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Research Article

A retrospective look at the impact of smoking on glycemic control and thyroid status in patients with type 1 diabetes (T1DM)

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Abstract

Smoking is one of the factors that can affect serum glucose in patients with type 1 diabetes (T1DM). Control of T1DM is associated with achieving glycated hemoglobin [HbA1c] levels of approximately 7%, which leads to reduced long-term cardiovascular risk. Thyroid hormones can also affect blood sugar levels. Therefore, appropriate control of diabetes can reduce the risk of developing thyroid disorders and vice versa. The aim of the study is to determine whether smoking affects glycemic control and thyroid function in established type 1 diabetes mellitus (T1DM). Materials and methods: A heterogeneous group of 73 patients aged 39±13 years were examined: men 49 (65.3%), women 24 (32%) with T1DM upon admission to the endocrinology department "St. Georgi" (Plovdiv); smokers 25; non-smokers 48. The following were investigated: blood glucose profile (BGP): serum glucose (mmol/L) at 6, 12, 17, 21, 24 hours; HBA1c (%); FT4 pmol/L; TSH mIU/L; TC: total cholesterol mmol/L; HDL-C mmol/L; LDL-C mmol/L; TAG mmol/L; BMI kg/m2 on admission to the ward. Results: In smokers compared to non-smokers, we found statistically significant, $p<0.05$: increased fasting serum glucose at 6 hours (15.59±6 mmol/L vs 9.67±3.3 mmol/L); decreased BMI (23.21±4.62 kg/m2 vs 26.16±4.79 kg/m2); HDL-C (1.05±0.35 mmol/L vs 1.57±0.43 mmol/L); as well as increased TSH (1.84±0.99 mIU/L vs 0.85±0.94 mIU/L). HBA1c at admission to the clinic showed no significant difference in smokers compared to non-smokers (9.15±1.66% vs. 10.07±1.98%, $p=0.574$). Conclusion: Smoking worsens glycemic control and thyroid function in patients with T1DM.

Keywords

established type 1 diabetes mellitus, smoking, thyroid function

Introduction

Smoking is recognized as the second leading risk factor for early death and disability worldwide (Doll et al. 2004). Smoking increases the risk of cardiovascular diseases through oxidative damage and dyslipidemia (Rempher 2006) and is independently associated with the incidence of diabetes mellitus (Hilawe et al. 2015). Smoking is one of the factors that may affect serum glucose in patients with type 1 diabetes mellitus (T1DM). Smokers with diabetes mellitus are more likely to experience difficulties with insulin dosing and disease control than nonsmokers (Śliwińska-Mossoń and Milnerowicz 2014). Control of T1DM is associated with achieving glycated hemoglobin

[HbA1c] levels of approximately 7%, which results in reduced microvascular risk in terms of retinopathy, neuropathy, and nephropathy (Nathan et al. 1993) and reduced long-term cardiovascular risk. HbA1c is one of the standard indices for the diagnosis of diabetes and is recommended for determining glucose control among people who have already been diagnosed with diabetes (Nathan 2009). HbA1c provides an idea of the average blood glucose over the past two to three months and is used to monitor glycemic control in patients with diabetes (Rohlfing et al. 2002). Thyroid hormones are important modulators of cardiovascular function. Both hypothyroidism and hyperthyroidism are known to contribute to increased cardiovascular risk. Thyroid hormones can affect blood glucose levels (Ardekani et al. 2010). Therefore, appropriate control of diabetes can reduce the risk of developing thyroid disorders and vice versa (Bivolarska et al. 2016a, 2016b).

Purpose

Does smoking affect glycemic control and thyroid function in cases of established type 1 diabetes mellitus (T1DM)?

Material and methods

A heterogeneous group of 73 patients aged 39±13—men 49 (65.3%), women 24 (32%)—with T1DM were studied upon their admission to the endocrinological ward at "St. Georgi" Hospital in Plovdiv: 25 smokers and 48 non-smokers. The following parameters were tested: Blood sugar profile: serum glucose (mmol/L) at 6, 12, 17, 21, 24 hours; HBA1c (%); FT4 pmol/L; TSH mIU/L; TC: total cholesterol mmol/L; HDL-C mmol/L; LDL-C mmol/L; TAG mmol/L; BMI kg/m^2 on admission to the ward. Serum glucose and HbA1c were measured by hexokinase and immunoturbidimetric methods (Cobas 6000 Analyser C501, Roche Diagnostics, Switzerland). Statistical analysis was performed using one-way ANOVA and SPSS 17. The present study was approved by the Committee of Scientific Ethics at the Medical University of Plovdiv, under outgoing number 3229 and record number 7 dated 17 Nov. 2021, with the rationale that the research meets the standards and criteria of scientific validity and ethics in accordance with the requirements of the Declaration of Helsinki on Ethics in Science.

Results

Table 1 shows the data from the paraclinical tests in the two studied groups—smokers and non-smokers with Type 1 diabetes mellitus—with one-way ANOVA. HBA1c at admission to the clinic showed no significant difference between smokers (9.15±1.66%) and non-smokers (10.07±1.98%), p=0.574 (Mann-Whitney U test). Regarding fasting serum glucose, significant differences were found only at 6 hours of clinic admission. Serum glucose was 15.59±6 mmol/L in smokers and 9.67±3.3 mmol/L in nonsmokers,

Table 1. Comparative analysis of the studied parameters upon admission to the clinic in smokers and non-smokers with (T1DM).

Parameters	Smokers (Means±SD)	Nonsmokers (Means±SD)	(p) One-Way ANOVA
Glucose 6 h. (mmol/lL)	15.59±6.0	9.67±3.38	0.004
Glucose 12 h.(mmol/lL)	12.11±6.18	11.14±5.88	>0.05
Glucose 17 h.(mmol/lL)	9.26±4.48	12.59±6.13	>0.05
Glucose 21 h.(mmol/lL)	11.95±4.25	13.02±4.8	>0.05
Glucose 24 h. (mmol/lL)	9.94±5.55	9.43±4.29	>0.05
HBA1c (%)	9.15±1.66	10.07±1.98	>0.05
TSH mIU/L	1.84±0.99	0.85±0.94	0.009
FT4 pmol/l	14.34±3.12	12.16±1.41	>0.05
TC mmol/lL	5.2±1.79	5.18±1.14	>0.05
HDL - C mmol/L	1.05±0.35	1.57±0.43	0.014
LDL - C mmol/L	3.23±1.63	3.07±1.48	>0.05
TAG mmol/L	2.04±1.21	1.78±0.37	>0.05
BMI kg/m2	23.21±4.62	26.16±4.79	0.028

Legend: smokers: 25; non-smokers: 48; fasting serum glucose (mmol/L) at 6, 12, 17, 21, 24 h upon admission to the clinic; HBA1c (%): glycated hemoglobin; TSH (mIU/L): thyroid stimulating hormone; FT4 (pmol/L): free tetraiodothyronine; TC (mmol/L): total cholesterol; HDL-C (mmol/L): high-density lipoproteins; LDL-C (mmol/L): low-density lipoproteins; TAG (mmol/L): triacyl glycerols; BMI (kg/m2): body mass index.

p=0.004 (one-way ANOVA), Fig. 1. We also found statistically significant (p<0.05): decreased BMI (23.21±4.62 vs 26.16±4.79), Fig. 2 and HDL-C (1.05±0.35 vs 1.57±0.43), Fig. 3, as well as increased TSH (1.84±0.99 vs 0.85±0.94), Fig. 4, in smokers compared to non-smokers.

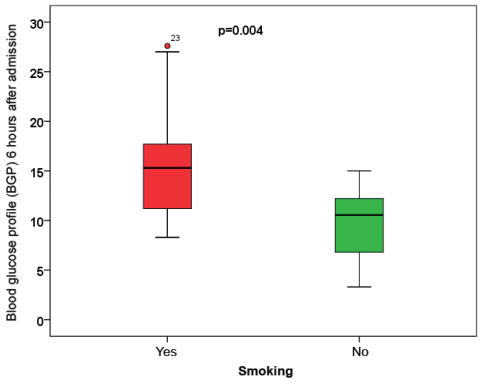

Figure 1. Comparative analysis of BGP (mmol/L) in 25 smokers and 48 nonsmokers with T1DM upon admission at 6 hours, one-way ANOVA. Blood glucose profile (BGP) 6 hours after admission to the clinic.

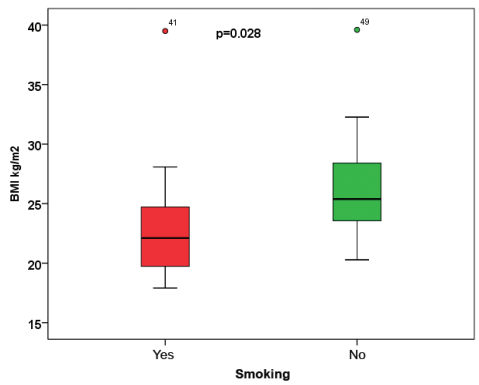

Figure 2. Comparative analysis of BMI (kg/m2) in 25 smokers and 48 non-smokers with T1DM upon admission, one-way ANOVA.

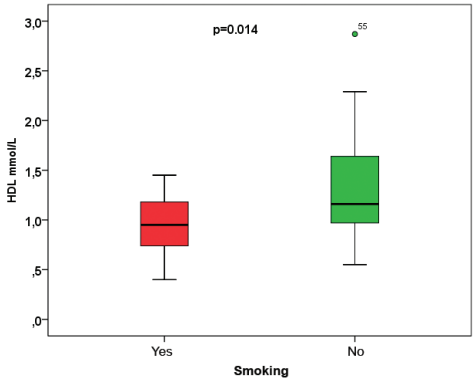

Figure 3. Comparative analysis of HDL (mmol/L) in 25 smokers and 48 nonsmokers with T1DM upon admission, one-way ANOVA.

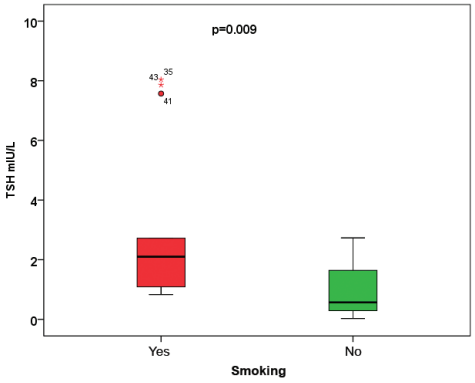

Figure 4. Comparative analysis of TSH (mIU/L) in 25 smokers and 48 non-smokers with T1DM upon admission, one-way ANOVA.

Discussion

Capillary blood glucose monitoring can help patients with T1DM achieve HbA1c control. For many people, an appropriate preprandial capillary plasma glucose is 4.4 to 7.2 mmol/L (80–130 mg/dL) (Holt et al. 2021). Smoking-induced oxidative stress may have some effect on serum glucose or may directly alter blood glucose homeostasis and cause insulin resistance. The exact pathophysiologic mechanism is not fully understood, but it is suspected that the high concentration of circulating catecholamines due to smoking may contribute to hyperglycemia by increasing the rate of hepatic gluconeogenesis and glycogenolysis (Mutiara et al. 2018). Smoking is one of the major risk factors for cardiovascular disease (Roth et al. 2017), as well as for macro- and microcirculatory complications caused subsequently. Smoking activates the oxidation of LDL-C, which is converted to oxLDL and, together with glycated LDL-C, yields the so-called advanced glycation end products (AGEs) that underlie the complications of diabetes and the development of atherosclerotic plaques. The formation of prostaglandins (PGI2) is inhibited (Jung et al. 2022). Current evidence suggests that regular smoking is an important risk factor for cardiovascular morbidity and mortality in patients with diabetes. The effect of smoking on glycaemic control in people with diabetes is insufficiently studied and has often conflicting results. One study found that cigarette smoking was independently associated with higher HbA1c concentrations in both men and women (Nishida et al. 2015). One possible explanation for this could be an increase in the glycation rate of HbA1c induced by exposure to glycotoxin from cigarette smoke or a relatively high degree of tissue hypoxia (Jansen et al. 2012). There is also evidence that smoking cessation often results in impaired glycemic control, possibly also because of the weight gain that often occurs after smoking abstinence (Bush et al. 2016). Our retrospective study found no significant increase in HbA1c in smokers with type 1 diabetes mellitus compared with nonsmokers, but smokers had significantly higher serum glucose values upon admission to the ward at 6 h. Clinical markers that assess vascular damage from smoking are lipids, but especially HDL-C and LDL-C (Zedler et al. 2006). Cigarette smoking is positively associated with elevated triglyceride levels and lower HDL-C levels (Gepner et al. 2011). Our retrospective study found statistically significant ($p<0.05$) decreased HDL-C in smokers compared to non-smokers with T1DM. Some authors have found a negative correlation between smoking and BMI (Smith et al. 2023), whereas other studies have found increased BMI in smokers (Park et al. 2022). Our study showed statistically significant ($p<0.05$) decreased BMI in smokers compared to nonsmokers with T1DM.

The link between diabetes and thyroid dysfunction has been known for decades. Most authors have studied the prevalence of thyroid disease among patients with T1DM due to the autoimmune nature of this disease (Franzese et al. 2000). There is conflicting data on the association between smoking and autoimmune thyroid disease. Fukata S et al. (Fukata et al. 1996) found that smoking increased the risk of developing hypothyroidism among smokers with Hashimoto's thyroiditis. Other researchers did not confirm the association between smoking and hypothyroidism (Vestergaard 2004), even arguing that smoking may reduce the risk of developing hypothyroidism (Asvold et al. 2007). Cho NH et al. (Cho et al. 2010) found that subclinical hypothyroidism was inversely proportional to current smoking status in female smokers. The protective role of smoking in the development of hypothyroidism has been suggested to be due to the immune system. We found a statistically significant increase in TSH levels in smokers compared to non-smokers with T1DM (Table 1 and Fig. 4). Several studies have shown that thyroid hormones can affect blood glucose levels. TSH can affect metabolic parameters and cause hyperglycemia by multiple mechanisms, including increasing leptin secretion (Duntas and Biondi 2013), increasing hepatic glucose production (Tian et al. 2010), and decreasing insulin production and secretion by pancreatic β cells (Santini et al. 2014). Appropriate control of diabetes can reduce the risk of developing thyroid disorders, and vice versa.

Conclusion

Smoking impairs glycemic control and thyroid function in patients with T1DM.

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