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Original Research Article

To study the prevalence of thyroid autoimmune antibody in chronic kidney disease patients with hypothyroidism in Eastern Uttar Pradesh

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ABSTRACT

Background: Chronic kidney disease (CKD) is defined as the presence of kidney damage. Hypothyroidism is prevalent in patients with CKD, including those on dialysis.
Materials and Methods : The present observational cross-sectional study included 120 patients (70 subjects and 50 controls) who had previously been diagnosed with chronic kidney disease and primary hypothyroidism, as well as those who presented with primary hypothyroidism alone. Serological Estimation of Anti-TPO Antibody, T3, T4 and TSH levels were done as per standard protocol.
Results: We enrolled 120 patients, 70 of whom had hypothyroidism in addition to CKD and 50 of whom had hypothyroidism without CKD. Twenty percent of the cases tested positive for anti-TPO antibodies, while 80 % tested negative for anti-TPO antibodies. 71.5 % of positive cases were female, while 28.5 % were male. Among the controls, 74% were positive for anti-TPO antibodies, and 26% were negative. 64.9 % of the positive controls were female, while 35.1 % were male.
Conclusion: Our findings indicate that primary hypothyroidism is prevalent in a subgroup of patients with chronic kidney disease. The non-autoimmune mechanism appears to be more prevalent. However, additional multi-centric studies will be necessary to improve the reliability and generalizability of the current study's observations.

1. Introduction

Chronic kidney disease (CKD) is defined as the presence of kidney damage, as measured by abnormal albumin excretion or decreased kidney function, as quantified by measured or estimated glomerular filtration rate (GFR), that persists for more than three months. The incidence and prevalence of chronic kidney disease are increasing, and patients continue to experience various complications, ranging from volume overload and electrolyte imbalances to abnormal mineral and bone metabolism and anaemia.[1,2]

Hypothyroidism is prevalent in patients with CKD, including those on dialysis.[3] It has been suggested that patients with chronic kidney disease may have a higher prevalence of clinically obvious hypothyroidism than the general population.[4] Today, diabetes and hypertension account for 40–60% of cases of chronic kidney disease.[5] With the increasing prevalence of these diseases in India, the prevalence of CKD is expected to increase, and this is clearly the primary target population.

The mechanism by which thyroid and kidney disease are related remains unknown. The thyroid gland affects kidney function through direct renal and systemic hemodynamic, metabolic, and cardiovascular effects. Numerous case studies have demonstrated that severe hypothyroidism results in increased creatinine levels, decreased plasma flow, and decreased GFR.[6] Another possibility is that patients with Hashimoto's thyroiditis develop immune-mediated

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glomerular injury.[7] Chronic kidney disease disrupts the hypothalamus-pituitary–thyroid axis and impairs the peripheral thyroid hormone metabolism. Additionally, it results in disturbed binding to carrier proteins, a possible decrease in tissue thyroid hormone content, and increased thyroid gland iodine storage.

Both triiodothyronine (T3) and thyroxine (T4) levels in the plasma are decreased. Serum T3 levels are low due to impaired extrathyroidal conversion of T4 to T3.[8] Similarly, the decrease in T4 results from circulating inhibitors that impair T4 binding to thyroxine-binding globulin. As a result, patients with chronic kidney disease may be at an increased risk of thyroid dysfunction. In chronic kidney disease (CKD), physiological compensation for low T3/T4 levels (with normal TSH levels) results in a decrease in protein catabolism, thereby increasing nitrogen waste overload.[9]

It is unknown when this physiological compensation becomes a maladaptive response. Patients with chronic kidney disease frequently have metabolic acidosis, which has been shown to cause changes in thyroid function tests (elevated TSH and decreased T4 and triiodothyronine [T3] levels), which can be partially reversed with oral sodium citrate therapy.[10] Because the kidneys are involved in the metabolism, degradation, and excretion of certain thyroid hormones and their metabolites, CKD patients may experience changes in regulating the hypothalamic-pituitary-thyroid axis.[11] Certain mechanisms of thyroid dysfunction may be observed, including decreased clearance, prolonged half-life, blunted pulsatility, altered glycosylation resulting in decreased bioactivity, and decreased response to TRH.[12]

Hypothyroidism has been linked to a variety of adverse cardiovascular outcomes in the general population. Patients with the end-stage renal disease face a seven- to tenfold increased risk of death (40 % of deaths are due to cardiovascular causes) compared to the general population.[13]

Glomerular involvement occurs in 10%–30% of patients with Autoimmune Thyroiditis.[14] Membranous Nephropathy is the most common histological pattern of glomerular injury in patients with autoimmune thyroiditis.[15] It is unknown whether this is due to the coexistence of two autoimmune diseases or whether autoimmune thyroiditis contributes to direct renal injury.[15]

1.1. Anti-thyroid peroxidase antibody

Anti-TPO antibodies have long been recognised as a marker of thyroid autoimmunity. Although elevated anti-thyroid antibody titres may indicate a risk of overt hypothyroidism, no correlation between antibody titre and risk of hypothyroidism has been discovered thus far.[16] Anti-TPO antibodies have been linked to various other diseases, including Hashimoto's Encephalopathy, SLE, rheumatoid arthritis, and myasthenia gravis. Anti-TPO positive hypothyroid patients had a lower eGFR than anti-TPO negative hypothyroid patients with nephrotic syndrome, according to a recent Indian study. Additionally, a higher level of proteinuria and a lower level of serum albumin was observed.[17]

The purpose of this study was to determine the prevalence of thyroid autoimmunity and primary hypothyroidism in a sample of unselected adult patients with varying degrees of kidney function.

2. Materials and Methods

The present observational cross-sectional study included 120 patients (70 subjects, 50 controls) who had previously been diagnosed with chronic kidney disease and primary hypothyroidism and those who presented with primary hypothyroidism without any co-morbid condition to the Medicine Department's OPD and ward between 2020 and 2021.

CKD Patients, as per The Kidney Disease Outcomes Quality Initiative (KDOQI) of the National Kidney Foundation (NKF) and the international guideline group Kidney Disease Improving Global Outcomes (KDIGO), patients coming in OPD/IPD with the diagnosis of CKD with eGFR<60 ml/min/1.73m^2 or with the diagnosis of primary hypothyroidism without any co-morbid conditions, of age >18 yrs were included in the present study. However, patients of age < 18 yrs and non-willing patients were excluded.

2.1. Calculation of GFR

COCKROFT-GAULT formula was used for the calculation of Creatinine clearance of the patients.

CrCl = {((140-age) * lean body weight) / (72*S_{Cr})} *0.85 (if female).

CrCl (creatinine clearance) = ml/min; Age = years; Lean body weight = kg; S_{Cr} = mg/dl.

Serological estimation of anti-TPO antibody, T3, T4 and tsh levels.

The serological estimation of T3, T4, and TSH using based Architect i1000 SR, was collected from the patient record.

2.2. Principle of the anti-tpo antibody estimation

It is based on the chemiluminescent microparticle immunoassay principle (CMIA). CMIA is a more sophisticated version of the Enzyme-Linked immunosorbent Assay (ELISA). The ARCHITECT Anti-TPO assay is a two-step immunoassay that utilises CMIA technology with flexible assay protocols, dubbed Chemiflex, to quantify thyroid peroxidase autoantibodies (anti-TPO) in human serum and plasma. The sample, assay diluent, and TPO-coated paramagnetic microparticles

are combined and incubated in the first step. Anti-TPO antibodies present in the sample bind to TPO-coated microparticles.

After washing, the second step involves the addition of an anti-human IgG acridinium-labelled conjugate. Following another incubation and wash, the reaction mixture is added to the pre-trigger and trigger solutions. The chemiluminescent reaction that results is quantified in terms of relative light units (RLUs). The amount of anti-TPO in the sample has a direct correlation with the RLUs detected by the ARCHITECT I system optics. The reference range taken for this study is: 0 – 5.6 IU/ml

2.3. Principle of the thyroid function test estimation

Additionally, it is based on the chemiluminescent microparticle immunoassay principle (CMIA). TSH CMIA is a sandwich immunoassay that consists of two steps. The first step involves the addition of sample anti-TSH antibody-coated paramagnetic microparticles and TSH assay diluent in TRIS buffer. After washing with a magnetic field, the second step involves the addition of an anti-TSH acridinium labelled conjugate. The reaction mixture is then added with a pre-trigger containing 1.32 % (w/v) H2O2 and a trigger containing 0.35N.NaOH. The chemiluminescent reaction that results is quantified in terms of relative light units (RLUs). There is a direct correlation between the amount of TSH in the sample and the RLUs detected. TSH concentrations are evaluated and calculated using a calibration curve established using calibrators with known TSH concentrations. The estimation of T3 and T4 are based on the same principle. Reference range taken for the study is as follows: TSH -0.3-4.9 uIU/ml ; T4- 4.8-11.7 ug/dl; T3- 0.3- 4.9 ng/ml

2.4. Statistical analysis

Statistical analysis was performed using SPSS software (SPSS Inc., Chicago, IL, USA) for Windows program (15.0 version). The continuous variables were evaluated by mean (standard deviation) or range value when required. The dichotomous variables were presented in number/frequency and were analyzed using the Chi-square test. For comparison of the means between the two groups, analysis by Mann-Whitney U test was used. With 95% confidence interval, a p-value of < 0.05 or 0.001 was regarded as significant.

3. Results

Over half of the patients in this study (52.8 %) were over the age of 50, followed by those aged 41-50 (24.3 %) and those aged 40-50 (24.3%) (22.9%). Patients had an average age of 48.19±13.54 years. The prevalence of TAN was higher in patients aged >50 years (27%) than in those aged 31-50 years (17.6%) and 40 years (40%) respectively (6.2 %). However, there was no statistically significant (p>0.05) correlation between the prevalence of TAN and age [Table 1].

Over half of cases (52.8 %) and 24% of controls were over the age of 50. Cases and controls had a mean age of 48.19±13.54 and 44.20±14.72 years, respectively. Between cases and controls, there was a significant (p=0.001) difference in age. Female patients had a higher prevalence of TAN (37%) than male patients (9.3 %). The prevalence of TAN was significantly associated with gender (p=0.005) [Figure 1].

Males accounted for more than half of cases (61.4 %) and 44 % of controls. There was, however, no statistically significant difference in gender between cases and controls (p>0.05). The mean weights of subjects with and without TAN were 84.21±14.90 and 75.79±14.49 kg, respectively. There was no significant difference in weight between groups receiving and not receiving TAN (p>0.05).

The chi-square distribution of thyroid autoimmune antibody (TAN) was significantly different between cases (with TAN =14; without TAN=56) and controls (with TAN =37; without TAN=13). The prevalence of TAN was 27.5%. The TSH, T3, T4, and TPO levels were 9.35±4.87, 0.61±0.40, 4.21±2.55 and 37.08±168.32 in cases and 9.75±5.07, 0.69±0.43, 4.73±2.85 and 126.76±257.39 in controls, respectively. There was, however, no significant difference in thyroid parameters between cases and controls (p>0.05) [Figure 2].

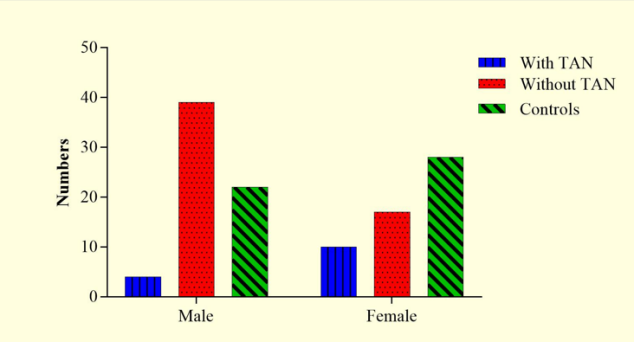

Fig. 1: Comparison gender distribution between cases and controls

Additionally, there was no significant difference in thyroid parameters between groups receiving and not receiving TAN (p>0.05). The Table 2 depicted the patient distribution by thyroid parameters and their association with the prevalence of thyroid autoimmune antibody (TAN). The prevalence of TAN was 20% in our cases. The TAN was found in 14.3 % of patients with abnormal T3. The TAN was found in 20.8 % of patients with abnormal T4 levels. There was, however, no significant (p>0.05) correlation between the prevalence of TAN and thyroid parameters.

The CBC (Hb/TLC/Platelet count) parameters were compared between groups receiving and not receiving TAN.

Table 1: Distribution of patients according to age and its association with prevalence of thyroid autoimmune antibody (TAN) in CKD cases (with and without TAN) and controls.

Age in years	With TAN		Without TAN		p-value
	No.	%	No.	%	
<40	1	6.2	15	93.8	
41-50	3	17.6	14	82.4	0.21
>50	10	27.0	27	73.0	
Mean±SD	53.64±12.92		46.82±13.45		
	Cases (n=70)		**Controls (n=50)**		
<40	16	22.9	26	52.0	
40-50	17	24.3	12	24.0	0.001*
>50	37	52.8	12	24.0	
Mean±SD	48.19±13.54		40.70±14.31		

Table 2: Distribution of patients according to thyroid parameters and its association with prevalence of thyroid autoimmune antibody (TAN)

Thyroid parameters	No. of patients (n=70)		With TAN		Without TAN		p-value [1]
	No.	%	No.	%	No.	%	
TSH							
Abnormal	70	100.0	14	20.0	56	80.0	-
Normal	0	0.0	0	0.0	0	0.0	
T3							
Abnormal	28	40.0	4	14.3	24	85.7	0.32
Normal	42	60.0	10	23.8	32	76.2	
T4							
Abnormal	48	68.6	10	20.8	38	79.2	0.79
Normal	22	31.4	4	18.2	18	81.8	

[1]Chi-square test

Table 3: Comparison of different biochemical parameters in CKD patients with and without TAN

	With TAN	Without TAN	p-value[1]
Thyroid parameters			
TSH	8.88±4.30	9.46±5.03	0.69
T3	0.70±0.41	0.59±0.40	0.35
T4	4.27±2.14	4.19±2.66	0.92
CBC parameters			
Hb	7.06±1.66	6.92±1.75	0.78
TLC	7735.71±3342.18	9267.11±5977.37	0.36
Platelet count	220214.29±102023.51	173172.32±74921.32	0.06
KFT parameters			
Serum urea	140.52±79.30	173.47±84.32	0.19
Serum creatinine	7.46±3.03	10.40±7.16	0.14
Protein parameters			
Serum B12	71.27±59.93	82.71±51.58	0.47
Ferritin	493.68±360.68	503.45±359.56	0.92
Phosphorus	9.28±13.41	17.71±30.73	0.32
Calcium	8.25±1.26	8.06±1.41	0.63
Vitamin D	12.75±7.73	13.45±6.42	0.72

[1]Unpaired t-test

Table 4: Comparison of BUN in CKD patients with and without TAN

	Groups	Mean±SD	p-value
Weight	With TAN	84.21±14.90	p=0.06
	Without TAN	75.79±14.49	
BUN	With TAN	84.50±32.35	p=0.33
	Without TAN	97.80±48.68	
GFR	With TAN	14.86±9.57	p=0.47
	Without TAN	12.96±8.72	
PTH	With TAN	406.91±281.02	p=0.46
	Without TAN	481.53±350.92	

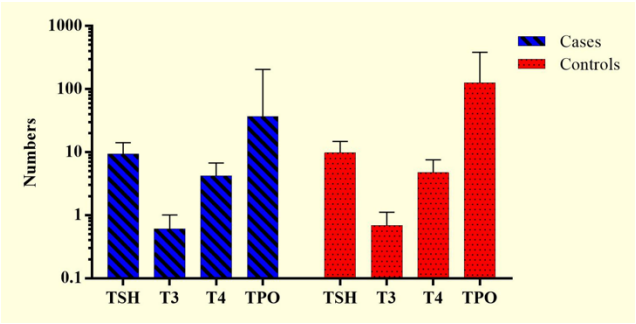

Fig. 2: Comparison of thyroid parameters between cases and controls

There was no significant difference in CBC parameters between groups receiving and not receiving TAN (p>0.05) [Table 3]. The PTH levels were 406.91±281.02 and 481.53±350.92 in the presence and absence of TAN, respectively. Although no significant difference (p>0.05) was observed [Table 3].

The metabolic parameters with and without TAN are compared in Table-13 & Fig.13. There was no significant difference in protein parameters (Serum B12, Ferritin, Phosphorus, Calcium, and Vitamin D). When KFT parameters were compared with and without TAN, no significant difference (p>0.05) was observed. The difference in BUN between groups with (84.50±32.35) and without (97.80±48.68) TAN was not statistically significant (p>0.05). The GFR was 14.869.57 in the presence of TAN and 12.96±8.72 in the absence of TAN. There was no significant difference in GFR between groups with and without TAN (p>0.05) [Table 4].

4. Discussion

The present cross-sectional study, conducted at the Department of Medicine, sought to determine the prevalence of thyroid autoimmune antibodies in patients with chronic kidney disease who also had hypothyroidism in the eastern United States.

Chemiluminescence immunoassay was used to determine thyroid function tests, specifically serum T3, serum T4, serum TSH, and anti-TPO antibody. In our study of 120 patients with hypothyroidism, 70 were diagnosed with CKD and 50 as not having CKD. Twenty percent of the cases tested positive for anti-TPO antibodies, while eighty percent tested negative for the TPO antibody. Females accounted for ten of the positive cases, while males accounted for four. 74% of controls, on the other hand, tested positive for the anti-TPO antibody.

Females made up 64.9% of the total positive controls, while males made up 35.1%, a female to male ratio of 1.9: 1. McGrogan et al.[18] conducted a similar study and discovered that the incidence of hypothyroidism is 350/100000/year in females and 80/100000/year in males, with a female to male ratio of 4.3: 1. Thus, hypothyroidism is much more prevalent in females than in males.

Female predominance is expected in this study, as thyroid disorders affect females more frequently than males. In our study, the mean (SD) age at which hypothyroidism occurred was 40.70 ±14.31 years in the control group. These observations were consistent with earlier studies of thyroid disorders conducted by Ogbera et al. in Lagos, Ojo et al.[19] in Ile-Ife, Chehade et al.[20] in the United States, and McGrogan et al.[18] in Nigeria, who discovered a mean age of 40 ±12.4 years, 42.7± 12.6 years, 47.8± 14.9 years, and 44.6 ±13.8 years, respectively.

In our study, the mean age of cases was 48.19 ±13.54 years. These observations were consistent with a study conducted by Lo CJ et al.,[21] who found a mean age of 48.7 ±18.9 years in their study. The mean TPO level was 37.08±168.32 IU/ml in cases and 126.76± 257.39 IU/ml in controls (P=0.06). Though this difference was not statistically significant, the mean TPO level in our control group was significantly higher than in the cases and significantly greater than the upper limit of normal.

According to Basu et al.[22] there is no increase in the incidence of autoimmune thyroid disease in patients with chronic kidney disease. Indeed, positive thyroglobulin and thyroid microsomal antibodies are uncommon in patients with chronic kidney disease. A similar finding was reported in our study, where 20% of 70 patients with chronic kidney disease and hypothyroidism tested positive for anti-TPO antibodies, while 74% of the 50 controls tested positive for anti-TPO antibodies.

The presence of anti-TPO antibodies does not correlate with chronic kidney disease. However, autoimmune thyroid disease can coexist with other autoimmune diseases associated with chronic kidney disease (CKD), such as lupus nephritis and type 1 diabetes mellitus. Therefore, it is critical to screen for antithyroid antibodies when an elevated TSH level is detected in conjunction with another autoimmune disease. The presence of CKD does not affect the management strategy for autoimmune thyroid disease.

5. Limitations of our Study

1. We lacked information regarding the causes of kidney diseases, coexisting medical conditions, and current medication use, including thyroid replacement therapy or anti-thyroid drugs.
2. There was no information on other, less common causes of transient or permanent primary hypothyroidism, such as drug-induced hypothyroidism, subacute thyroiditis, radiation thyroiditis, or postpartum thyroiditis.
3. We defined CKD using eGFR rather than directly measured GFR. It is well established that current GFR equations are imprecise and consistently underestimate measured GFR at higher values (i.e., in populations without known CKD).
4. Our patient population is extremely diverse, with eGFR values ranging from 48 to less than 10 ml/min. In addition, we made no distinction between patients on hemodialysis and those who were not on hemodialysis.
5. Due to the cross-sectional nature of our study, causal or temporal relationships between thyroid autoimmunity, primary hypothyroidism, and kidney disease cannot be established. However, it is well established that the prevalence of chronic kidney disease, thyroid autoimmunity, and TSH concentrations all increase significantly with age. As a result, additional prospective studies are required to elucidate these issues.

6. Conclusion

In conclusion, our findings indicate that primary hypothyroidism is prevalent in a subgroup of patients with chronic kidney disease. The non-autoimmune mechanism appears to be more prevalent. Given the abundance of contradictory and supportive studies, additional experimental and prospective studies with large sample size and uniform patient characteristics are required to confirm these findings and to elucidate the underlying biologic mechanisms before causality can be established with certainty.

7. Source of Funding

None.

8. Conflicts of Interest

There is no conflict of interest.

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