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# Aberrations in serum proteins and thyroid size in Nigerians on the Jos Plateau and their relation to thyrometabolic function

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**Summary**

Controlled serum protein and thyroid function studies were carried out among 208 African patients with endemic goitre. The changes seen in the serum protein constituents were studied and evaluated in relation to thyroid hormone levels, goitre grade and the clinical state of the patient. Mean serum TBG and thyroglobulin values rose sharply in the early phase of goitre, and remained elevated throughout. Although the mean serum total T4 was statistically and significantly higher in endemic goitre compared with the normal subjects ($t$ = 3.72. $P$ = 0.005), the free thyroxine index was significantly lower and serum T4 fell progressively ($r$ = 0.975) with increasing thyroid grade. In contrast, mean serum T3 rose continuously ($r$ = 0.910) but appeared inflated due to a proportionate increase in TBG in several cases, giving a misleading picture of T3 — thyrotoxicosis in otherwise clinically normal subjects. An increase in T3/T4 ratio and consequently in thyroid function, therefore occurs with advancing thyroid size. Inconsistent binding of T4 to TBG causes wide variations in thyroxine index. Mean serum albumin and betalipoprotein appeared inversely related to thyroid function. These changes imply that in order to evaluate a patient with endemic goitre accurately a detailed biochemical investigation of the thyroid may be necessary.

**Résumé**

Etudes sur le sérum protéin controllé et des activités de thyroïde étaient effectués auprès de 208 patients africains du goître endémique. Les changements constates dans les constituants du sérum protéin etaient étudiés et évalués en relation avec les niveaux d'hormone de thyroide, le niveau de goitre et l'état clinique du patient. Les valeurs moyens du sérum TBG et de thyroglobuline ont monté brusquement dans la phase initiale du goître, restant éntièrement élevées.

Quoique le moyen du sérum total T4 était statistiquement et significativement plus élevé dans le goître endémique, vis à vis les sujets normals ($t$ = 3.72, $P$ = 0.05), l'index de thyroxine libre était significativement inférieur alors que le sérum T4 baisse progresivement ($r$ = 0.975) avec une augmentation du niveau de thyroïde. Par contre, le moyen du sérum T3 augmentait sans interruption ($r$ = 0.910) mais semblait exaggéré à cause d'une augmentation proportionnelle de TBG dans plusieurs cas, donnant une image trompeuse de T3 — thyrotoxicosis pour les sujets autrement cliniquement normals. Une augmentation en terme du rapport T3/T4 et conséquemment à l'égard du fonctionnement de thyroïde se produit ainsi, avec une evolution de la taille de thyroïde. La fixation inconsistante du T4 au TBG provoque de grandes variations en ce qui concerne le rendement fixatif de la thyroxine et l'index de thyroxine libre. Le moyen du sérum albumin et de bétalipoprotéin semblait inversement proportionnel à la fonction de la thyroïde. Ces changements indiquent que pour une évaluation précise d'un patient du goître endemique, il faut faire une enquêtre biochemique de détail de la thyroïde.

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## Introduction

Serum protein changes in health [1,2,3] and disease [4,5,6] have been studied to some extent in the past amongst Africans. In some respects they differ from Caucasians. Hardly is any information, however, available on serum protein changes in thyroid disease generally, and endemic goitre in particular. Most of the current laboratory methods for thyroid function investigations depend on the measurement of total thyroid hormone content of serum, which in turn depends on the amount of

hormone-binding proteins present. The bound hormones constitute over 99 per cent of thyroid hormones but is physiologically inactive, in contrast to the free hormone which determines the clinical state of the patient. The knowledge of the variations which occur in serum protein patterns in thyroid disease, therefore, is vital for accurate laboratory evaluation of thyroid function for the diagnosis of thyroid disease. This study which provides a detailed picture of serum protein profile in endemic goitre discusses the alterations seen in the serum of 208 African patients with varying degrees of thyroid enlargement, and evaluates their significance and impact on thyroid function parameters.

## Materials and methods

### *Clinical materials*

Two hundred and eight blood samples were collected from a group of patients with endemic goitre residing in the endemic region of Plateau State in Northern Nigeria. The patients were randomly selected from the 4989 subjects examined by us during a thyroid disease survey [7]. The enlarged thyroid glands were graded from 1–4 according to the classification recommended by DeMaeyer [8]. Eighty samples were also collected from a control group of clinically normal subjects with no visible or palpable evidence of thyroid enlargement.

### *Laboratory materials*

The materials used for the determination of the various serum protein constituents were supplied by Boehringer Mannheim GmbH, W. Germany. Test kits were used to estimate the concentration of total protein, albumin, lipoprotein and immunoglobulins; immunodiagnostic kits (ELISA) for the estimation of total serum thyroxine (T4) triiodothyronine (T3), thyroxine binding globulin (TBG) and thyroxine binding capacity (TBC). The materials for the determination of thyroglobulin and thyroid antibodies were obtained from Japan through JICA (Japanese International Co-operation Agency).

All tests were carried out before the expiry dates indicated on the various kits, and the precautions for proper storage of serum samples as well as storage and reconstitution of reagents were adhered to. Control samples, as well as standards, were put through the assay procedure, and the reactions of the test samples related to the control and standard reactions.

### *Methods*

Approximately 10 ml of blood was collected from each subject or from patient. After allowing adequate time for clot retraction, the unhaemolysed serum was separated from the cells by centrifugation. The average serum volume obtained was 4 ml from which the serum proteins and thyroid parameters were determined. In the ELISA technique, the tests on each sample were carried out in triplicate and for duplicate analysis to give six different observations on each sample. Variations between two duplicate analyses as determined by students *t* test was found to be statistically insignificant at 5% level of significance.

The determination of thyroid hormones and values for TBG and TBC was based on the competitive protein binding technique involving two incubation periods. In the first incubation step, serum T4, T3, TBG or TBC and POD — labelled T4, T3, TBG or TBC (POD conjugate soln la) compete for a limited quantity of specific antibodies coated onto the inside wall of the test vial. After a "bound-free" separation step, the addition of $H_20_2$ and a chromogen (ABTS) results in formation of a coloured complex whose concentration is proportional to the enzyme activity bound to the vial wall. The intensities of the coloured complexes formed were measured against the substrate/buffer solutions after incubation for the specified time. The test principle for thyroid stimulating hormone (TSH) is different from the others being based on the "sandwich" principle. All the results were obtained from a calibration curve set up by using the standards provided in the various kits.

Serum thyroglobulin was determined by RIA. The detection of circulating thyroid antibodies was done by measuring the titre in serum through indirect agglutination. The biuret technique was applied in the estimation of serum total protein. Serum albumin was determined by the dye binding method using bromocresolgreen as described by Doumas *et al.* (1971). Beta- lipoprotein levels were determined with special kits provided by Boehringer Mannaheim, GmbH, W. Germany. The immunoglobulins (IgD, IgA, IgM)

were determined by the manual end-point assay method, the principle of which is based on the immunological turbidity test. The extent of turbidity of the serum samples after reaction with bovine anti-IgG, IgA, IgM was spectrophotometrically measured at 340 nm.

For comparison and to check our results with another well established method, a parallel study of some of the thyroid function parameters (serum total T4 and TSH) were carried out on the same samples by radioimmunoassay in Japan. The samples were frozen and remained frozen under special precautions in transit to Japan, where special arrangements had been made for their analysis at the First Department of Medicine, University of Nagasaki Medical School, Nagasaki, Japan by the kind courtesy of Prof. S. Nagataki and Dr. I. Morimoto under the auspices of JICA (Japan International Co-operation Agency). A reasonably good correlation ($r$ = 0.7658) was established between T4 by ELISA and T4 by RIA. Similarly we found a good correlation ($r$ = 0.7106) between TSH by ELISA and TSH by RIA. TBG values obtained were higher by RIA compared to ELISA but for the purposes of this paper the results obtained by ELISA have been used almost entirely throughout except where otherwise indicated. One possible explanation of the higher TBG in RIA is that RIA has a higher degree of sensitivity than ELISA. The results obtained by the two different methods were not pooled together but were treated, evaluated and applied separately.

Figure (A) shows our laboratory internal quality control for the ELISA technique over a period of five months.

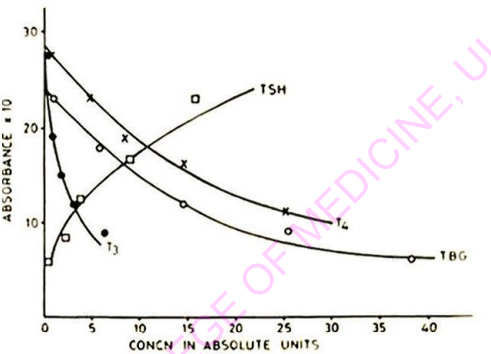


Fig. (A): The internal mean values of absorbance plotted against absolute concentrations of thyroid parameters (ELISA) over a period of 5 months.

## Results

(For details see Figs. 1–3 and Table 1–4.)

*Specific and non-specific serum proteins*

There is abundant evidence of a generalized hyperproteinaemia, affecting specific and non-specific serum proteins studies in the serum of our patients with endemic goitre compared to the normal subjects used as the control in the present investigation.

The increase in the mean serum TBG level was highly significant ($t$ = 8.74, $P$ = 0.005). The rise which occurred in the early phase of the disease was sustained throughout but there was no correlation ($r$ = 0.276) between increasing goitre grades and serum TBG levels (Fig. 1). Table 2 gives the frequency distribution of TBG in nmol/l as determined by RIA. The impact of variations in TBG on thyroid hormone levels and thyroid function is shown in Table 3 and Fig. 2. Serum Tg ranged from 0–43 ng/ml for the normal control group and from 44–1000 ng/ml for the patients with endemic goitre. The mean levels increased significantly with increase in thyroid grade and positive linear correlation ($r$ = 0.975) was seen with increasing size (Fig. 1). In contrast, tests done to detect thyroglobulin and microsomal antibodies were virtually negative.

All classes of immunoglobulin (Ig) differed considerable between the two groups of normal and pathological subjects with significantly higher values in the latter, which were almost double the former (see Table 1 and Fig. 3). The mean serum total protein was substantially higher in endemic goitre and few of the values found approached the levels sometimes seen in paraproteinaemia. Hypothyroid patients had higher mean serum albumin compared to euthyroids or the hyperthyroid group (Table 1). Similarly a three-fold increase in the mean serum

beta-lipoprotein value and the associated hypercholesterolaemia were found in thee former compared to the latter groups.

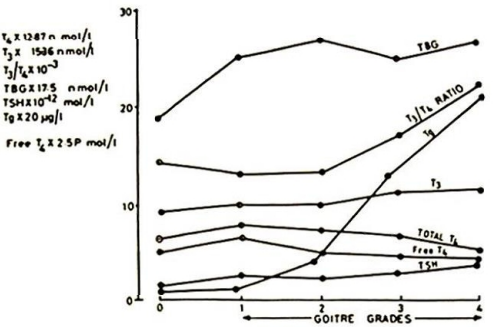


**Fig. 1:** The impact of variations in thyroid size on mean specific serum protein and parameters of thyroid function in the present investigation.
Co-efficient of correlation *(r)* with thyroid grades:
For TBG = 0.2766; T3 = 0.910; Tg = 0.7652
For T4 = 0.975; T3/T4 ratio = 0.941

*Thyroid function parameters*

There was reciprocal relationship in the concentrations of serum total T4 and T3 with progressive increase in thyroid size, as a result the T3/T4 ratio continuously increased (Fig. 1). There was a negative linear correlation ($r$ = 0.975) between serum total T4 and goitre grade, but a positive one ($r$ = 0.91) with serum T3. About 45% of the patients had T3 values above normal level.

In contrast to the picture seen in the serum total T4 value, the mean free thyroxine index ($FT_4I$) was significantly lower in the group with endemic goitre compared to that found in normal subjects ($t$ = 2.07 $P$ = 0.05). Similarly the mean T4/TBG ratio was higher in the latter compared with the former but the range was much wider in the goitrous group, which had hypothyroid as well as hyperthyroid values. A linear correlation was found between goitre grades and mean thyroid

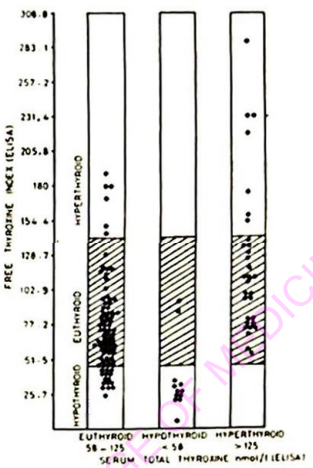


**Fig. 2:** The relationship between serum total thyroxine and the free thyroxine index in the present investigation illustrating errors in diagnosis.

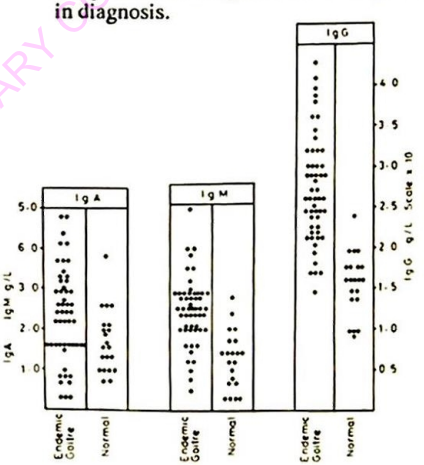


**Fig. 3:** The unusually high levels of immunoglobulin (IgG, IgA and IgM) seen in the individual African patients with endemic goitre compared to the levels seen in normal control subjects in the present investigation.

stimulating hormone (TSH) levels ($r$ = 0.851), but the values seen were considered generally low for patients with hypofunctional thyroid glands and low serum T4. The raised T3 values found in this group may have been responsible for this finding. A linear correlation analysis between thyroxine

binding globulin and age of patients was not significant ($r$ = 0.3498) but a fair correlation coefficient ($r$ = 0.5196) was established between thyroxine binding globulin (TBG) and thyroid stimulating hormone (TSH).

## Discussion

The most important aberration in thyroid function parameters seen in our patients, is a falsely elevated serum T4 (Fig. 2). This is caused by a proportionate increase in TBG and carries with it the risk of erroneous diagnosis and management. Estimation of serum total T4 alone has, therefore, no place in the evaluation of thyroid function in African patients with endemic goitre. It is imperative to determine thyroxine binding capacity (TBC), for the derivation of the free thyroxine index ($FT_4I$) which provides more accurate information about the patient. Although this index and the T4/TBG ratio which is said to provide a more precise picture of thyroid function [9] appear to be reliable in a good proportion of the patients, their usefulness is limited by the inconsistent binding of T4 to TBG in goitrous patients, which renders them unsuitable in a proportion of cases. Alternatively, where the facility is available, free hormone determination by RIA is preferred. Figure 2 and Table 3A demonstrate clearly these errors in classification and diagnosis; Figure 2 shows that in our present cases, 19 hypothyroid patients were wrongly classified as euthyroids and 24 euthyroids wrongly classified as hyperthyroids using serum total T4 as the only basis for the evaluation of the patients.

At the other end of the spectrum, there is a small group with very low level of TBG. Total serum T4 are low, partly because TBG are correspondingly low. This is not surprising as many cases of protein-calorie malnutrition were seen in the area during the present investigation. Triiodothyronine appeared inflated due probably to increased TBG, giving a misleading picture of T3-thyrotoxicosis in otherwise clinically normal subjects. Free serum T3 could not be estimated due to limited equipment. The progressive rise in T3/T4 ratio suggests that the sustained elevated levels of serum TBG provides a compensatory device in endemic environment to maintain thyroid hormone and thyroid function at a higher level (Fig. 1). There is some evidence from initial clinical studies in the literature that there is a decline in serum TBG levels and goitre size, but a rise in thyroid hormones in iodine-deficient goitrous patients after iodized oil medication [10]. This may partly explain why over one-third of our euthyroid patients have almost twice the normal serum TBG in the present investigation. Similarly it is likely that the comparatively higher mean serum albumin seen in the present investigation in hypothyroid patients, compared to euthyroid or hyperthyroid groups (see Table 1) may have the same effect. Our investigations further show that serum albumin appears to be higher in subjects residing within the endemic zone, compared to those outside it (Table 4).

Thyroglobulinaemia was profound among our patients. Similar but lower increases in serum thyroglobulin level have been described by previous authors in other endemic regions [11,12] but our investigations show that Tg increases progressively with thyroid enlargement (Fig. 1). Our work confirms the baseline value obtained for euthyroid non-goitrous Nigerians [13]. The exact cause of hyperthyroglobulinaemia in endemic goitre is still not clear, but inadequate maturation of thyroglobulin due to iodine lack has been blamed [14]. All endemic goitre patients investigated by us (with a single exception) had very high levels of immunoglobulin (Ig), which was nearly double the amount found in healthy subjects (Table 1 and Fig. 3). The immunoglobulin of the IgG class dominated the picture, followed by IgM. Autoimmune thyroid disease is unlikely, and virtually all the tests done to detect thyroglobulin and microsomal antibodies were negative, except in three cases. Drexhage, Doniach and Batazzo [15] provided convincing evidence through nucleic acid cytophotometry and thymidine autoradiography for the presence of thyroid growth immunoglobulin (TGI) in 10 patients with simple colloid goitre. The biological effect of this growth-stimulating Ig is said to be thyroid specific and provokes thyroid cellular growth by binding to TSH receptor sites. The very high Ig seen in our patients with advanced goitre (grades 2–4) in the presence of generalized inappropriately low levels of TSH, perhaps due to pituitary unresponsiveness or destruction of source Ig found in our patients, may have the same effect. Further research is required to explore the significance of this finding in our patients. But the diagnostic implications of an

**Table 1:** Aberrations in serum proteins and indices of thyroid function in African patients with endemic goitre in the present investigation

Serum hormone levels (nmol/l):
T4 = 109(43–276): Ref. values = 91(58–125)
T3 = 2.3(1.08–6.9) " " = 1.7(1.0–2.38)

| | Endemic Goitre | | Normal Subjects | | *t* | *P* |
|---|---|---|---|---|---|---|
| Serum Protein Constituents: | Mean | Range | Mean | Range | | |
| Thyroxine binding globulin (TBG) nmol/l | 306.3 | 107–734 | 271.3 | 173–369 | 8.74 | 0.005 |
| Thyroxine binding capacity (TBC) | 1.30 | 0.54–3.50 | 1.2 | 0.83–1.55 | 7.5 | 0.005 |
| Free Thyroxine Index ($FT_4I$) | 83.7 | 36–174 | 94.0 | 48.0–143.0 | 2.07 | 0.05 |
| $T_4$/TBG ratio | 3.7 | 0.43–11.5 | 4.5 | 3.4–7.6 | 0.75 | 0.05 |
| Immunoglobulins g/l: | | | | | | |
| IgG | 27.37 | 17.0–42 | 15.54 | 9.0–22.0 | 6.72 | 0.005 |
| IgA | 2.40 | 0.5–4.5 | 1.70 | 0.8–3.6 | 2.08 | 0.05 |
| IgM | 2.50 | 1.2–4.5 | 1.50 | 0.8–2.5 | 4.6 | 0.005 |
| Thyroglobulin (Tg) ng/ml | 436.0 | 44–100 | 13.0 | 0–43 | 12.39 | 0.005 |
| Tg — Antibody (Tg-Ab) % | 5.9 | 3.49–8.29 | 10.0 | 7.36–12.64 | 16.4 | 0.005 |
| Microsomal Antibody (Micros-Ab) | positive in 3 patients only | | — | negative | — | — |
| Total Serum Protein g/l | 82.5 | 68.1–96.0 | 76.0 | 58.4–89.0 | 3.2 | 0.005 |
| Albumin g/l: | | | | | | |
| Hypothyroids | 42.0 | 31.6–56.2 | | | | |
| Euthyroids | 37.2 | 25.6–48.8 | | | | |
| Hyperthyroids | 38.7 | 25.3–52.1 | | | | |
| Beta-Liproproteins g/l: (total) | | | | | | |
| Hypothyroids | 6.55 | 4.61–8.49 | | | | |
| Euthyroids | 2.94 | 2.53–3.35 | | | | |
| Hyperthyroids | 2.10 | 1.84–2.36 | | | | |

**Table 2:** Aberrations and frequency distribution of thyroxine binding globulin in African patients with endemic goitre in the present investigation

| Thyroxine Binding Globulin | | | | Normal Subjects | | Endemic Goitre | |
|---|---|---|---|---|---|---|---|
| Mean nmol/l(μg/ml) | | Range | | Number of Subjects | % Frequency | Number of Subjects | % Frequency |
| 96.3 | (5.5) | 35–155.8 | (2–8.9) | 4 | 12 | — | — |
| 218.8 | (12.5) | 157.5–278.3 | (9–15.9) | 6 | 18 | 3 | 2 |
| 341.3 | (19.5) | 280–400.8 | (16–22.9) | 20 | 61 | 45 | 31 |
| 463.8 | (26.5) | 402.5–523.3 | (23–29.9) | 3 | 9 | 54 | 37 |
| 586.3 | (33.5) | 525–645.8 | (30–36.9) | — | — | 26 | 19 |
| 708.8 | (40.5) | 647.5–768.3 | (37–43.9) | — | — | 11 | 8 |
| 831.3 | (47.5) | 770–890.8 | (44–50.9) | — | — | 3 | 2 |
| 945 | (54.0) | 892.5–995.8 | (51–56.9) | — | — | 1 | 1 |
| | | | | 33 | 100 | 143 | 100 |
| Reference Mean and Range | | | | | | | |
| 318 | (18.2) | 219–464 | (12.5–26.5) | | | | |

**Table 3a:** Variations and impact of serum protein parameters, on thyroid hormone levels, thyroid hormone indices and the clinical state of the patient

| Group Name | Serum Protein Values TBG nmol/l(μg/ml) | TBC | Hormone Levels T4 nmol/l(μg/dl) | [$FT_4I$] | T4/TBG ratio | T3 nmol/l | Goitre Grade | | Biochemical and Clinical Features |
|---|---|---|---|---|---|---|---|---|---|
| A.A–M | 262(150) | 1.25 | 111(8.6) | 89(6.8) | 4.1 | 2.14 | 4 | (i) | Normal euthyroid pattern |
| A.E–F | 262(15.0) | 1.40 | 1098(8.5) | 78(6.0) | 4.0 | 1.70 | 4 | (ii) | Note normal TBG and TBC |
| E.Y–F | 262(15.0) | 1.25 | 90(7.0) | 72(5.6) | 3.4 | 2.57 | 2 | (iii) | Patient is biochemically and clinically normal |
| T.A–F | 280(16.0) | 1.25 | 109(8.5) | 89(6.8) | 3.8 | 2.06 | 2 | | |
| A.W–F | 665(38.0) | 2.15 | 203(15.8) | 94(7.3) | 2.9 | 1.99 | 4 | (i) | Very high TBG |
| A.A–F | 595(34.0) | 1.95 | 185(14.4) | 95(7.4) | 3.0 | 2.28 | 2 | (ii) | Very high serum total T4 |
| A.A–F | 359(20.5) | 1.65 | 180(14.0) | 110(8.5) | 4.9 | 1.76 | 2 | (iii) | Moderate to high TBC |
| A.A–F | 525(30.0) | 1.90 | 170(13.2) | 89(6.9) | 3.2 | 1.48 | 4 | (iv) | Normal $FT_4I$ and T4/TBG ratio |
| | | | | | | | | (v) | Patient is clinically normal but biochemically abnormal |
| *A.E–F | 175(10.0) | 1.10 | 193(45.0) | 175(13.6) | 10.8 | — | 3 | (i) | Low to low normal TBG |
| B.W–F | 193(11.0) | 0.60 | 172(13.4) | 287(22.3) | 8.8 | 6.4 | 1b | (ii) | Very high serum T4, $FT_4I$ and T4/TBG ratio |
| J.M–F | 280(16.0) | 0.60 | 139(10.8) | 315(18.0) | 4.9 | 1.84 | 3 | (iii) | Patient is clinically and biochemically thyrotoxic |
| *M.E–F | 227(13.0) | 0.20 | 154(12.0) | 772(60.0) | 6.6 | 2.03 | 2 | | *(A.E. and M.E. are classic cases of Graves disease. Note the striking difference in serum TBG and TBC) between groups B and C |
| L.A–F | 131(7.5) | 1.65 | 54(4.2) | 32(2.5) | 4.0 | 4.52 | 2 | (i) | Very low serum total T4 |
| T.K–F | 149(8.5) | 1.25 | 33(2.6) | 27(2.1) | 3.0 | 1.25 | 3 | (ii) | Low, normal or high TBG |
| Y.A–M | 210(12.0) | 1.65 | 44(3.4) | 27(2.1) | 2.8 | 1.59 | 4 | (iii) | High TBC |
| D.R–F | 420(24.0) | 1.44 | 6.4(0.05) | 15(1.2) | 2.0 | 1.70 | 4 | (iv) | Low $FT_4I$ and T4/TBG ratio low to normal |
| | | | | | | | | (v) | Patient is clinically and biochemically hypothyroid |
| S.M–F | 149(8.5) | 1.25 | 62(4.8) | 49(3.8) | 4.0 | 6.0 | 4 | (i) | Increased T3 the dominant biochemical feature |
| V.E–F | 166(9.5) | 3.5 | 113(8.8) | 32(2.5) | 6.6 | 4.6 | 2 | (ii) | TBG may be high, normal or low |
| A.L– | 166(9.5) | 2.55 | 54(4.2) | 22(1.7) | 3.2 | 6.0 | — | (iii) | Increased TBG may stimulate hyperthyroid T3 value (T3 – toxicosis?) |
| T.Z–F | 245(14.0) | 3.10 | 32(2.5) | 32(2.5) | 3.9 | 7.37 | 4 | (iv) | Patient may be thyrotoxic or normal |
| Reference Values: | | | | | | | | | |
| Mean | 274(15.7) | 1.2 | 91.4(7.1) | 94(7.3) | 4.0 | 1.7 | | | |
| Range: | 175–368 (10.0–21) | 0.83–1.55 | 58–125 (4.5–9.7) | 48–143 (3.6–10.1) | 3.4–7.6 | 1.0–2.38 | | | |

**Table 3b:** Changes that occur in the thyroid hormone levels and thyroid hormone indices and various grades of goitre

| Goitre grades | No. of Samples | XTBG | S.D | XTBC | S.D | XTT4 | S.D | XFT4I | S.D | XT4/TBG | S.D |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 0 | 83 | 16 | 2.85 | 1.13 | 0.43 | 7.2 | 1.31 | 7.4 | 1.86 | 0.45 | 0.18 |
| 1(a 2b) | 50 | 27 | 6.37 | 1.35 | 0.69 | 7.0 | 1.67 | 8.9 | 2.1 | 0.26 | 0.05 |
| 2 | 40 | 28.4 | 6.77 | 1.58 | 0.67 | 6.9 | 2.14 | 5.3 | 2.19 | 0.24 | 0.05 |
| 3 | 68 | 29 | 8.60 | 1.03 | 0.39 | 6.5 | 1.87 | 5.6 | 1.78 | 0.22 | 0.03 |
| 4 | 50 | 31.8 | 10.00 | 1.67 | 0.62 | 6.1 | 3.15 | 4.7 | 1.91 | 0.20 | 0.09 |

**Table 4:** A comparison of the mean and range values for total protein, albumin and globulin among the residents of endemic and non-endemic goitrous zones

| | No. of Subjects | Mean Values (g/l) | | | Range Values (g/l) | | |
|---|---|---|---|---|---|---|---|
| | | Total Protein | Albumin | Globulin | Total Protein | Albumin | Globulin |
| Endemic Goitre (Plateau State, N. Nigeria) — present investigation | (50) | 82.5 | 42.5 | 40.0 | 68 – 97 | 31.6 – 53.6 | 22.1 – 57.7 |
| Normal Subjects Plateau State, N. Nigeria (endemic zone) — present investigation | (50) | 76.0 | 44.5 | 31.5 | 58.4 – 89.0 | 25.5 – 58.0 | 22.0 – 40.4 |
| Normal Subjects (Isichei, 1975), Zaria, N. Nigeria (non-endemic zone) | (205) | 78.0 | 38.0 | 42.0 | 72 – 89 | 32 – 44 | 40 – 45 |
| Normal Subjects (Edozien, Ibadan, S. Nigeria 1959) (non-endemic zone) | | 69.5 | 35.0 | 34.5 | 58 – 88 | 24 – 45 | 25 – 45 |

unexplained massive rise in Ig levels in our cases, however, lie in the fact that, apart from TBG, albumin and pre-albumin, other proteins such as alpha-1, beta-lipoprotein [16] as well as antibodies [17] also have the property to bind thyroid hormones, though in reduced amounts. The appearance of very high Igs in the serum of our patients may therefore result in changes of thyroid function parameters, which do not mirror the thyroid status of the patient. This probably explains why no close correlation was found between thyroxine binding globulin and thyroxine binding capacity in our patents. Beside it is possible that antibodies measured (antithyroglobulin and antimicrosomal) were not the appropriate antibodies causing this pathology.

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