T4 with stages of CKD are shown in Table 2. Mean TSH is significantly higher in CKD stage V as compared to stage IV and III and mean T3 and T4 were found to be higher in CKD stage III as compared to Stage IV and V although it was not statistically significant.Maximum patients (47%) in CKD Stage V had TSH in the normal range.Maximum patients (49%) in CKD Stage V had T3 in the normal range andMaximum patients (50%) in CKD Stage V had T4 in the normal range.

Table 2 showing relation between mean serum TSH, T3 and T4 with stages of CKD				
	CKD V	CKD IV	CKD III	P Value
	(GFR<15)	(GFR15-29)	(GFR 30-59)	
	n =61	n=25	n=14	
TSH µIU/ml	4.5 ± 2.8	3.76 ±4.73	3.08 ± 3.72	0.07
T3 ng/ml	1.35 ± 0.34	1.84 ±0.61	2.47 ± 0.79	0.13
T4 µg/ml	83.3±21.98	106.84 ±23.27	125.04 ±36.6	0.4

.

Abnormal thyroid function.

Out of 100 Chronic kidney disease patients, 18 (18%) had Hypothyroidism,4(4%) had sub-clinical hypothyroidism and 78 (78%) were Euthyroid (TSH,T3 and T4 in normal range). None of the patients had hyperthyroidism (Figure 4).





Maximum number of hypothyroid patients i.e. 13 (72.2%) were in CKD stage V followed by 3 (16.6%) patients in CKD stage IV and 2 (11.2%) patients in CKD stage III.Maximum 3 (75%) patients of Sub-clinical Hypothyroidism occurred in CKD stage V and 1(25%) patients in CKD stage IV(Figure 5).



Figure 5

Amongst the cause of CKD, the prevalence of hypothyroid was maximum in Lupus Nephritis (33.3%) followed by obstructive uropathy (26%) and the prevalence of Sub-clinical hypothyroidism was maximum (5.7%) in Diabetes mellitus followed by Obstructive Uropathy (4.3%), (Table 3).

Table 3showing thyro	id status and etiologies of CKD
----------------------	---------------------------------

Parameter	Hypothyroid(%)	Subclinical Hypothyroid(%)
DM	8.5%	5.7%
OBU	26%	4.3%
CGN	20%	2.8%
ADPKD	0%	0%

Lupus Nephritis	33.3%	0%
Total		100

IV. Discussion

A total of 100 patients of CKD irrespective of cause, duration and stages were taken up for the study. Diabetic nephropathy (35%) and Chronic glomerulonephritis (35%) was the commonest cause of CKD.In a study of 30 CKD patients by Avasthi G et al²⁵, Chronic glomerulonephritis (30%) was the commonest cause followed by Obstructive uropathy (6%) .In a study by Mittal S et al²⁶, the commonest cause of CKD in India was Chronic glomerulonephritis.According to third annual report of CKD registry of India by Indian society of Nephrology²⁷, the commonest cause of CKD was diabetic nephropathy (30%),followed by undetermined cause (15.6%).In a study by Gibi J et al²⁸ conducted in RIMS Hospital in 2002, the commonest cause of CKD was Chronic glomerulonephritis (48.64%) followed by Obstructive uropathy (15.54%).But we found that diabetes has overtaken Chronic glomerulonephritis as the commonest cause of CKD. Our study shows that diabetic nephropathy and Chronic glomerulonephritis are the commonest cause of CKD which agrees with other studies.

The body mass index ranged from 13.04 to 30.50 kg/m² (mean of 21.68 \pm 3.34). In this study most of the patients 70 (70%) had normal BMI range,14 (14%) were underweight and 14 (14%) were overweight .Bjorn O A et al²⁹ in their study found that mean body mass index (kg/m2) of the patients were 26.5 which is similar to the present study.Maheshwari et al³⁰ in their study found that 48% of patients on maintenance haemodialysis had BMI <18.5 kg/m2 and 42% had normal BMI. Our study also shows that most of the patients of CKD had normal BMI.

In our study TSH level ranged from $1-23\mu$ IU/ml (mean $4.11\pm3.5 \mu$ IU/ml). Maximum patients 79 (79%) had TSH level in the normal range (0.25-5 μ IU/ml)and 21 (21%)patients had TSH level above normal range(>5 μ IU/ml) and there were no patients with low level of TSH.Avasthi G et al²⁵ in their study of thirty patients of CKD showed that mean TSH level and free T4 was significantly increased in patients of CKD as compared to controls (4.73±2.60ug/ml, vs 2.69±.34ug/ml p<0.05).Mehta H J et al³¹ in their study measured the level of TSH, Total T3,free T3,Total T4 and free T4 in 127 patients of CKD and found that the patients on conservative management for CKD the serum TSH and FT4 were not significantly altered.Patients with low T3,T4 and free T4 showed a high TSH suggesting maintenance of pituitary thyroid axis in CKD patients which is similar to our study and mean TSH was not high and TSH response to TRH was distinctly blunted, suggesting possibility of pituitary dysfunction as well.

Tri-iodothyronine (T3)level ranged from 0.5-3.1 η g/ml (mean 1.63± 0.63). Maximum patients 81 (81%) had T3 level in normal range(1.3-3.1 η g/ml) and 19(19%) patients had low T3(<1.3 η g/ml).Among these patients (19) with low T3 level, CGN had maximum 10 (52.6%) patients, followed by 7 (36.8%) of Diabetic nephropathy and 2 (10.5%) obstructive uropathy. Ramirez G et al³² in their study showed that in patients on dialysis, mean serum thyroxin and triiodothyronine levels are lower than normal. Spector D A et al³³ studied thyroid function and metabolic status in 38 patients with chronic renal insufficiency who were in a dialysis programme. They found mean serum total triiodothyronine (T3) concentration to be normal but 43% of the group had low serum T3 and 54% had low serum free T3 concentrations. Serum TSH concentrations were normal in all but four subjects who had very slight elevations.In our study ,the findings are comparable with other previous studies showing decrease level of T3 in uremic patients.

Thyroxine(T_4) level ranged from 39.60 -158 µg/ml (mean 95.05±29.1). Most of the study patients 84 (84%) had Thyroxin (T_4) level in the normal reference range (66-181 µg/ml) and 16 (16%) patients had low T4 (<66 µg/ml.). Maximum patients 8(50%) of CGN had low T4 followed by 6(37.5%) patients of Diabetic nephropathy and 2(12.5%) patient of obstructive uropathy. Jasso et al ³⁴found that uremic patients had low serum TT3 and elevated T3 resin uptake suggesting a decrease in TBG. However actual measurement of TBG was normal .They postulated that uremic toxin might have displaced T4 from TBG. Anima X et al ³⁵evaluated the level of TT4,T3 and TSH in 96 clinically euthyroid patients with chronic renal failure and 25 healthy individual as control and they found that there was significant decrease in total T4 in 26 (42%) patients. In our study 16 (16%) patients had T4 level below normal which is comparable with previous studies. The different studies mentioned various reasons for decreased level of T4. The decreased level of T4 may be secondary to the protein loss which occurs in CKD. Serum albumin and thyroid binding Pre-albumin level are decreased in patients of CKD. Decrease in T4 is also attributed to the presence of the circulating inhibitors which impairs binding of T4 to thyroxin binding globin.

In our study, out of 100 Chronic kidney disease patients, 18 (18%) had Hypothyroidism, 4(4%) had sub-clinical hypothyroidism and 78 (78%) were in Euthyroid state. None of them had Hyperthyroid. ChoncholM et al³⁶ in their study of 3089 patients found that most participants (84.6%) had serum thyroid function test results within the reference range whereas 9.5% had subclinical biochemical hypothyroidism and 5.9% had subclinical biochemical hyperthyroidism. Lo JC et al ³⁷ in their study found that the prevalence of hypothyroidism was 8.62%. Among the 18 hypothyroid patients maximum number of hypothyroid patients i.e.

13(72.2%) were in CKD stage V followed by 3(16.6%) patients in CKD stage IV and 2(11.2%) patients in CKD stage III. Among the 4 Subclinical hypothyroid patients, maximum 3 (75%) patients of Sub-clinical Hypothyroidism occurred in CKD stage V and 1(25%) patients in CKD stage IV. In our study that the prevalence of hypothyroidism was increased in patients with reduced GFR, ranging from 14.2% for patients with estimated GFR 30-59 mL/min/1.73m2 (CKD stage III) to 21.3% in patients with estimated GFR <15 mL/min/1.73m2(CKD stage V).However this findings was not statistically significant.

In our study, among the etiology of CKD the prevalence of hypothyroid is maximum in Lupus Nephritis (33.3%)The prevalence of Sub-clinical hypothyroidism is maximum (5.7%) in Diabetes mellitus followed by Obstructive Uropathy (4.3%)and Chronic glomerulonephritis 2.8%, though not reaching statistical significance.

Mean TSH is higher in CKD stage V as compared to stage IV and III showing that the mean TSH level increases along with decrease in GFR. Mean T3,T4 were found to be higher in CKD stage III as compared to CKD Stage IV and V ,which also shows that mean T3 and T4 level were found to be higher in patients with higher GFR .Lo JC et al ³⁷ in their study found that prevalence of hypothyroidism progressively increases from patients with higher GFR to patients with low GFR .Chonchol M et al ³⁶ studied 3089 patient of CKD and they found that the prevalence of subclinical hypothyroid increases progressively from 7.6% in patients with GFR \geq 90 ml/min/1.73m² to 21% in patients with GFR <60 ml/min/1.73m².The findings in our study are comparable with others previous studies showing that there is an increase prevalence of hypothyroidism in patients with low GFR.

V.Conclusion

The most common cause of CKD was Diabetic Nephropathy (35%) and Chronic Glomerulonephritis (35%). The mean age of the patients ranged from 21 to 77 years (mean age 50.38 ± 14.91 yrs). The body mass index ranged from 13.04 to 30.50 kg/m² (mean of 21.68 ± 3.34). Mean TSH level was 4.11 ± 3.5 µIU/ml. 79% patients had TSH level in the normal range (0.25-5 µIU/ml) and 21 patients had TSH level above normal range (>5 µIU/ml). Mean T3 level was 1.63 ± 0.63 . 81% Patients had T3 level in normal range (1.3-3.1 ng/ml) and 19% patients had low T3(<1.3 ng/ml) and of these patients with low T3, CGN had maximum patients (52.6%). Mean T₄was 95.05±29.1. Maximum 84% patients had T₄ level in the normal reference range (66-181 µg/ml) and 16% patients had low T4 (<66 µg/ml). The prevalence Hypothyroidism was 18%. 4% had sub-clinical hypothyroidism was found to be increased in patients with reduced GFR. The prevalence of hypothyroid was maximum in Lupus Nephritis (33.3%). The prevalence of Sub-clinical hypothyroidism was 5.7% in Diabetes followed by 4.3% in Obstructive Uropathy and 2.8% in Chronic glomerulonephritis. Mean TSH level increased along with decline in GFR. Mean T3,T4 were found to be higher in CKD stage III as compared to CKD Stage V showing that mean T3 andT4 level were higher in patients with higher GFR.

References

- [1]. National kidney foundation. DOQI Kidney disease outcome quality initiative. Am J Kidney Dis 2002; 39: 51- 66.
- [2]. Mani MK. Prevention of CRF at the community level. Medicine update 2002; 12: 768-74.
- [3]. Ma KW, Greene EL, Raij L. Cardiovascular risk factors in chronic renal failure and haemodialysis population. Am J Kidney Dis 1992; 6:505-13.
- [4]. Eggers PW. Mortality rates amongs dialysis patients in Medicare's end stage renal disease program. Am J Kidney Dis 1990; 15: 414-21.
- [5]. Rostang SG, Kirk KA, Rutsky EA. Relationship of coronary risk factors to haemodialysis associated ischaemic heart disease, Kidney Int 1982; 22: 304-8.
- [6]. Boromini V, Orsoni B, Sorrentine MA, Todeschini P. Hormone change in haemodialysis. Blood1990; 8:54-68.
- [7]. Kaptein EM. Thyroid Hormone metabolism and thyroid disease in chronic renal failure. Endor Rev1996; 17:45-63.
- [8]. Lukinac L, Kusi CZ, Kes P, Nothig-hus D. Effect of chronic haemodialysis on thyroid function test in patients with end stage renal disease. Acta Med Croactica1996; 50:65-80.
- [9]. Bartalena L, Pacchiarotti A, Palla R, Autonagr li L, Mammali C, et al. Lack of nocturnal serum thyrototropin(TSN) surge in patient with chronic renal failure undergoing regular haemofiltration: a case of central hypothyroidism. ClinNephrol 1990; 34:30-4.
- [10]. Kaptein EM, Quion-Verde H, Choolijan CJ, Tang WW, Freidman PE, Rodriquez HJ, et al. The thyroid in end stage renal disease. Medicine (Baltimore)1998; 67:187-97.
- [11]. Hegedus L, Andersen JR, Poulsen LR, Perrild H, Holm B, Gundtoft E. Thyroid gland volume and serum concentration of thyroid hormones in chronic renal failure. Nephron 1985 ; 40 : 171-4.
- [12]. Kutlay S, Atlia T, Koseogullari O, Nergizoglu G, Duman N and Gullu S. Thyroid disorders in haemodialysis patients in an iodinedeficient community. Artificial Organs 2005; 29: 329-32.
- [13]. Miki H, Oshimo K, Inoue H, Kawano M, Morimoto T, Monden Y, et al. Thyroid carcinoma patients with secondary hyperparathyroidism. J SurgOncol 1992; 49: 168-71.
- [14]. Kaptein EM, Quinon-Verde H, Chooljian CJ, etal. The thyroid in end stage renal disease. Medicine (Baltimore) 1988; 67:187-97.
- [15]. Wartofsky L, Burman KD. Alteration in thyroid function in patient with systemic illness; the "euthyroid sick syndrome". Endor Rev 1982; 3: 164-217.
- [16]. Wiederkehr MR, Karogiros J, Krapt R. Correction of metabolic acidosis improves thyroid and growth hormone axes in haemodialysis patients. Nephrol Dial Transplant 2004; 19:1190-7.

- [17]. Silverberg DS, Ulan RA, Fawcett DM, Dossetor JB, Grace M and Bettcher K. Effects of chronic haemodialysis on thyroid function in chronic renal failure. CMAJ 1973; 109: 282-6.
- [18]. Czernichow P, Dauzet MC, Broyer M, Rappaport R. Abnormal TSH, PRL and GH response to TSH releasing factor in chronic renal failure. J ClinEndocrinolMetab 1976; 43: 630-7.
- [19]. Duntas L, Wolf CF, Keck FS, Rosenthal J. Thyrotropin releasing hormone, pharmacokinetic and pharmacodynamic properties in chronic renal failure. ClinNephrol 1992; 38: 214-8.
- [20]. Lin CC, Chen TW, Ng YY, Chou YH, Yang WC. Thyroid dysfunction and nodular goiter in hemodialysis and peritoneal dialysis patients. Perit Dial Int 1998; 18: 516-21.
- [21]. Ramirez G. Abnormalities in the hypothalamic-hypophyseal axes in patients with chronic renal failure. Semin Dial 1994; 7:138-42.
- [22]. Yashpal et al. Thyroid functions in uraemia. Ind J Nephrol 1991; 1: 2.
- [23]. Chopra IJ,NelsonJC,Solomon DH and Beall GW:A radioimmuno assay of thyroxine and tri-iodothyronine.J.Clinical endocrinol.1971; 33: 865.
- [24]. Hopton MR and HarrapJJ.Immunoradiometric assay of thyrotropin as a first line thyroid function test in the routine laboratory.Clinical chemistry.1986; 32; 691.
- [25]. Avasthi G Avasthi G, Malhotra S, Narang APS, Sengupta S. Study of thyroid function in patients of chronic renal failure. Ind J Nephrol 2001; 11:165-9.
- [26]. Mittal S, KherV, GulatiS, Agarwal LK, Arora P.Chronic renal failure in India.KidneyInt 2005;68:S 42-5.
- [27]. Third annual report of CKD registry of India.Indian society of Nephrology.
 [28]. Gibi J .A prospective study of spectrum of chronic renal failure patients in Manipur and comparasion of acetate and bicarbonate
- haemodialysis ,Thesis for MD;RIMS,2002
 [29]. Bjorn OA,Trine B and Lars J. Association of thyroid function with estimated glomerular filtration rate in a population-based study: the HUNT Study. Eur J of Endocrinol. 2011; 164 :101–105.
- [30]. MaheshwariN,AnsariMR,Laghari MS, Lal K,AhmedK.Pattern of lipid profile in patients on maintenance hemodialysis.Saudi J Kidney Dis transpl 2010;21(3):565-70.
- [31]. Mehta HJ,JosephLJ,DesaiKB,MehtaMN,SamuelAM,AlmiedaAF,AcharyaVN.Totla and free thyroid hormone levels in chronic renal failure 2012.J postgrad Med 1991;37(1):79-83.
- [32]. Ramirez G Ramirez g,O'NeillW, jr, Jubiz W and Bloomer HA. Thyroid dysfunction in uraemia, evidence for thyroid and hypophysealabnormalities. Annals of internal Medicine. 1976; 84: 672-676.
- [33]. Spector DA, Davis PJ, Helderman JH, Bell B, Utiger RD. Thyroid function and metabolic state in chronic renal failure. Annals of Internal Medicine 1976; 85(6):724-730.
- [34]. Jasso A I, Murray P C, ParkinJ, Robertson M R.Abnormalities in vitro thyroid function test in renal failure. Q J Med1974; 43:245-61.
- [35]. Anima X, Gupta A, Kumar U, Sharma H P, Murari K. Evaluation of thyroid hormone in chronic renal failure. Indian J Pathol Microboil. 1999;43(1):129-133.
- [36]. Chonchol M ,Lippi G,SalvagnoG,ZoppiniG,MuggenoM,TargherG.Prevalance of subclinical hypothyroidism in patients with chronic kidney disease. Clin J Am soc Nephrol.2008;3:1296-1300.
- [37]. Lo JC Lo JC, Chertow GM, Go AS, Hsu CY. Increase prevalence of subclinical and clinical hypothyroidism in persons with chronic kidney disease. Kidney Int 2005; 67:1047-52.

L.Romesh Sharma. "Thyroid Profile in Chronic Kidney Disease Patients- A Study in Rims Hospital." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 12, 2019, pp 62-68.

DOI: 10.9790/0853-1812106268