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Evaluation of Thyroid Function Tests in Patients with Diabetes Mellitus: A Hospital-Based Study

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ABSTRACT

Thyroid hormones are essential regulators of carbohydrate metabolism, and their imbalance may disproportionately affect individuals with type 2 diabetes mellitus (T2DM). This study aimed to assess the profile of thyroid function among T2DM patients attending a tertiary care hospital in Hazaribagh, with the goal of identifying potential hormonal disturbances that could influence diabetic management. A total of 100 patients diagnosed with T2DM were selected from various clinical departments-Medicine, Obstetrics & Gynecology, and Surgery-at SBMC ,Hazaribagh. Participants were enrolled regardless of age or gender, and informal consent was obtained prior to inclusion. Fasting and postprandial blood glucose levels were determined using the Glucose Oxidase-Peroxidase (GOD-POD) method. Serum levels of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were evaluated using the Enzyme-Linked Immunosorbent Assay (ELISA) technique. Additional data were collected on socio-demographic variables, duration of diabetes, and anthropometric measurements. Waisthip ratio (WHR) was computed, and statistical analyses-including means, standard deviations, and chi-square tests—were performed to compare findings across groups. Of the 100 T2DM patients analyzed, a majority exhibited normal thyroid function. However, approximately one-fifth showed subclinical abnormalities, such as euthyroid sick syndrome, subclinical hyperthyroidism, and subclinical hypothyroidism. The WHR was notably higher among those with diabetes. No participants showed signs of overt thyroid dysfunction. A mild upward trend in TSH levels was observed with longer diabetes duration, though this was not statistically significant. This study highlights the importance of evaluating thyroid function in patients with diabetes, even in the absence of overt symptoms. Identifying subclinical thyroid issues could support better glycemic control and help mitigate the risk of long-term complications.

INTRODUCTION

Diabetes mellitus (DM) is a complex metabolic disorder marked by disturbances in the metabolism of carbohydrates, fats, and proteins. These disruptions primarily arise due to insufficient production or ineffective utilization of insulin-a hormone essential for regulating glucose, lipid, and amino acid metabolism. DM can lead to both acute and chronic complications, often resulting in significant illness and increased mortality risk. According to estimates from the International Diabetes Federation (IDF), approximately 463 million adults were living with diabetes globally in 2019, and this number is expected to rise to 700 million by 2045[1].

The thyroid gland, another critical endocrine organ, also plays a vital role in the regulation of metabolism. Its hormones-triiodothyronine (T3) and thyroxine (T4)-influence carbohydrate metabolism and insulin dynamics. In people with diabetes, particularly those with type 2 diabetes mellitus (T2DM), disturbances in thyroid function are not uncommon. These hormones are intricately linked to the body's metabolic processes, and alterations in one system can directly influence the function of the other[2,3].

T2DM may impact thyroid function by affecting thyroid-stimulating hormone (TSH) secretion from the pituitary, altering the peripheral conversion of T4 to T3, or through the direct effect of hyperinsulinemia on thyroid tissue. In turn, thyroid dysfunction may worsen glycemic control and contribute to complications in diabetic individuals[4]. The association between thyroid disorders and diabetes has been observed since the late 1970s[5], and a number of studies have since reported a higher prevalence of thyroid abnormalities in T2DM patients compared to non-diabetic individuals[6-8].

Assessing thyroid function is essential not only for diagnosing thyroid disorders but also for monitoring treatment outcomes. Standard thyroid profile tests typically include serum measurements of TSH, free or total T3, and free or total T4. The reliability of these assays has improved significantly with the advent of fourth-generation immunoassay technologies[10,11].

Diabetes mellitus, being one of the most common endocrine disorders worldwide, contributes heavily to global health burdens due to its complications. Its rising prevalence-particularly in developing countries-is largely driven by rapid urbanization, lifestyle changes, and economic growth[9]. Chronic hyperglycemia, a hallmark of diabetes, may also negatively influence thyroid function, making coexisting thyroid disease a significant clinical concern in diabetic populations.

Although autoimmune thyroid disorders are more frequently seen in type 1 diabetes, type 2 diabetes patients are not immune to thyroid dysfunction. Given the systemic nature of diabetes and its effect on multiple organ systems, including the thyroid gland, evaluating thyroid function in T2DM patients becomes clinically relevant.

Despite extensive literature on this subject, there is limited data from our region regarding thyroid dysfunction in diabetics. Hence, the present study was undertaken at Shaheed Nirmal Mahto Medical College (SBMC), Hazaribag, as a hospital-based, prospective, cross-sectional analysis, aiming to identify the spectrum of thyroid function profiles among patients with T2DM attending diabetic outpatient services at our institution. The study employed GOD-POD method for blood glucose estimation and ELISA for measuring serum T3, T4, and TSH levels, thus ensuring reliable diagnostic output using standard biochemical methods.

MATERIALS AND METHODS

This hospital-based, descriptive cross-sectional study was conducted at Sheikh Bhikari Medical College (SBMC), Hazaribag, a tertiary care teaching hospital located in Jharkhand, India.

Ethical Considerations: Approval was obtained from the Institutional Ethics Committee prior to initiating the study. Informal verbal consent was taken from all participating patients after explaining the purpose and nature of the research.

Study Population and Sampling: A total of 100 patients with diagnosed type 2 diabetes mellitus (T2DM) were selected for this study, regardless of their age or sex. These individuals were recruited from various clinical departments including Medicine, Obstetrics & Gynecology, and Surgery.

Inclusion Criteria:

- Patients with clinically diagnosed T2DM
- Fasting plasma glucose level = 7 mmol/L on their most recent two clinical visits
- No history of thyroid disease or thyroid surgery
- No exposure to neck irradiation
- Provided informed consent to participate

Exclusion Criteria:

- Pregnant women
- Patients already on thyroid-related medications
- Those with diagnosed thyroid disorders
- Individuals with prior thyroid surgery or radiation exposure
- Critically ill or non-consenting individuals

Clinical and Anthropometric Assessment: Each participant underwent a structured interview using a pretested questionnaire to collect demographic and

clinical information such as age, education level, occupation, and diabetes duration.

Physical Measurements Included:

- Body weight (measured using a calibrated digital scale)
- Height (using a portable stadiometer)
- Body Mass Index (BMI) was computed as weight (kg) divided by height squared (m^2)

Underweight: < 18.5 kg/m^2 o Normal: 18.5–24.9 kg/m^2 **Overweight:** 25–29.9 kg/m^2 o Obese: = 30 kg/m^2 • Waist circumference (WC) was measured midway between the xiphisternum and umbilicus

- Hip circumference (HC) was measured at the level of the greater trochanters
- Waist–Hip Ratio (WHR) was calculated as WC divided by HC

Blood pressure (BP) was recorded using a standard mercury sphygmomanometer. A BP reading below 130/80 mmHg was considered normal.

Biochemical Analysis: Blood samples were collected in the morning following overnight fasting, coinciding with the patients' regular outpatient appointments.

- Fasting and postprandial blood glucose levels were measured using the Glucose Oxidase-Peroxidase (GOD-POD) method.
- Thyroid function tests (T3, T4, TSH) were performed using the Enzyme-Linked Immunosorbent Assay (ELISA) technique. The tests were conducted using a second-generation automated immunoanalyzer (CLIA-based ELISA platform).

The laboratory adhered to quality control standards, and all assays were run in accordance with manufacturer instructions. The detection limits were as follows:

- TSH: 0.0062 µIU/mL
- fT3: 0.742 pg/mL
- fT4: 0.28 ng/dL

Serum samples were centrifuged at 3000 rpm for 5 minutes and stored at -20°C until further analysis. Interpretation of Thyroid Function Test (TFT) Results Thyroid hormone profiles were categorized based on standard biochemical criteria[10,11]:

- Euthyroid: Normal levels of TSH, fT3, and fT4
- Primary hyperthyroidism: Elevated fT3 and/or fT4 with suppressed TSH
- Subclinical hyperthyroidism: Normal fT3 and fT4 with low TSH
- Primary hypothyroidism: Low fT3 and/or fT4 with elevated TSH
- Subclinical hypothyroidism: Normal fT3 and fT4 with elevated TSH
- Euthyroid Sick Syndrome (ESS): Normal or low fT3 and/or fT4 in presence of abnormal TSH in the context of non-thyroidal illness

Statistical Analysis: All data were analyzed using Statistical Package for Social Sciences (SPSS) version 20.

- Continuous variables (e.g., age, BMI, WHR) were expressed as mean ± standard deviation.
- Independent t-tests and one-way ANOVA were used to assess mean differences between groups.
- Categorical variables (e.g., gender, education) were analyzed using Chi-square tests, with Yates' correction where necessary. A p-value < 0.05 was considered statistically significant.

RESULTS AND DISCUSSIONS

In this hospital-based cross-sectional study conducted at SBMC, Hazaribagh, a total of 100 patients with type 2 diabetes mellitus (T2DM) were enrolled. Among these, 78 participants completed the full evaluation, comprising 56 diabetic patients and 22 non-diabetic individuals serving as controls. The gender distribution showed a higher proportion of females (51) compared to males (27), with 67.9% of diabetic females and 59.1% of non-diabetic females, respectively, reflecting a trend consistent with gender-based prevalence noted in earlier epidemiological studies[2].

Socio-demographically, 43.6% of the participants were educated up to the tertiary level or higher, while 37.2% were engaged in skilled or professional occupations, and around 70% of participants were married-characteristics similar to those seen in rural and semi urban Indian populations with access to tertiary healthcare[2,9].

Clinically, patients with T2DM were generally older than the control group, although this difference was not statistically significant. The waist-hip ratio (WHR) among diabetics was 0.92 ± 0.05, which was significantly higher than that of the non-diabetics (0.88 ± 0.06), indicating a greater central obesity risk factor among diabetics, consistent with metabolic syndrome profiles[6,9].

Thyroid pattern	T2DM%
Euthyroid	78.6
Euthyroid sicksyndrome	8.9
Subclinical Hyperthyroid	7.1
Subclinical Hyperthyroid	5.4

There was no significant difference in body mass index (BMI) between the two groups. However, systolic and diastolic blood pressure readings were significantly higher among diabetic patients (136.48 ± 21.55 / 78.46 ± 11.6 mmHg) compared to non-diabetic individuals

(116.36 ± 11.77 / 71.36 ± 6.21 mmHg), reinforcing the well-documented link between diabetes and hypertension[2,36].

In terms of thyroid function, 78.6% of the diabetic patients were found to have euthyroid profiles, while 21.4% showed abnormal thyroid function patterns, including:

- Euthyroid Sick Syndrome (ESS): 8.9%
- Subclinical Hyperthyroidism: 7.1%
- Subclinical Hypothyroidism: 5.4%

No patient from the diabetic group exhibited overt hypothyroidism or hyperthyroidism, aligning with similar findings from previous regional studies[14-16]. Among the nondiabetic participants, only 4.5% showed abnormal thyroid profiles, all being subclinical hyperthyroid cases.

In the overall population, 13 participants (both diabetic and non-diabetic) exhibited abnormal thyroid profiles, evenly distributed between males and females. In the diabetic group, there was no statistically significant difference between males and females in the prevalence of thyroid dysfunction.

Furthermore, more than half (53.6%) of the T2DM patients had been diagnosed less than five years ago. A trend was observed where TSH levels increased with the duration of diabetes, while fT3 levels decreased, although neither trend reached statistical significance. This pattern suggests a possible progressive decline in thyroid conversion function with prolonged hyperglycemia exposure, as previously reported[15,16].

In this hospital-based study conducted at SBMC, Hazaribagh, thyroid-stimulating hormone (TSH) levels and thyroid hormone profiles among individuals with type 2 diabetes mellitus (T2DM) showed no statistically significant differences when compared to non-diabetic individuals (NDM). However, TSH levels were generally higher in the T2DM group. This observation aligns with the findings of Udiong *et al*., who reported similar TSH values among diabetic patients in Calabar, Nigeria, and noted that the difference from controls was not significant[12]. A comparable result was also documented by Islam *et al*. in Southeast Asia, where no substantial variation in TSH levels between diabetics and non-diabetics was detected[13].

Thyroid hormones play a crucial role in regulating metabolism and are known to act in opposition to insulin. In diabetes, a decline in the secretion of thyrotropin-releasing hormone (TRH) may contribute to the observed lower levels of circulating thyroid hormones. This is consistent with our finding that thyroid hormone levels tended to be lower in diabetic patients despite being within reference limits[18,30].

In the current study, 21.4% of diabetic patients exhibited thyroid dysfunction. This is somewhat lower than the 29.7% prevalence reported by Ghazali and Abbiyesuku in Ibadan[14], but higher than the 12.4% reported in Enugu by Ogbonna and Ezeani[15]. Our nondiabetic control group had a 4.5% prevalence of thyroid dysfunction, similar to the 2.8% reported among controls in Ibadan[14]. These variations across studies could be attributed to sample size, population differences, or methodology.

Additional studies outside Nigeria have also demonstrated a wide range of thyroid dysfunction prevalence in T2DM patients. For instance, studies in northeast India reported a 30% prevalence among diabetic individuals[16], while similar trends were observed in Greece (12.3%), Saudi Arabia (16%), and Jordan (12.5%)[6-8]. These variations may reflect demographic diversity, sample sizes, and differences in screening criteria.

In our analysis of 100 diabetic patients at SBMC, we included individuals who met glycemic control criteria (fasting glucose = 7 mmol/L), based on both the current and prior clinic visit. The spectrum of thyroid abnormalities identified included euthyroid sick syndrome (8.9%), subclinical hyperthyroidism (7.1%), and subclinical hypothyroidism (5.4%), with no cases of overt thyroid disease. The majority of diabetic patients (78.6%) had normal thyroid function.

Similar thyroid function patterns have been documented in previous studies, where alterations in thyroid hormone levels—though often subclinical-were common among T2DM patients[17-19]. Subclinical hypothyroidism was observed more frequently than subclinical hyperthyroidism in various investigations, reinforcing the importance of routine screening for both conditions in diabetic populations[20-24].

It is worth noting that many of our diabetic patients were on oral hypoglycemic agents, particularly metformin and sulfonylureas. Metformin, in particular, has been associated with reduced TSH levels without corresponding changes in fT3 or fT4 concentrations in both treated and untreated hypothyroid patients. These effects have been documented in several studies[25-27] and may partly explain our observation of higher TSH in the diabetic group despite mean levels remaining within reference range.

The interplay between hyperglycemia and thyroid hormone synthesis, peripheral conversion, and hypothalamic-pituitary axis regulation could also contribute to subtle changes in thyroid function seen in diabetics[28,29]. TSH response to TRH is often diminished in poorly controlled diabetes, and nocturnal TSH peaks are frequently blunted or absent.

In our study, over 50% of the T2DM patients had a disease duration of less than five years, and we did not find a statistically significant association between diabetes duration and thyroid dysfunction. However,

TSH levels showed a mild upward trend with increasing disease duration. While similar findings have been reported in other studies[31,32], Ogbonna and Ezeani[15] suggested that having diabetes for more than five years may increase the risk of developing thyroid dysfunction. This has also been echoed by studies that associate prolonged diabetes duration with higher rates of thyroid abnormalities[33-35].

The average blood pressure recorded in the diabetic group was 136.5 mmHg systolic and 78.5 mmHg diastolic, significantly higher than the 116.4/71.4 mmHg noted in the nondiabetic group. This trend aligns with reports by Zimmet et al. and other studies linking T2DM with increased hypertension risk[36].

From a socio-demographic perspective, our study population had a higher proportion of females among both diabetic and non-diabetic groups-a pattern observed in other similar studies[22,37,38]. This supports evidence suggesting that diabetes and central obesity tend to be more prevalent in females[39,40].

Most diabetic patients in our rural setting were unemployed or unskilled workers, a reflection of the agrarian nature of the community. The presence of a tertiary medical facility may account for the inclusion of some professional or skilled workers in the sample. Educational status was evenly distributed across the groups, with no significant trends, although prior studies have shown that socioeconomic status (SES) significantly affects the risk, progression, and outcomes of diabetes[41-43].

SES is a multidimensional construct encompassing factors such as education, income, occupation, geographic location, debt level, and financial liabilities. Each factor plays a role in shaping disease vulnerability and access to care[44,45]. Although there is ongoing debate about whether diabetes disproportionately affects those with low SES, the influence of economic and educational disparities on diabetes prevalence is well-established[46,47]. A study conducted in Thailand found that low educational attainment and other SES markers were significantly associated with diabetes risk[48].

CONCLUSION

This hospital-based study emphasizes the need for routine thyroid screening among diabetic patients. Though overt thyroid disease was absent, subclinical dysfunction was relatively common, and may influence glycemic regulation. Identifying thyroid abnormalities early can improve metabolic control, reduce cardiovascular risks, and enhance patient outcomes.

REFERENCE

1. International Diabetes Federation. IDF diabetes atlas, 2019. https://www.idf.org/aboutdiabetes/what-is-diabetes/factsfigures.html (accessed 10 September 2020).
2. Ray S, Ghosh S. Thyroid disorders and diabetes mellitus: double trouble. J Diabetes Res Ther 2016; 2: 1–7.
3. Nair A, Jayakumari C, Jabbar PK, et al. Prevalence and associations of hypothyroidism in Indian patients with type 2 diabetes mellitus. J Thyroid Res 2018; 2018: 5386129.
4. Feely J, Isles TE. Screening for thyroid dysfunction in diabetics. Br Med J 1979; 1: 1678.
5. Perros P, McCrimmon RJ, Shaw G, et al. Frequency of thyroid dysfunction in diabetic patients: value of annual screening. Diabet Med 1995; 12(7): 622–627.
6. Papazafiropoulou A, Sotiropoulos A, Kokolaki A, et al. Prevalence of thyroid dysfunction among Greek type 2 diabetic patients attending an outpatient clinic. J Clin Med Res 2010; 2: 75–78.
7. Akbar DH, Ahmed MM, Al-Mughales J. Thyroid dysfunction and thyroid autoimmunity in Saudi type 2 diabetics. Acta Diabetol 2006; 43(1): 14–18.
8. Radaideh AR, Nusier MK, Amari FL, et al. Thyroid dysfunction in patients with type 2 diabetes mellitus in Jordan. Saudi Med J 2004; 25(8): 1046–1050.
9. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. Lancet 2016; 387: 1513–1530.
10. Dayan CM. Interpretation of thyroid function tests. Lancet 2001; 357: 619–624.
11. Carvalho GA, Perez CL, Wards LS. The clinical use of thyroid function tests. Arq Bras Endocrinol Metab 2013; 3: 193–204.
12. Udiong CEJ, Udoh AE, Etukudoh ME. Evaluation of thyroid function in diabetes mellitus in Calabar, Nigeria. Indian J Clin Biochem 2007; 22(2): 74–78.
13. Islam S, Yesmine S, Khan SA, et al. Comparative study of thyroid hormone levels in diabetic and non-diabetic patients. Southeast Asian J Trop Med Public Health 2008; 39(5): 913–916.
14. Ghazali SM, Abbiyesuku FM. Thyroid dysfunction in Nigerian type 2 diabetic patient. Nig J Physiol Sci 2010; 25: 176–179.
15. Ogbonna SU, Ezeani IU. Risk factors of thyroid dysfunction in patients with type 2 diabetes mellitus. Front Endocrinol 2019; 10: 440.
16. Chutia H, Bhattacharyya H, Ruram AA, et al. Evaluation of thyroid function in type 2 diabetes in northeastern part of India: a hospital-based study. J Family Med Prim Care 2018; 7: 752–755.
17. Smithson MJ. Screening for thyroid dysfunction in a community population of diabetic patients. Diabet Med 1998; 15(2): 148–150.
18. Suzuki J, Nanno M, Gemma R, et al. The mechanism of thyroid hormone abnormalities in patients with diabetes mellitus. Nippon Niabunpi Gakki Zasshi 1994; 70(4): 465–470.

19. Celani MF, Bonati ME, Stucci N. Prevalence of abnormal thyrotropin concentrations in patients with type 2 diabetes mellitus. Diabetes Res 1994; 27(1): 15–25.
20. Maxzud MC, Rasjido LG, Calafiore FA, et al. Prevalence of thyroid dysfunction in patients with type 2 diabetes mellitus. Medicina 2016; 76: 355–358.
21. Palma CC, Pavesi M, Nogueira VG, et al. Prevalence of thyroid dysfunction in patients with diabetes mellitus. Diabetol Metab Syndr 2013; 5: 58.
22. Ozair M, Noor S, Raghav A, et al. Prevalence of thyroid disorders in North Indian type 2 diabetic subjects: a cross sectional study. Diabetes Metab Syndr 2018; 12: 301–304.
23. Khatiwada S, Sah SK, Kc R, et al. Thyroid dysfunction in metabolic syndrome patients and its relationship with components of metabolic syndrome. Clin Diabetes Endocrinol 2016; 2: 3.
24. Demitrost L, Ranabir S. Thyroid dysfunction in type 2 diabetes mellitus: a retrospective study. Indian J Endocrinol Metab 2012; 16(Suppl. 2): S334–S335.
25. Vigersky RA, Filmore-Nassar A, Glass AR. Thyrotropin suppression by metformin. J Clin Endocrinol Metab 2006; 91: 225–227.
26. Isidro ML, Penín MA, Nemiña R, et al. Metformin reduces thyrotropin levels in obese, diabetic women with primary hypothyroidism on thyroxine replacement therapy. Endocrine 2007; 32(1): 79–82.
27. Cappelli C, Rotondi M, Pirola I, et al. TSH-lowering effect of metformin in type 2 diabetic patients: differences between euthyroid, untreated hypothyroid, and euthyroid on LT4 therapy patients. Diabetes Care 2009; 32: 1589–1590.
28. Gursoy NT, Tuncel E. Relationship between glycemic control and the hypothalamuspituitary-thyroid axis in diabetic patients. Turk J Endocrinol Metab 1999; 4: 163–168.
29. Granner DK. Thyroid hormones. In: Murray RK, Granner DK, Mayes PA, et al. (eds) Harper's Biochemistry. 25th ed. London: Prentice-Hall International Inc., 2000, pp. 533–538.
30. De Greef WJ, Rondeel JM, Van-Haasteren GA, et al. Regulation of TRH production and release in rats. Acta Med Austria 1992; 19: 77–79.
31. Diez JJ, Sanchez P, Iglesias P. Prevalence of thyroid dysfunction in patients with type 2 diabetes. Exp Clin Endocrinol Diabetes 2011; 119: 201–207.
32. Ravishankar SN, Champakamalini, Venkatesh, et al. A prospective study of thyroid dysfunction in patients with type 2 diabetes in general population. Arch Med 2013; 5: 2.
33. Telwani AA, Wani ZH, Ashraf Y, *et al*. Prevalence of thyroid dysfunction in type 2 diabetes mellitus: a case control study. Int J Res Med Sci 2017; 5: 4527–4531.
34. Sreelatha M, Kumar VS, Shekar GC, et al. Study of thyroid profile in patients with type 2 diabetes mellitus. Int J Sci Stud 2017; 5: 211–220.
35. Al-Lehibi KI, Abdulraham MI, Albassam AE. Thyroid dysfunction in type 2 diabetic patients and the effect of diabetes duration and anti-glycemic medications on mean TSH and A1c levels: a retrospective study. Int J Med Res Health Sci 2019; 8: 117–122.
36. Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. Nature 2001; 414: 782–787.
37. Jali MV, Kambar S, Jali SM, et al. Prevalence of thyroid dysfunction among type 2 diabetes mellitus patients. Diabetes Metab Syndr 2017; 11: S105–S108.
38. Witting V, Bergis D, Sadet D, et al. Thyroid disease in insulin-treated patients with type 2 diabetes: a retrospective study. Thyroid Res 2014; 7: 2.
39. Okosun IS, Forrester TE, Rotimi CN, et al. Abdominal adiposity in six populations of West African descent: prevalence and population attributable fraction of hypertension. Obes Res 1999; 7: 453–462.
40. Scavini M, Stidley CA, Shah VO, et al. Prevalence of diabetes is higher among female than male Zuni Indians. Diabetes Care 2003; 26(1): 55–60.
41. Hu FB. Globalization of diabetes: the role of diet, lifestyle, and genes. Diabetes Care 2011; 34(6): 1249–1257.
42. World Health Organization. Noncommunicable diseases and mental health. UN highlevel meeting on NCD prevention and control, 2011.
43. Secretary's Advisory Committee. Healthy People 2020: Societal Determinants of Health in the U.S., 2010. [44] Hwang J, Shon C. Relationship between socioeconomic status and type 2 diabetes: results from KNHANES 2010–2012. BMJ Open 2014; 4: 005710.
44. Thomas C, Nightingale CM, Donin AS, et al. Socio-economic position and type 2 diabetes risk in UK children. PLoS ONE 2012; 7(3): e32619.
45. Berkowitz SA, Karter AJ, Lyles CR, et al. Low SES is associated with hypoglycemia in diabetes: DISTANCE study. J Health Care Poor Underserved 2014; 25(2): 478–490.
46. Adler NE, Newman K. Socioeconomic disparities in health: pathways and policies. Health Aff 2002; 21(2): 60–76.
47. Suwannaphant K, Laohasiriwong W, Puttanapong N, et al. Association between SES and diabetes mellitus: Thai national socioeconomic survey. J Clin Diagn Res 2017; 11: LC18–