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

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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. The World Health Organization (WHO) classified 7% of the world population as suffering from clinically apparent goitre. Most patients are in developing countries, where the disease is attributed to iodine deficiency.

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.

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). Other autoimmune thyroid antibodies 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.

In a study carried out by Olusi et al., 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., 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., 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., was higher than that reported in previous studies in Africans. 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-
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 FT3, low FT4 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 FT3 and/or elevated FT4 and low sTSH were classified as hyperthyroid. Group III: patients who had goitre but without symptoms suggestive of hypothyroidism or thyrotoxicosis and normal FT3, FT4 and sTSH were classified as euthyroid.

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 \( (p = 0.716, \chi^2 = 0.133) \).

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 hyperthyroidism 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 pressure was 78.3 ± 5.9 mmHg, 74.5 ± 10.4 mmHg and 83.7 ± 10.0 mmHg respectively.\( ^{16} \)

### Table 2: Characteristics of study population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Subjects</th>
<th>Controls</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age ± SD (Years)</td>
<td>44.6 ± 13.8</td>
<td>43.5 ± 16.7</td>
<td>0.681</td>
</tr>
<tr>
<td>Gender:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>88 (88)</td>
<td>45 (90)</td>
<td>0.716</td>
</tr>
<tr>
<td>Male</td>
<td>12 (12)</td>
<td>5 (10)</td>
<td></td>
</tr>
<tr>
<td>IOD</td>
<td>86 (86)</td>
<td>50 (100)</td>
<td>0.021</td>
</tr>
<tr>
<td>FHG</td>
<td>14 (14)</td>
<td>–</td>
<td>0.005</td>
</tr>
<tr>
<td>HGN</td>
<td>7 (7)</td>
<td>–</td>
<td>0.096</td>
</tr>
</tbody>
</table>

\( SD = \) standard deviation, \( IOD = \) ingestion of iodized salt, \( FHG = \) family history of goitre, \( HGN = \) history of goitre in neighbourhood.

### Table 3: Frequency of reported symptoms of thyrotoxicosis

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Frequency ( n (%) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excessive sweating</td>
<td>38 (100)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>38 (100)</td>
</tr>
<tr>
<td>Palpitation</td>
<td>33 (86.8%)</td>
</tr>
<tr>
<td>Hyperdefecation</td>
<td>30 (78.9%)</td>
</tr>
<tr>
<td>Heat intolerance</td>
<td>28 (73.7%)</td>
</tr>
</tbody>
</table>

\( ^{16} \)
levels of TgAb were 124.7, 113.9 and 64.2 IU/ml, respectively. The median concentrations of TPOAb in subjects with goitre was 73.7 ± 53.1 IU/ml (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). 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 204.6 IU/ml while that of the control group was 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 38.6 ± 36.9 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, Kolawole18 in Ille-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 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 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 Bella23 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 is comparable to those reported in Caucasians.10,24

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.,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 Africans12,26,30 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% 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...

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intra-assay precision (%)</th>
<th>Inter-assay precision (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT3 (pg/ml)</td>
<td>5.47</td>
<td>7.77</td>
</tr>
<tr>
<td>FT4 (ng/dl)</td>
<td>3.25</td>
<td>6.01</td>
</tr>
<tr>
<td>sTSH (IU/ml)</td>
<td>7.1</td>
<td>7.7</td>
</tr>
<tr>
<td>TPOAb (IU/ml)</td>
<td>4.6</td>
<td>5.8</td>
</tr>
<tr>
<td>TgAb (IU/ml)</td>
<td>4.2</td>
<td>4.7</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects with goitre n (%)</th>
<th>Control n (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TPOAb</td>
<td>35 (35%)</td>
<td>4 (8%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>TgAb</td>
<td>24 (24%)</td>
<td>6 (12%)</td>
<td>0.083</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hypo n (%)</th>
<th>Euthy n (%)</th>
<th>Hyper n (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TPOAb</td>
<td>2 (100%)</td>
<td>4 (7%)</td>
<td>29 (76.3%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>TgAb</td>
<td>1 (50%)</td>
<td>9 (15.8%)</td>
<td>14 (36.8%)</td>
<td>0.068</td>
</tr>
</tbody>
</table>

Hypo = hypothyroidism, Euthy = euthyroidism, Hyper = hyperthyroidism, TPOAb = thyroid peroxidase antibody, TgAb = thyroglobulin antibody.
with hyperthyroidism, 50% of subjects with hypothyroidism and 15.8% of subjects with euthyroid goitre. Chiyanja et al.12 and Kuria and Amayo28 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 0.035 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.7,33,34 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.

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