Journal of Endocrinology, Metabolism and Diabetes of South Africa 2019; 24(3):92–97
https://doi.org/10.1080/16089677.2019.1640490

JEMDSA
ISSN 1608-9677 EISSN 2220-1009

ARTICLE

Prevalence and clinical relevance of thyroid autoantibodies in patients with goitre in Nigeria

OA Ojo[a*], RT Ikem[b], BA Kolawole[b], OE Ojo[a] and MO Ajala[c]

[a]*Department of Medicine, Federal Medical Centre, Owo, Nigeria*
[b]*Department of Medicine, Obafemi Awolowo University, Ile Ife, Nigeria*
[c]*Department of Chemical Pathology, Lagos State Laboratory Services, General Hospital, Lagos*
**Corresponding author, email: ayoolaplus@yahoo.co.uk*

Background: Thyroid autoimmunity was thought to be rare in Africans but there is evidence that its prevalence is increasing. Since undetected autoimmune thyroid disease carries considerable morbidity, this study set out to determine the proportion of patients with goitre who have thyroid autoantibodies and the relationship, if any, between the presence of thyroid autoantibodies, thyroid function and thyroid size.
Methods: The study was cross-sectional and conducted over a 12-month period. It involved 100 subjects with goitre and 50 apparently healthy controls without goitre, matched for age and sex. Thyroid dysfunction was assessed by history, clinical examination and biochemical tests, thyroid peroxidase and thyroglobulin antibodies. The size of the thyroid gland was assessed by ultrasound.
Results: Fifty-seven percent (57%) of study subjects were euthyroid, 38% were hyperthyroid, while 2% were hypothyroid. The overall prevalence of elevated thyroid peroxidase antibody (TPOAb) in the subjects with goitre was 35% and 8% in the controls ($p < 0.001$). Elevated thyroglobulin antibody (TgAb) was found in 24% of subjects with goitre and 12% of controls ($p = 0.083$). Elevated TPOAb was found in 76.3% of subjects who were hyperthyroid, 7% of subjects who were euthyroid and 100% of subjects who were hypothyroid ($p < 0.001$). Elevated TgAb level was present in 36.8%, 15.8% and 50% of subjects with hyperthyroid, euthyroid and hypothyroid goitre respectively ($p = 0.068$). A positive correlation was observed between TPOAb and erythrocyte sedimentation rate ($r = 0.582$, $p < 0.001$) and TgAb and erythrocyte sedimentation rate ($r = 0.176$, $p = 0.08$). The correlation between TPOAb and thyroid volume ($r = –0.139$, $p = 0.167$) and that of TgAb and thyroid volume ($r = –0.119$, $p = 0.238$) was not significant.
Conclusion: The prevalence of thyroid autoantibodies in patients with goitre is high in Nigeria. Thyroid peroxidase antibody is more prevalent than thyroglobulin antibody in thyroid disorders and appears to be a better marker than thyroglobulin antibody in detecting autoimmune thyroid dysfunction.

Keywords: Thyroid autoantibodies, Goitre, Autoimmune Thyroid disease

Introduction

Thyroid disorders are the second most common endocrine disorder in Nigeria after diabetes mellitus.[1] The World Health Organization (WHO) classified 7% of the world population as suffering from clinically apparent goitre.[2] Most patients are in developing countries, where the disease is attributed to iodine deficiency.[3]

Endocrine disease of the thyroid may result in either under- or over-activity of the gland. This may be due to congenital factors, inadequate levels of dietary iodine intake, pregnancy, radiotherapy, viral infection, surgery, underlying disease such as infiltrative disorders or autoimmunity.[4–6]

The classic autoimmune thyroid disorders, Graves' disease (GD) and Hashimoto's thyroiditis (HT), are characterised by the presence of elevated levels of serum antibodies directed against thyroid antigens, namely thyroglobulin antibody (TgAb) and thyroid peroxidase antibody (TPOAb).[7] Other autoantibodies in autoimmune thyroid disorders include thyroid stimulating hormone receptor antibody, which is specific for Graves' disease and antibody to sodium iodide symporter, which currently has no demonstrable diagnostic role in thyroid autoimmunity.[7]

In a study carried out by Olusi *et al.*[8] 4.6% of patients with goitre were found to have significantly positive autoantibody titres against thyroglobulin (Tg) while none of the 59 normal controls matched for age and sex had demonstrable autoantibodies. Isichei *et al.*,[9] in a survey of endemic goitre in Jos, showed that goitre is highly endemic in the area with prevalence varying from 1% to 23%. Females showed a markedly higher prevalence of goitre. Though urine samples indicated that iodine excretion was similar to that in iodine-deficient areas of the world, no relationship was observed between the prevalence of goitre and urinary iodine. It could therefore not be concluded that the aetiology of endemic goitre in this area was associated with iodine deficiency. It was thus concluded that endemic goitre may be an interplay of multiple factors of aetiological importance. Okosieme *et al.*,[10] in a study on the prevalence of thyroid antibodies in Nigerian patients, found that TgAb and TPOAb were found in 4% and 7%, respectively, of healthy adult controls, 11.6% and 76.8% of patients with GD, 25% and 12.5% of patients with toxic nodular goitre (TNG) and 9.52% and 14.29% of patients with simple non-toxic goitre (SNTG). The prevalence of thyroid autoantibodies found by Okosieme *et al.*[10] was higher than that reported in previous studies in Africans.[11,12] This may be due to the use of agglutination method in previous studies, a less sensitive method compared with enzyme-linked immunosorbent assay (ELISA), which was used by Okosieme *et al.*

Autoimmune thyroid disease (AITD) is a common organ-specific autoimmune disorder resulting in dysfunction of the thyroid gland. AITD includes chronic autoimmune thyroiditis or

Hashimoto's thyroiditis (HT) and its variants (painless postpartum and sporadic thyroiditis), autoimmune atrophic thyroiditis or primary myxoedema and Graves' disease (GD). Genetic and environmental factors appear to interact, leading to the formation of autoantigens and accumulation of antigen-presenting cells (APCs) in the thyroid. Due to loss of immune tolerance, autoreactive immune cells activated by APCs invade the thyroid gland, interacting with thyroid cells.

Hashimoto's thyroiditis and atrophic thyroiditis are differentiated from each other based on clinical findings. Hashimoto's thyroiditis is characterised by the presence of goitre, thyroid autoantibodies against thyroid peroxidase and thyroglobulin in the serum and varying degrees of thyroid dysfunction. It results from immune response, which leads to infiltration of autoantigen-specific lymphoid cells and destruction of thyroid follicles. The intrathyroidal lymphocytes are both T and B lymphocytes, with predominant Th-1 subtype. The overall effect is hypothyroidism due to destruction of thyroid cells. Atrophic thyroiditis is characterised by a small thyroid gland with lymphocytic infiltration and replacement of normal thyroid parenchyma by fibrous tissue. It presents with clinical hypothyroidism. Graves' disease is characterised by follicular hyperplasia, patchy lymphocytic infiltration of the thyroid and occasional formation of lymphoid germinal centres.

The predominant thyroid-infiltrating T lymphocytes act mainly as CD4+ Th2 cells. Graves' disease is due to antibodies to the thyroid stimulating hormone receptor (TSHR), which stimulate thyroid growth and function.

Autoimmune diseases of the thyroid gland are polygenic disorders resulting from the combination of a genetic predisposition and an environmental trigger. Genetic factors are predominant, accounting for 80%, and environmental factors, accounting for 20%, of susceptibility to develop autoimmune thyroid disease.

Environmental factors implicated in the development of autoimmune diseases include iodine, drugs like amiodarone, interferon-α, interleukin-2, highly active antiretroviral therapy, infectious organisms, cigarette smoking, selenium intake, stressful events, external and internal radiation. Antibodies produced in response to certain infectious agents like *Yersinia enterocolitica* react with human cell proteins, due to their structural resemblance. Other precipitating or predisposing factors include sex steroids and trauma.

Iodine is an important environmental agent known to increase the risk of thyroid autoimmunity. Studies support a role for iodine in the initiation and promotion of autoimmune thyroid disease. Studies have shown that the appearance of thyroid autoantibodies has been associated with iodination of salt in iodine-deficient areas.[13,14] Several mechanisms have been proposed for the induction of thyroid autoimmunity by excess iodine. Iodination of thyroglobulin increases its immunogenicity by altering its stereochemical structure, leading to the production of iodine-containing determinants and the loss of some and appearance of other hidden epitopes. These may enhance the presentation of thyroglobulin by antigen presenting cells and increase the affinity of the T lymphocyte receptor (TCR) for the thyroglobulin, leading to specific T lymphocyte activation. Another mechanism is toxic destruction of thyroid cells through the generation of oxygen radicals. Excessive amounts of the iodide ion are oxidised by thyroid peroxidase producing large amounts of oxidative intermediates and these molecules are capable of oxidising membrane lipids and proteins, thus damaging thyroid cell membranes. Iodine also has direct stimulation effects on macrophages, dendritic cells, and B and T lymphocytes. Enhanced macrophage myeloperoxidase activity, augmentation of dendritic cell maturation, increase in the number of circulating T lymphocytes and stimulation of immunoglobulin production are some of the possible iodine effects on the immune system.[15]

Three main thyroid autoantigens are involved in AITD. These are thyroid peroxidase (TPO), thyroglobulin (Tg) and TSH receptor. Other autoantigens, such as the sodium/iodide symporter (NIS) have also been described but are of unknown significance at this time.[16]

To date, studies on the prevalence of thyroid autoantibodies in patients with goitre in Nigeria remain sparse. Since undiagnosed thyroid diseases and autoimmunity carry considerable morbidity, it is imperative to study the relationship between thyroid function, thyroid size and thyroid autoantibodies in Nigerians. This study therefore tested the hypothesis that there is no relationship between thyroid autoantibodies and thyroid function.

Methodology

The study was conducted at the Obafemi Awolowo University Teaching Hospital Complex (OAUTHC). The study population included all patients who were 18 years and above, who presented with goitre within the study period to both the medical and surgical outpatient units of OAUTHC. It was a cross-sectional study. A data proforma was used to document demographic data and clinical parameters. The presence of thyrotoxicosis and hypothyroidism was determined through interviews, physical examination and laboratory findings. This study was conducted over a period of one year. The sample size was calculated using Fisher's formula.[17] The calculated sample size was 96; however, 100 patients were recruited for the study. Fifty apparently healthy subjects without goitre who gave their consent served as controls. Consecutive patients who presented with goitre within the study period and met the inclusion criteria were recruited and examined. Approval of the Ethics and Research committee of the Obafemi Awolowo University Teaching Hospital Complex was obtained for the study. Informed consent was obtained from each patient and healthy controls after a discussion session in the patient's best understood language.

Patients with goitre who were aged 18 years and above, treatment naive and ambulant were recruited. The exclusion criteria included unwillingness to participate in the study, pregnant women, patients on steroids, patients younger than 18 years, patients presenting with a febrile illness, patients diagnosed with cancer or on treatment for cancer, and patients known to have connective tissue disease.

Enzyme immunoassay test kits (Cusabio Biotech Company Ltd, Houston, TX, USA) were used for free thyronine (FT_3), free thyroxine (FT_4), sensitive thyroid stimulating hormone (sTSH), TPOAb and TgAb. Before proceeding with assays, all reagents, sera references for this study and controls were brought to room temperature. Test samples were serum type collected in batches and stored frozen at −20 degrees Celsius without repeated thawing and re-freezing. All assays were performed on a fully automated ELISA Microwell ChemWell 2910 auto-

analyzer (Awareness Technology Inc, Palm City, FL, USA) and in duplicate.

Participants were placed into three groups (Table 1) according to symptoms and biochemical profile. Group I: patients who had goitre and symptoms suggestive of hypothyroidism such as cold intolerance, weight gain, constipation and supporting biochemical findings of low FT_3, low FT_4 and elevated sTSH were classified as hypothyroid. Group II: patients who had goitre and symptoms suggestive of thyrotoxicosis such as heat intolerance, weight loss, hyperdefecation and supporting biochemical findings of elevated FT_4 and/or elevated FT_3 and low sTSH were classified as hyperthyroid. Group III: patients who had goitre but without symptoms suggestive of hypothyroidism or thyrotoxicosis and normal FT_3, FT_4 and sTSH were classified as euthyroid. Normal range for FT_3 was 1.4–4.2 pg/ml, normal range for FT_4 was 0.8–2.0 ng/dl, normal range for sTSH was 0.5–5.6 UIU/ml, elevated TPOAb was defined as level > 40 IU/ml while elevated TgAb was defined as level > 125 IU/ml.

Data were entered and analysed using the Statistical Package for the Social Sciences (SPSS) version 17 (SPSS Inc, Chicago, IL, USA). The prevalence of thyroid autoantibodies in patients with goitre was determined using percentages. Descriptive analyses were presented with frequency tables, charts and graphs as appropriate. Association between qualitative variables was determined using chi-square analysis. Comparison of means was by Student's *t*-test when two groups were considered and analysis of variance (ANOVA) was used when more than two groups were involved. Mann–Whitney *U* and Kruskal–Wallis *H* tests were used for skewed data. Correlation of means between groups was assessed using Pearson's correlation coefficient. The level of statistical significance was set at $p < 0.05$.

Results

A total of 150 subjects participated in the study. This comprised 100 subjects with goitre and 50 apparently healthy subjects without goitre. Characteristics of the study population are given in Table 2. The age range for the subjects with goitre was 18–70 years while that of the controls was 22–65 years. The mean (±SD) age for subjects with goitre was 44.6 ± 13.8 years while that of the controls was 43.5 ± 16.7 years ($p = 0.681$, $t = 0.412$).

Male subjects accounted for 17 (11.3%) of the overall population studied while 133 (88.7%) subjects were female. Among the subjects with goitre, 12 (12%) were male while 88 (88%) were female giving a female to male ratio of 7.3:1. Among the control group, 5 (10%) were male and 45 (90%) were female. There was no statistical difference in the sex distribution of the subjects with goitre and the controls ($p = 0.716$, $\chi^2 = 0.133$).

Table 1: Definition of subjects with goitre

Groups	Symptoms	Biochemical profile
I: Hypo	Symptoms of hypothyroidism	Low FT_3
		Low FT_4
		Elevated sTSH
II: Hyper	Symptoms of hyperthyroidism	Elevated FT_4 and/or elevated
		FT_3
		Low sTSH
III: Euthy	No symptoms of hypo- or hyperthyroidim	Normal FT_3
		Normal FT_4
		Normal sTSH

Hypo = hypothyroidism, Hyper = hyperthyroidism, Euthy = euthyroidism, FT_3 = free triiodothyronine, FT_4 = free thyroxine, sTSH = sensitive thyroid stimulating hormone.

Table 2: Characteristics of study population

Parameter	Subjects *n* (%)	Controls *n* (%)	*p*-value
Mean age ± SD (Years)	44.6 ± 13.8	43.5 ± 16.7	0.681
Gender:			
Female	88 (88)	45 (90)	0.716
Male	12 (12)	5 (10)	
IID	86 (86)	50 (100)	0.021
FHG	14 (14)	–	0.005
HGN	7 (7)	–	0.096

SD = standard deviation, IID = ingestion of iodized salt, FHG = family history of goitre, HGN = history of goitre in neighbourhood.

Eighty-six (86%) subjects with goitre used iodised salt in their food while all the subjects in the control group ingested iodised salt. Some 14% of the subjects with goitre had a family history of a similar neck swelling while only 7% of them had a history of a similar neck swelling in their neighbourhood. None of the subjects in the control group reported a history of anterior neck swelling in members of their family or in their neighbourhood.

The mean (±SD) duration of neck swelling in subjects with goitre was 34.0 ± 48.9 months. Based on the history and clinical examination findings, 60% (60) of the subjects with goitre were euthyroid, 38% (38) were hyperthyroid, while only 2% (2) were hypothyroid. Of the subjects with hyperthyroidism, 18 (47.4%) subjects had Graves' disease clinically.

The frequency of reported symptoms of thyrotoxicosis is given in Table 3. Symptoms most commonly reported by subjects with hyperthyroid goitre were: excessive sweating 38 (100%) and weight loss 38 (100%). Palpitations were reported in 33 (86.8%), hyperdefecation in 30 (78.9%) and heat intolerance in 28 (73.7%). The 2 (100%) subjects with hypothyroidism reported symptoms of constipation, cold intolerance and weight gain.

The mean ± standard deviation (SD) pulse rate in subjects with goitre was 90.1 ± 14.9 bpm while that of the control group was 79.2 ± 8.4 bpm ($p < 0.001$). The mean (± SD) pulse rate in subjects with hypothyroidism was 79.0 ± 12.7 bpm, in subjects with euthyroidism 79.6 ± 8.3 bpm and in subjects with hyperthyroidism 106.3 ± 5.9 bpm ($p < 0.001$).

The mean systolic blood pressure (±SD) in subjects with hypothyroidism, euthyroidism and hyperthyroidism was 111.0 ± 1.4 mmHg, 122.8 ± 13.1 mmHg and 137 ± 19.3 mmHg respectively ($p < 0.001$) while the mean diastolic blood

Table 3: Frequency of reported symptoms of thyrotoxicosis

Symptoms	Frequency *n* (%)
Excessive sweating	38 (100%)
Weight loss	38 (100%)
Palpitation	33 (86.8%)
Hyperdefecation	30 (78.9%)
Heat intolerance	28 (73.7%)

Table 4: Intra-assay and inter-assay precision for thyroid function tests and thyroid autoantibodies

Variable	Intra-assay precision (%)	Inter-assay precision (%)
FT_3 (pg/ml)	5.47	7.77
FT_4 (ng/dl)	3.25	6.01
sTSH (UIU/ml)	7.1	7.7
TPOAb (IU/ml)	4.6	5.8
TgAb (IU/ml)	4.2	4.7

FT_3 = free triiodothyronine, FT_4 = free thyroxine, sTSH = sensitive thyroid stimulating hormone, TPOAb = thyroid peroxidase antibody, TgAb = thyroglobulin antibody.

pressure ± SD in hypothyroid, euthyroid and hyperthyroid goitres was 65.0 ± 7.1 mmHg, 71.5 ± 9.1 mmHg and 69.7 ± 10.9 mmHg respectively ($p = 0.853$). The mean systolic and diastolic blood pressure for the control group were 112.1 ± 9.3 mmHg and 66.9 ± 5.9 mmHg respectively.

The results of intra-assay and inter-assay precision for thyroid function tests and thyroid autoantibodies are given in Table 4. The coefficients of variation were within acceptable limits.

Elevated TPOAb was found in 35 (35%) subjects with goitre and 4 (8%) of the control group ($p < 0.001$), and elevated TgAb was found in 24 (24%) subjects with goitre and 6 (12%) subjects without goitre. Table 5 compares the prevalence of thyroid autoantibodies between subjects with goitre and the control group.

The mean TPOAb ± SD in the subjects with goitre was 128.1 ± 204.6 IU/ml while that of the control group was 38.6 ± 104.7 IU/ml ($p = 0.001$). The mean TgAb ± SD in the subjects with goitre was 109.3 ± 112.3 while that of the control group was 73.7 ± 53.1 IU/ml ($p = 0.035$). Table 6 shows the prevalence of thyroid autoantibodies among the various groups with goitre. The median concentrations of TPOAb in subjects with hypothyroidism, hyperthyroidism and euthyroidism were 273.1, 229.5 and 101.5 IU/ml respectively, while the median levels of TgAb were 124.7, 113.9 and 64.2 IU/ml, respectively.

Discussion

The mean (±SD) age of occurrence of goitre in this study was 44.6 ± 13.8 years. This is similar to the mean age observed in earlier studies of thyroid disorders by Ogbera *et al.*[1] in Lagos, Kolawole[18] in Ile-Ife and Chehade *et al.*[19] in the United States, who found a mean age of 40 ± 12.4 years, 42.7 ± 12.6 years and 47.8 ± 14.9 years respectively. Most of the subjects with goitre were female with a female to male ratio of 7.3:1. This is also similar to the female to male ratio observed from other studies in this environment.[1,18,20,21] Female preponderance is expected in this study as thyroid disorders occur more commonly in females compared with males. Being female carries a 10–20-fold risk of developing autoimmune disease compared with being male. This association does not apply only to autoimmune thyroid disease but also applies to the development

Table 5: Comparison of the prevalence of thyroid autoantibodies between subjects with goitre and the control group

Variable	Subjects with goitre *n* (%)	Control *n* (%)	*p*-value
TPOAb	35 (35%)	4 (8%)	< 0.001
TgAb	24 (24%)	6 (12%)	0.083

TPOAb = thyroid peroxidase antibody, TgAb = thyroglobulin antibody.

Table 6: Comparison of the prevalence of thyroid autoantibodies among the different groups with goitre.

Variable	Hypo *n* (%)	Euthy *n* (%)	Hyper *n* (%)	*p*-value
TPOAb	2 (100%)	4 (7%)	29 (76.3%)	< 0.001
TgAb	1 (50%)	9 (15.8%)	14 (36.8%)	0.068

Hypo = hypothyroidism, Euthy = euthyroidism, Hyper = hyperthyroidism, TPOAb = thyroid peroxidase antibody, TgAb = thyroglobulin antibody.

of multinodular goitre and differentiated thyroid carcinoma but not undifferentiated thyroid carcinoma.[22] The mechanism of this is not clear, but it has been suggested that females generally have greater reactivity of the thyroid gland, or subject it more to greater stress. It has been suggested that there may be specific receptors on the promoter for human leukocyte antigen-D related (HLA-DR) genes that make them responsive to the oestrogen receptor.[22]

The two most common symptoms of hyperthyroidism observed in this study were excessive sweating and weight loss. These symptoms were among the five most common symptoms reported by Ogbera *et al.*[1] In a study of 44 Nigerians with thyrotoxicosis, Famuyiwa and Bella[23] also observed similar features but weight loss and palpitations appeared to be the two most frequent symptoms. The frequency of symptoms of thyrotoxicosis reported in this study are comparable to those reported in Caucasians.[19,24]

Elevated thyroid peroxidase antibody (TPOAb) was found in 35% of subjects with goitre but only in 8% of the control group ($p < 0.001$). These findings agree with the report by Okosieme *et al.*[10] in a study of the prevalence of thyroid autoantibodies in Nigerian patients. These findings were also comparable to those of AL-Naqdy *et al.*[25] and Kuria and Amayo,[26] who reported prevalence rates of 39% and 51.4% respectively. A higher prevalence of 89% was found by Shinto *et al.*,[27] who studied subjects with histologically proven autoimmune thyroid disease. In this study, TPOAb was elevated in 76.7% of subjects with hyperthyroid goitre. Chiyanga *et al.*[12] and Hawa *et al.*,[28] however, reported a lower prevalence of 39% and 44% respectively in subjects with hyperthyroidism. The higher prevalence of elevated TPOAb in hyperthyroid subjects in this study could be due to the fact that about half of these patients had Graves' disease clinically. All subjects with hypothyroid goitre had elevated TPOAb and this result is comparable to the findings of Chaieb *et al.*[29] The prevalence of TPOAb observed in this study is higher than previously reported in Africans[12,28,30] and this may reflect an increase in the prevalence of autoimmune thyroid disorders. There is strong evidence that the pattern of thyroid disorders in a population is dependent on environmental iodine intake.[31] Iodine deficiency disorders abound in areas with inadequate iodine intake while autoimmune thyroid disorders are rare in iodine deficiency but become more prevalent with transition to iodine sufficiency.[32] In this study most of the subjects with goitre ingested iodised salt and this may indicate transition to iodine sufficiency, and therefore increased prevalence of autoimmune thyroid disorders.

Elevated thyroglobulin antibody (TgAb) was found in 24% of the subjects with goitre and 12% of the control group. Okosieme *et al.*[10] also found a prevalence of 9.52–25% in the subjects with goitre. The prevalence of TgAb found in this study was also comparable to the prevalence of 36.1% reported by Kuria and Amayo.[26] Elevated TgAb was found in 36.8% of the subjects

with hyperthyroidism, 50% of subjects with hypothyroidism and 15.8% of subjects with euthyroid goitre. Chiyanga *et al.*[12] and Kuria and Amayo[26] also found a comparable prevalence of 39% and 33% respectively in subjects with thyrotoxicosis.

In this study, thyroid peroxidase antibodies were observed to be more prevalent than thyroglobulin antibodies in subjects with hypothyroidism and hyperthyroidism. This may reflect the fact that TPOAb is a more specific test than TgAb for detecting autoimmune thyroid diseases.

The mean TPOAb and TgAb were found to be significantly higher in subjects with goitre when compared with the control group ($p = 0.001$ and 0.035 respectively). Comparison of the median levels of TPOAb and TgAb among subjects with hypothyroidism, euthyroidism and hyperthyroidism was found to be statistically significant only for TPOAb ($p < 0.001$ and $p = 0.893$ respectively). The median level of TPOAb was highest in subjects with hypothyroidism, followed by hyperthyroidism and euthyroidism. TPOAb was elevated in 100% of subjects with hypothyroid goitre, but in 76.3% and 7% of subjects with hyperthyroid and euthyroid goitre respectively. TPOAb thus tends to be more commonly associated with thyroid dysfunction of autoimmune origin than TgAb, and elevated TPOAb is mostly associated with hypothyroidism. These findings are similar to those previously reported by Okosieme *et al.*[10] and other authors.[27,33,34] The findings in this study are in keeping with the fact that high titres of TPOAb and TgAb are generally found in patients with autoimmune thyroid diseases. These more frequently occur in subjects with hypothyroidism compared with hyperthyroidism and occasionally are found in euthyroid goitres. Thyroid peroxidase antibody is found in up to 95% of subjects with autoimmune hypothyroidism and in 70–80% of subjects with Graves' disease, which commonly presents with hyperthyroidism. Thyroglobulin antibody also occurs more frequently in subjects with autoimmune hypothyroidism than in subjects with Graves' disease.

Conclusions

The prevalence of thyroid autoantibodies in patients with goitre in Nigeria is higher than previously reported. Thyroid peroxidase antibody is more prevalent than thyroglobulin antibody in thyroid disorders and it appears to be a better marker than thyroglobulin antibody in detecting autoimmune thyroid dysfunction. In a resource-challenged setting where testing for thyroid autoantibodies is expensive and not readily available, screening for autoimmune thyroid dysfunction may be done by testing for thyroid peroxidase antibody alone.

Disclosure statement – No potential conflict of interest was reported by the authors.

ORCID

OA Ojo http://orcid.org/0000-0002-6576-9596
BA Kolawole http://orcid.org/0000-0002-8242-3968
OE Ojo http://orcid.org/0000-0001-9429-4000

References

1. Ogbera AO, Fasanmade O, Adediran O. Pattern of thyroid disorders in the Southwestern Region of Nigeria. Ethn Dis. 2007;17:327–30.
2. Kally FC, Snedden WW. Prevalence and Geographical distribution of endemic goitre. Bull World Health Organ. 1958;18:5–173.
3. Okosieme OE. Impact of iodination on thyroid pathology in Africa. J R Soc Med. 2006;99:396–401.
4. Vanderpump MP, Tunbridge F. Epidemiology and prevention of clinical and subclinical hypothyroidism. Thyroid. 2002;12:839–47.
5. Wiersinga WM. Sub clinical hypothyroidism and hyperthyroidism. I.prevalence and clinical relevance. Neth J Med. 1995;46:197–204.
6. Brownlie BE, Wells JE. The epidemiology of thyrotoxicosis in New Zealand: incidence and geographical distribution in north Canterbury, 1983-1985. Clin Endocrinol. 1990;33:249–59.
7. Weetman AP, McGregor AM. Autoimmune thyroid disease: developments in our understanding. Endocr Rev. 1994;15:788–802.
8. Olusi SO, Ogunrin OA, Oluwole SF. Serum thyroglobulin autoantibody titres and immunoglobulin concentrations in Nigerians with goitre. East Afr Med J. 1991;68:261–5.
9. Isichei UP, Morimoto I, Das SC, et al. Endemic goitre in the Jos Plateau region of northern Nigeria. Endocr J. 1995;42:23–9.
10. Okosieme OE, Taylor RC, Ohwovoriole AE, et al. Prevalence of thyroid antibodies in Nigerian patients. QJM. 2007;100:107–12.
11. Njemini R, Meyers I, Demanet C, et al. The prevalence of autoantibodies in an elderly sub-Saharan African population. Clin Exp Immunol. 2002;127:99–106.
12. Chinyanga EA, Benni A, Siziya S. Thyroid status and the levels of thyroid auto- antibodies in the sera of hyperthyroid and goitrous subjects. Cent Afr J Med. 2000;46:251–5.
13. Zois C, Stavrou I, Kalogera C, et al. High prevalence of autoimmune thyroiditis in school children after elimination of iodine deficiency in northwestern Greece. Thyroid. 2003;13:485–9.
14. Premawardhana LD, Parkes AB, Smyth PP, et al. Increased prevalence of thyroglobulin antibodies in Sri Lankan schoolgirls—is iodine cause? Eur J Endocrinol. 2000;143:185–8.
15. Luo Y, Kawashima A, Ishido Y, et al. Iodine excess as an environmental risk Factor for autoimmune thyroid disease. Int J Mol Sci. 2014;15:12895–912.
16. Marcocci C, Chiovato L. Thyroid -directed antibodies. In: Thyroid BL, editor. AU Williams and Wilkins. Philadelphia: Lippincott; 2000. p. 414–31.
17. Araoye MO. Subjects Selection. In: Research Methodology with Statistics for Social Sciences. 1st Edition. Ilorin: Nathadex Publishers; 2004:115–29.
18. Kolawole BA. Study of Cardiovascular function in Thyrotoxic patients. Part II Dissertation, West African College of Physicians 1999.
19. Chehade JM, Lim W, Silverberg AB, et al. The incidence of Hashimoto's disease in nodular goitre: the concordance in serological and cytological findings. Int J Clin Pract. 2010;64:29–33.
20. Ikem R, Adebayo J, Soyoye D, et al. Spectrum of thyroid disorders in Obafemi Awolowo University teaching hospital complex, Ile-Ife. Presented at Society for Endocrinology BES 2010, Harrogate, UK. Endocrine Abstracts 21 P366.
21. Ogbera AO. A two year audit of thyroid disorders in an urban hospital in Nigeria. Nig Q Hosp Med. 2010;20:81–5.
22. De Groot LJ. Graves' disease and the Manifestations of Thyrotoxicosis. www.endotext.com 6:36pm 14/06/12.
23. Famuyiwa OO, Bella AF. Thyrotoxicosis in Nigeria: analysis of a five year experience. Trop Geogr Med. 1990;42:248–54.
24. Boelaert K, Torlinska B, Holder RL, et al. Hyperthyroidism. Clin Thyroidol. 2010;22:8–11.
25. AL-Naqdy A, Kutty I, AL- Harthi S, et al. Anti-thyroglobulin and Anti-thyroid Microsomal Antibodies in thyroid disorders. Bahrain Med Bull. 2003;25:1–8.
26. Kuria JG, Amayo A. Prevalence of antithyroid antibodies in patients with primary thyroid disorders. East Afr Med J. 2008;85:459–62.
27. Shinto AS, Pachen L, Sreekanth TK, et al. Prevalence of Antithyroid antibodies in histologically proven autoimmune thyroid diseases and correlation with thyroid dysfunction in South India. Thyroid Science. 2010;5:1–5.
28. Hawa MI, Picardi A, Constanza F, et al. Frequency of diabetes and thyroid autoantibodies in patients with autoimmune endocrine disease from Cameroon. Clin Immunol. 2006;118:229–32.
29. Chaieb L, Chaieb MC, Zebidi A, et al. Antithyroid antibodies. prevalence in primary hypothyroidism in Central Tunisia. Ann Endocrinol. 1991;52:331–3.

30. Cardoso C, Ohwovoriole AE, Kuku SF. A study of thyroid function and prevalence of thyroid autoantibodies in an African diabetic population. J Diabetes Complications. 1995;9:37–41.
31. Laurberg P, Pedersen KM, Hreiderson A, et al. Iodine intake and the pattern of thyroid disorders; a comparative epidemiological study of thyroid abnormalities in the elderly in Iceland and in Jutland, Denmark. J Clin Endocrinol Metab. 1998;83:765–9.
32. Doufas AG, Mastorakos G, Chatziioannou S, et al. The predominant form of non toxic goitre in Greece is now thyroiditis. Eur Jcrinol. 1999;140:505–11.
33. Zelaya AS, Stott A, Nader S, et al. Antithyroid peroxidase antibodies in patients with high normal range thyroid stimulating hormone. Fam Med. 2010;42:111–5.
34. Hoogendoom EH, Hermus AR, Vegt F, et al. Thyroid function and prevalence of Anti- thyroid peroxidase Antibodies in a population with borderline Sufficient iodine intake; influences of Age and Sex. Clin Chem. 2006;52:104–11.

Received: 16-01-2019 Accepted: 3-07-2019