# ASSOCIATION OF THYROID DYSFUNCTION WITH YOUNG DIABETIC SUBJECTS IN A BANGLADESHI POPULATION

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**Abstract**

The study was a prospective study carried out in the Research Division of Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM), Dhaka. The present study was designed to investigate the thyroid dysfunction specially total and free levels of serum $T_3$ and $T_4$, and TSH in young diabetic subjects. A total number of 49 diabetic subjects and 28 controls were recruited in this study. Plasma glucose was measured by glucose oxidase method, $HbA_{1C}$ by modified HPLC method, serum total $T_3$, total $T_4$, free $T_3$, free $T_4$, TSH and C-peptide were measured by fluorescence-based ELISA technique. Age and BMI of the controls, Type 2 diabetic and FCPD subjects were matched. Total $T_3$ concentration in FCPD and type 2 groups were significantly lower than the control group ($p<0.001$). $T_3$ in FCPD was lower than that of type 2 ($p<0.01$). No significant difference was shown in $T_4$ concentrations among the three groups of study subjects but there was significant lower in $T_3$/ $T_4$ ratio in FCPD and type 2 than control groups ($p<0.001$, $p<0.01$ respectively). The concentration of Free $T_3$ was lower in FCPD group ($p<0.01$) than controls and type 2 DM. Free $T_4$ and TSH concentration were similar in all the groups. With increasing of fasting serum glucose and $HbA_{1C}$ in the subjects, serum $T_3$, Free $T_3$, and $T_3$/ $T_4$ ratio were decreased significantly. Irrespective of groups, fasting serum glucose and $HbA_{1C}$ showed negative correlation with serum $T_3$ (FSG: r= -0.591, p= 0.001; $HbA_{1C}$: r= -0.68, p= 0.001) and Free $T_3$ (FSG: r= -0.421, p= 0.001; $HbA_{1C}$: r= -0.381, p= 0.001). C-peptide showed positive correlation with $T_3$ and Free $T_3$ ($T_3$: r= 0.429, p= 0.001; Free $T_3$: r= 0.228, p= 0.05). The existance of low $T_3$ syndrome is confirmed in young Bangladeshi diabetic population regarding free levels of $T_3$ and $T_4$. The values of the free hormones (low $FT_3$ and normal $FT_4$) as well as normal TSH expain clinically euthyroid state of the subjects. The data also demonstrated that the lowering of $T_3$ in diabetic subjects seems to be related with their degree of hyperglycemia.

**Key Words:** *Thyroid Dysfunction, Young Diabetes Mellitus*.

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**Introduction**

Both clinical and animal studies have demonstrated that diabetes mellitus is commonly associated with altered thyroid function. In both Type 1 and Type 2 diabetes there is significant decrease in $T_3$ and increase serum $rT_3$. Serum $T_4$ is normal and and the basal serum TSH is not usually altered or slightly elevated. Both the groups are euthyroid on clinical evaluation[1-2]. In poorly controlled diabetic patients serum $T_3$ is significantly lowered than those of better controlled ones. A signifcantly negtive correlation is found between fasting blood glucose and serum $T_3$ levels[2-4]. There is also an inverse correlation between fasting blood glucose and $T_3/T_4$, which suggests a relationship between impairment of $T_3$ production and severity of reduced glucose uptake and utilization[5-7]. However other factors like body weight, age and duration of diabetic symptoms have failed to demonstrate any relationship with any of the thyroid function tests[1,3].

The low serum $T_4$ concentration is usually found in severely ill patients. The more critical the clinical condition, the greater is the decrease in serum $T_3$ and $T_4$. Some authors have found a direct correlation between lowering of serum $T_4$ and mortality rate[8]. Almost all observers now agree that patients suffering from nonthyroid illness (NTI), having decrease serum $T_3$ and $T_4$, appear euthyroid on clinical evaluation and this conclusion is supported by the usual findng of normal serum TSH in most of the patients[9,10]. Although TSH regulation may not be entirely normal in patients with NTI, it is likely that seum TSH will be increased in most patients who also have significant thyroid failure[11]. In a study in children with Type 1 DM a significantly higher TSH value has also been obsered along with low serum $T_3$ and $T_4$ levels. However it has been proposed that low serum $T_3$ and $T_4$ and a slighly elevated TSH levels are not reliable indicators of primary hypothyroidism in uncontrolled Type 1 DM[12]. Type 1 diabetes is associated with some other autoimmune diseasee and subclinical hypothyroidism has been reported in pregnant women[13]. 7.3 % of children with Type 1 diabetes have been reported to have thyroid dysfunctions and about 19% Type 1 diabetes patients had antichromosomal antibodies. The incidence of hypothyroidism in diabetes mellitus varies from 0.2 to 1.7%. The rate of prevalence was considerably higher in type 1 diabetes than that of adolescent control who did not have typical diabetes [14,15]. In another study of thyroid function and prevalence of thyroid autoantibody in an African diabetic popultion it was found that thyroid hormone levels were significantly lower in Type 1 patients than in control and Type 2 population; subclinical hypothyroidism was present in 21% of the 28 Type 1 diabetic patients [16]. Thyroid function, autoimmunity and morphology were studied by some investigators in Type 1 diabetic patients where they found that high proportion of Type 1 patients without any clinical signs of thyroid disease have markers of thyroid autoanibodies[17].

A substantial number of young diabetic patients in Bangladesh do not show typical characteristics of either Type 1 or Type 2 diabetes mellitus. The traditional insulin dependent ketosis prone childhood diabetes is rarely seen in Bangladesh. However, patients within age 10-30 years are not uncommon in this population. About 50% of patients under 30 years of age attending BIRDEM Out-Patient Department present with atypical diabetes and among them about one-fourth present with pancreatic calcification known as Fibrocalculus Pancretic Diabetes (FCPD)[18]. Total $T_3$ and $T_4$ may be varied with the change in the thyroid binding proteins (TBP) or presence of drugs that modify the total $T_3$ and $T_4$ but not the amount of free hormone. If the TBP is normal in the subject then these are reliable indices of thyroid function. Free $T_3$ and $T_4$ are not bound to TBP and these are the most important parameters to elucidate thyroid dysfunction in the groups, which were not included in the previous study. In the above context the present study was designed to investigate the thyroid dysfunction in the young Bangladeshi diabetic subjects by measuring the total and free levels of $T_3$ and $T_4$ and also TSH in their serum.

**Subjects and Methods**

Forty nine (25 male and 24 female) diabetic subjects (32 Type 2 and 17 FCPD), 18-30 years of age were recruited from the out patient department (OPD) of BIRDEM hospital. Prior to recruitment, diabetes mellitus was confirmed according to WHO criteria[19]. Subjects having no clinical thyroid diseases or other evident systemic diseases and who were taking drugs that influenced the thyroid hormone concentrations in the serum were excluded from this study. Control subjects (n=28, 15 male and 13 female) were selected from the healthy students of Dhaka University within 5 years of age band without family history of diabetes or thyroid disease. Informed written consent was taken from all recruited diabetic and control subjects for the purpose of the study.

Subjects were requested to fast overnight for at least eight hours and in the subsequent morning 10 ml of venous blood was drawn from the antecubital vein. One ml of collected venous blood was taken in an anticoagulant-containing vial for estimation of HbA1_c, rest of the blood was centrifuged for 15 minutes at a rate of 3000 rpm. Then the serum sample was preserved immediately at -40° C for future analysis.

Detailed socio-economic and clinical data were recorded in a pre-designed case record form. Anthropometric indices of all the controls and diabetic subjects were measured according to standard technique.

Plasma glucose was measured by glucose oxidase method, $HbA_{1C}$ by modified HPLC method (Bio-Rad variant), Serum total $T_3$, total $T_4$, free $T_3$, free $T_4$ and TSH were measured by fluorescence-based ELISA technique. Serum C-peptide was measured by ELISA method.

**Statistical analysis**

All the data were expressed as mean ±SD and median (range) as appropriate. Statistical analysis was done by using SPSS 10.0 packages for windows. Appropriate statistical test of significance like unpaired t test, one way analysis of variance (ANOVA) and Mann-Whitney test was used as necessary. $P < 0.05$ was taken as minimum level of significance.

**Results**

Age and BMI of the controls (23±3, 19.7±3.2) Type 2 diabetic (23±4, 18.7±3.4) and FCPD subjects (23±3, 18.2±3.0) were matched. The mean ± SD, fasting serum glucose (in mmol/l: 12.10 ± 7.41 in Type 2, 14.19± 6.72 in FCPD and 4.75± 0.68 in control. The mean SD, $HbA_{1C}$ (%): 11.33 3.78 in Type 2, 11.58 ± 2.46 in FCPD and 5.51±0.39 in control. Fasting serum glucose and $HbA_{1C}$ were significantly higher ($p<0.001$) in FCPD and Type 2 diabetic subjects than control. No difference between was found between FCPD and type 2 diabetes. Total $T_3$ concentration in FCPD and type 2 groups were significantly lower than the control group ($p<0.001$). $T_3$ in FCPD was lower than that of type 2 ($p<0.01$). No significant difference was shown in $T_4$ concentrations among the three groups of study subjects but there was significant lower in $T_3/T_4$ ratio among FCPD and type 2 than control groups ($p<0.01$, $p<0.001$ respectively. The concentration of Free $T_3$ were lower in FCPD group ($p<0.01$) than control group and type 2 DM. No difference between control and type 2 groups was observed. No significant difference was found in Free $T_4$ in all the groups of study subjects. TSH concentration was also similar in all the groups (Table-I).

**Table-I:** *Glycemic and serum $T_3$, $T_4$, TSH and $T_3/T_4$ ratio of the study subjects*

| Variables | Control (n= 28) | Type 2 (n= 32) | FCPD (n= 17) |
|---|---|---|---|
| Fasting Glucose(mmol/l) | 4.75± 0.68 | 12.10± 7.41$^{a***}$ | 14.19± 6.72$^{a***}$ |
| $HbA_{1C}$ (%) | 5.51±0.39 | 11.33 ±3.78$^{a***}$ | 11.58 ± 2.46$^{a***}$ |
| $T_3$ (nmol/l) | 1.97 ± 0.21 | 1.18 ± 0.65a** | 0.90 ± 0.27a***, b** |
| $T_4$ (pmol/l) | 86.0 (56.9-108.7) | 83.6 (24.7-143.2) | 76.2 (49.3-128.4) |
| $FT_3$ (pmol/l) | 5.48 ± 0.40 | 5.10 ± 1.16 | 4.61 ± 1.17a** |
| $FT_4$ (pmol/l) | 14.23 ± 1.72 | 13.61 ± 3.10 | 14.82 ± 3.59 |
| TSH (mIU/ml) | 1.8 (0.70-5.30) | 1.62 (0.63-7.4) | 2.08 (0.01-5.6) |
| $T_3/T_4$ | 0.02 ± 0.004 | 0.01 | ± 0.01a** 0.012 ± 0.01a***, b** |

Results are expressed as Mean ± SD and Median (range). ANOVA (Bonferroni) for parametric and Mann-whitney U tests for nonparametric data were performed as the tests of significance. *p<0.05, **p<0.01, ***p<0.001. [a]vs Control, [b] vs Type 2, [c]vs FCPD.

With increasing of fasting serum glucose in the subjects, serum $T_3$, Free $T_3$, and $T_3/T_4$ ratio were decreased significantly [$T_3$ (nmol/l): (mean ± SD); 1.60 ± 0.49 for the range of FSG <4.35, 1.70 ± 0.53 (FSG 4.36-17.0), 1.10 ± 0.53 (FSG 17.1-25.0), 0.61 ± 0.19 (FSG<25.1). $T_3/T_4$ ratio: 0.02 ± 0.001 (FSG <4.35), 0.02 ± 0.01 (FSG 4.36-17.0), 0.02 ± 0.01 (FSG 17.1-25.0), 0.01 ± 0.001 (FSG<25.1). Free $T_3$ (nmol/l): (mean ± SD); 5.53 ± 0.53 (FSG <4.35), 5.41 ± 0.88 (FSG 4.36-17.0), 4.86 ± 0.83 (FSG 17.1-25.0) and 4.24 ± 1.28 (FSG>25.1)] (Table-II).

With increasing of $HbA_{1C}$ in the subjects, serum $T_3$, Free $T_3$, and $T_3/T_4$ ratio were decreased significantly [$T_3$ (nmol/l): (mean ± SD); 1.97 ± 0.20 for the range of $HbA_{1C}$<6.0%, 1.20 ± 0.58 ($HbA_{1C}$ 6.1-11%), p<0.001; 0.95 ± 0.49 ($HbA_{1C}$ 11.1-16%), p<0.001; 0.84 ± 0.54 ($HbA_{1C}$<16.1%), P<0.001; $T_3/T_4$: 0.02 ± 0.001 ($HbA_{1C}$<6.0%), 0.02 ± 0.01 ($HbA_{1C}$ 6.1-11%), p<0.001; 0.01 ± 0.01 ($HbA_{1C}$ 11.1-16%), p<0.001; 0.01 ± 0.01 ($HbA_{1C}$<16.1%), P<0.001; Free $T_3$ (pmol/l): 5.46 ± 0.41 ($HbA_{1C}$<6%), 5.21 ± 1.0 ($HbA_{1C}$ 6.1-11%), 4.72 ± 1.42 ($HbA_{1C}$ 11.1-16%) and 4.11 ± 0.58 ($HbA_{1C}$<16.1%), P<0.05] (Table-III).

Irrespective of groups, fasting serum glucose and $HbA_{1C}$ showed negative correlation with serum $T_3$ (FSG: -0.591, p= 0.001; $HbA_{1C}$: r= -0.68, p= 0.001) and Free $T_3$ (FSG: r= -0.421, p= 0.001; $HbA_{1C}$: r= -0.381, p= 0.001) (Table-IV).

**Table-II:** *Serum $T_3$, $T_4$, TSH and $T_3/T_4$ ratio in the study subjects based on the different ranges of fasting serum glucose*

| Group | $T_3$(nmol/l) | $T_4$(pmol/l) | $FT_3$(pmol/l) | $FT_4$(pmol/l) | TSH(mIU/ml) | $T_3/T_4$ |
|---|---|---|---|---|---|---|
| FSG<4.3(n= 5) | 1.6 ± 0.9 | 82.3± 18.6 | 5.5 ± 0.5 | 15.0 ± 2.2 | 1.8 ± 0.7 | 0.02 ± 0.004 |
| FSG<4.3-17.0(n= 44) | 1.7 ± 0.5 | 86.5 ± 17.8 | 5.4 ± 0.9 | 13.9 ± 1.9 | 2.0 ± 1.3 | 0.02 ± 0.01 |
| FSG<17.1-25.0(n= 17) | 1.1 ± 0.5b*** | 72.5 ± 20.5 | 4.8± 0.9 | 13.6 ± 2.0b*** | 2.1 ± 1.8 | 0.02 ± 0.01 |
| FSG>25.1(n= 11) | 0.6 ± 0.2a**, b*** | 91.8 ± 24.9 | 4.2± 1.2b** | 15.1± 5.7a**, b*** | 1.8± 1.1 | 0.01 ± 0.001a**,b***, c** |

Results are expressed as Mean ± SD. ANOVA (Bonferroni) was performed as the tests of significance. *p<0.05, **p<0.01, ***p<0.001. [a]vs FSG<4.35, [b]vsFSG4.36-17.0, [c]vsFSG17.0-25.0, [d]vsFSG>25.1

**Table-III:** *Serum $T_3$, $T_4$, TSH and $T_3/T_4$ ratio in the study subjects based on the different ranges of $HbA_{1C}$*

| Group | $T_3$(nmol/l) | $T_4$(pmol/l) | $FT_3$(pmol/l) | $FT_4$(pmol/l) | TSH(mIU/ml) | $T_3/T_4$ |
|---|---|---|---|---|---|---|
| $HbA_{1C}$ <6% (n= 29) | 1.9 ± 0.2 | 85.1± 13.6 | 5.4 ± 0.4 | 14.2 ± 1.7 | 1.8 ± 1.1 | 0.02 ± 0.001 |
| $HbA_{1C}$ <6.1-11% (n= 25) | 1.2 ± 0.5a*** | 85.9 ± 20.9 | 5.2 ± 1.0 | 14.4 ± 2.8 | 2.1 ± 1.3 | 0.02 ± 0.01a*** |
| $HbA_{1C}$ <11.1-16% (n= 18) | 0.9 ± 0.5a*** | 80.4 ± 26.5 | 4.7± 1.4 | 13.3 ± 4.1 | 2.5 ± 1.8 | 0.01 ± 0.01a*** |
| $HbA_{1C}$ >16% (n= 5) | 0.8 ± 0.5a*** | 79.3 ± 27.4 | 4.1± 0.5a* | 14.3± 2.2 | 1.5± 0.8 | 0.01± 0.01a*** |

Results are expressed as Mean ± SD. ANOVA (Bonferroni) was performed as the tests of significance. *p<0.05, **p<0.01, ***p<0.001. [a]vs $HbA_{1C}$ <6%, [b]vs $HbA_{1C}$ 6.1-11%, [c]vs $HbA_{1C}$ 11.1-16%, [d]vs $HbA_{1C}$ >16.1%.

**Table-IV:** *Correlation of serum $T_3$, $T_4$, TSH, $FT_3$ and $FT_4$ with Fasting serum glucose, $HbA_{1C}$ and C-peptide in the study subjects*

| Group | $T_3$ | | $T_4$ | | TSH | | $FT_3$ | | $FT_4$ | |
|---|---|---|---|---|---|---|---|---|---|---|
| | r | p | r | p | r | p | r | p | r | p |
| FSG | -0.59 | 0.001** | -0.009 | 0.93 | -0.014 | 0.90 | -0.421 | 0.001** | 0.054 | 0.63 |
| $HbA_{1C}$ | -0.68 | 0.001** | -0.100 | 0.38 | 0.117 | 0.31 | -0.38 | 0.001** | -0.075 | 0.517 |

**Discussion**

It is found that diabetes mellitus may be associated with altered thyroid functions. But this phenomenon has been observed mainly in Type 1 and Type 2 diabetic patients. In Bangladesh a substantial number of young diabetic patients do not show typical characteristics of either Type 1 or Type 2 diabetes mellitus. The main peculiarities of this group include low BMI and marked hyperglycemia without ketosis even in the presence of only residual insulin (fasting C-peptide below 1 ng/m1)[18]. In this study, free $T_3$ and $T_4$ of the age and BMI matched control subjects were 5.48± 0.40 and 14.23±1.72 pmol/l (mean ± SD). The reference ranges of free $T_3$ and $T_4$ values in adults are 3-8 and 12-24 pmo/1[20]. Free $T_3$ and $T_4$ values in the control subjects are within the range of the reference value.

In this study Bangladeshi type 2 diabetic and FCPD patients have shown significantly low $T_3$ level. The same result has been shown in the earlier two studies. Total $T_4$ and TSH did not differ significantly in both the groups compared to control subjects in the present study. In the previous two studies TSH did not differ but $T_4$ was marginally lowered in the study of Mollah (1996) [21]. Free $T_3$ was low and free $T_4$ did not differ compared to control subjects in the present study, which confirm that $T_3$ is truly low.

The present studies confirm the groups of young diabetic subjects are suffering from low $T_3$ syndrome. The clinical hypothyroidism seems to be absent in this group as TSH as well as $FT_4$, were normal. Low serum $T_4$ concntration is usually found in severely ill patients. Some authors have found direct correlation between lowering of $T_4$ and mortality rate[8]. Although the level of fasting blood glucose is considerably high in diabetic groups, particularly in FCPD, it seems that even at this degree of hyperglycemia a clinically euthyroid state is maintained. To explore the associaton of lowered $T_3$ with the progression of diabetes, correlation of thyroid hormones were done with serum glucose and $HbA_{1C}$. A highly singnificant negative correlation of $T_3$ with glucose and $HbA_{1C}$ were found. The data confirmed that hyperglycemia is a major factor in the development of low $T_3$ syndrome.

The following conclusion can be made from the present study: The existance of low $T_3$ syndrome is confirmed in young Bangladeshi diabetic population by the measurement of free levels of $T_3$ and $T_4$. The values of the free hormones (low $FT_3$ and normal $FT_4$) as well as normal TSH expain clinically euthyroid state of the subjects. The lowering of $T_3$ in diabetic subjects seems to be related with their degree of hyperglycemia.

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