ORIGINAL ARTICLE

# Small intestinal bacterial overgrowth in adult patients with type 1 diabetes: its prevalence and relationship with metabolic control and the presence of chronic complications of the disease

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**KEY WORDS**

diabetes type 1, hydrogen breath test, intestinal complications, small intestinal bacterial overgrowth

**ABSTRACT**

**INTRODUCTION** Gastrointestinal symptoms may occur in 50% to 70% of patients with diabetes.

**OBJECTIVES** The aim of this study was to evaluate the prevalence of small intestinal bacterial overgrowth (SIBO) in patients with diabetes, as well as the relationship between SIBO and metabolic control of diabetes and the presence of chronic complications of the disease.

**PATIENTS AND METHODS** The study group included 148 patients with type 1 diabetes, treated in the years 2013–2015. The control group consisted of 41 healthy volunteers. The presence of SIBO was assessed with a noninvasive breath test using 20 g of lactulose suspended in 200 ml of water, with the assessment of exhaled hydrogen concentrations. The measurements were performed at 15-minute intervals in the first hour and at 30-minute intervals in the second hour of the test. A positive result was considered as the output value of exhaled hydrogen of 20 parts per million (ppm) or higher or an increase in the output value of the exhaled hydrogen of 12 ppm during the first 60 minutes of the test.

**RESULTS** We observed a lower prevalence of SIBO in the study group in comparison with controls (56 patients [37.8%] vs 30 healthy volunteers [73%]; $P = 0.006$). In the logistic regression model, this association was independent of age, sex, body mass index, cigarette smoking, serum C-reactive protein concentrations, and estimated glomerular filtration rate (odds ratio, 0.26; 95% confidence interval, 0.10–0.68; $P = 0.006$).

**CONCLUSIONS** The prevalence of SIBO in patients with type 1 diabetes is lower than that in healthy subjects. One of the possible causes might be the beneficial effect of nutritional therapy in patients with diabetes.

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**INTRODUCTION** Despite advances in the treatment of diabetes, the prevention of its chronic complications remains a significant clinical challenge. It seems that the duration and metabolic control of diabetes have major impact on the degree of organ involvement. It is believed that in the course of diabetes the gastrointestinal symptoms may occur in 50% to 70% of patients.[1,2] One of the possible causes is so called small intestinal bacterial overgrowth (SIBO). This is a heterogeneous syndrome, defined as the amount of nonpathogenic bacteria at or exceeding $10^5$ colony forming units (CFUs) per ml of the contents of the small intestine. The value considered as

**TABLE 1** Clinical characteristics of the study and control groups

| Variables | Study group (n = 148) | Control group (n = 41) | *P* value |
|---|---|---|---|
| age, y | 45 (35–54) | 31 (27–39) | <0.001 |
| sex, female/male, n | 54/94 | 24/17 | 0.001 |
| duration of diabetes, y | 20 (13.5–28) | – | – |
| smoking, n (%) | 36 (24.3) | 1 (2.4) | 0.007 |
| hypertension, n (%) | 71 (47.9) | 2 (4.88) | <0.001 |
| clinical symptoms, n (%) | 58 (53.7) | 28 (68.3) | 0.07 |
| BMI, kg/m$^2$ | 24.8 (22.7–28.4) | 22.6 (20.8–24.7) | 0.0002 |
| waist circumference, m | 0.9 (0.8–1.0) | 0.77 (0.72–0.9) | <0.001 |
| $HbA_{1c}$, % | 7.7 (7.1–8.7) | – | – |
| hs-CRP, mg/l | 1.16 (0.56–2.47) | 0.65 (0.46–1.3) | 0.007 |
| TG, mmol/l | 1.03 (0.76–1.4) | 0.91 (0.68–1.17) | 0.17 |
| LDL cholesterol, mmol/l | 2.71 (2.2–3.4) | 2.87 (2.38–3.57) | 0.28 |
| HDL cholesterol, mmol/l | 1.8 (1.45–2.1) | 1.74 (1.45–1.97) | 0.5 |
| creatinine, mg/dl | 0.89 (0.79–1.01) | 0.87 (0.81–0.92) | 0.23 |
| eGFR (MDRD), ml/min/m$^2$ | 115.3 (100.6–139.5) | – | – |
| peripheral neuropathy, n (%) | 50 (33.8) | – | – |
| autonomic neuropathy, n (%) | 14 (9.5) | – | – |

Data are presented as median (interquartile range) unless otherwise stated.

The Mann–Whitney test was used to compare continuous variables, and the Fisher exact test—to compare categorical variables.

Abbreviations: BMI, body mass index; eGFR, estimated glomerular filtration rate; $HbA_{1c}$, hemoglobin $A_{1c}$; HDL, high--density lipoprotein; hs-CRP, high-sensitive C-reactive protein; LDL, low-density lipoprotein; MDRD, Modification of Diet in Renal Disease; TG, triglycerides

the upper normal range is $10^4$ CFU/ml or less.[3,4] The syndrome may occur with or without gastrointestinal symptoms, which may include nonspecific abdominal pain, bloating, excessive gas production, diarrhea, and weight loss.[5,6] In severe cases, the consequences of SIBO may be avitaminosis, fat malabsorption, and malnutrition.[5] Stotzer et al[7] proved that patients with SIBO had low bone mineral density, which may be the result of reduced concentrations of vitamin $D_3$ (serum 25-hydroxycholecalciferol) and impaired calcium metabolism.

The microflora of the human digestive tract is a part of the complex ecosystem, regulated by the defense mechanisms of the host, environmental factors, and interactions between bacteria. Numerous factors may interfere with this balance, for instance, anatomic anomalies, motoric disorders (eg, scleroderma and other inflammatory diseases of the connective tissue in the digestive tract, diabetic autonomic neuropathy, radiation enteropathy, and the syndrome of chronic intestinal pseudo-obstruction), as well as conditions conducive to bacterial overgrowth: achlorhydia, long-term treatment with proton pump inhibitors or $H_2$-receptor blockers (drugs that inhibit acid secretion are often overused in clinical practice),[8] insufficiency of the exocrine pancreas, as well as congenital and acquired immunodeficiency syndromes. The majority of cases involve more than 1 causative factor.[1,3,5]

The diagnosis of SIBO is made on the basis of invasive and noninvasive tests. Invasive tests require aspiration of contents present in the light of the small intestine. Noninvasive tests, including hydrogen tests (BT-$H_2$, $H_2$-breath tests), require administration of glucose or lactulose.[3,4] The aim of this study was to investigate the prevalence of SIBO in patients with type 1 diabetes, as well as to examine associations of SIBO with the metabolic control and prevalence of diabetic neuropathy.

**PATIENTS AND METHODS** The study included 148 people with type 1 diabetes (94 men, 54 women), who remained under the care of the Department of Internal Medicine and Diabetology at the Poznan University of Medical Sciences, Poznań, Poland, in the years 2013–2015. The exclusion criteria were as follows: age <18 years, duration of diabetes <5 years, systemic treatment with antibiotics, proton pump inhibitors, or $H_2$-receptor blockers within 30 days before the test, history of comorbidities (inflammatory bowel disease, irritable bowel syndrome, celiac disease, endocrinopathies, malignant neoplasms, or immune deficits).

The control group consisted of 41 healthy volunteers with normal carbohydrate metabolism (based on medical history). Almost half of them (43.9%) were recruited from the personnel of the hospital and their relatives. The clinical characteristics of the study and control groups are presented in TABLE 1. All participants

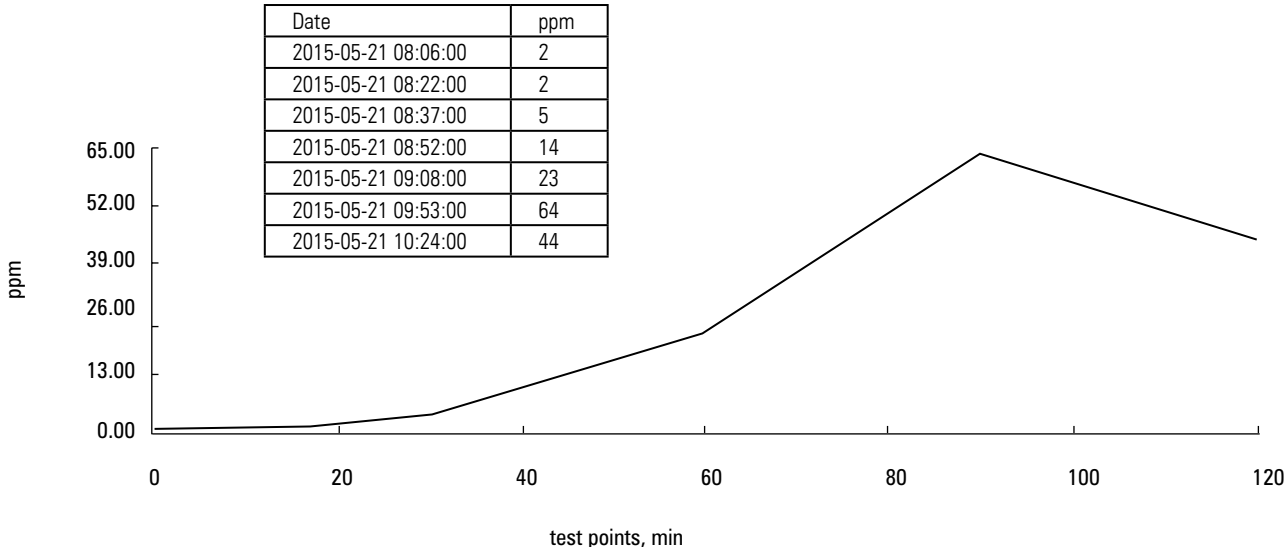


**FIGURE 1** Example of a positive test result for the presence of small intestinal bacterial overgrowth
Abbreviations: ppm, parts per million

gave written informed consent to participate in the study, which received approval of a local bioethics committee.

The study was conducted in the Department of Internal Medicine and Diabetology, Poznan University of Medical Sciences.

**Data collection** Patients with diabetes and healthy volunteers participating in the study completed a questionnaire containing demographic data (age, sex), smoking-related data, family record of diabetes, and information about other concomitant diseases and medication use. All subjects underwent a physical examination including anthropometric measurements (weight, height, waist circumference, and hip circumference) and blood pressure (twice using a sphygmomanometer in a sitting position after 10 minutes of rest). Participants completed a questionnaire on gastrointestinal symptoms: incidence of flatulence, excessive amounts of gas, occurrence of abdominal pain, number of bowel movements per day, presence of loose stools, fatty stools, as well as information about the loss of body weight, incidence of skin lesions, and duration of symptoms. The questionnaire for patients with type 1 diabetes also included data on duration of the disease and method of treatment.

**Laboratory tests** Blood samples were obtained after 10 hours of fasting, after a period of rest, with minimum occlusion of the vein in a Monovette system. The serum concentrations of total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides (TG), and creatinine were measured by standard methods.

The estimated glomerular filtration rate (eGFR) was calculated in accordance with the Modification of Diet in Renal Disease Study Group. The serum concentrations of C-reactive protein (CRP) were determined by a high-sensitivity method (hs-CRP). In the diabetes group, glycated hemoglobin was also determined ($HbA_{1c}$) by high-performance liquid chromatography, the values of which were calibrated with respect to the Diabetes Control and Complications Trial/National Glycohemoglobin Standardisation Program and the albumin-to-creatinine ratio taken for analysis from the first sample of urine in the morning. Laboratory tests were performed in a certified laboratory.

**Assessment of neuropathy in type 1 diabetes patients** Symptoms of peripheral neuropathy were evaluated on the basis of the medical history. During the standard examination, touch sensation was evaluated with the use of 10-g monofilament; feeling of vibration, with the 128-Hz tuning fork; temperature sensation, with TipTherm; and pain sensation, with neurotips. Achilles tendon reflexes were also evaluated. Diabetic neuropathy was diagnosed in patients with 2 or more of the following 5 elements: the occurrence of symptoms, abnormal sensation of touch, vibration, and/or temperature, and absence of ankle reflexes. Cardiac autonomic neuropathy was assessed with the use of ProsciCard III program. Heart rate variability at rest in a supine position and under standardized stimuli (deep breathing test, Valsalva maneuver, orthostatic test) was studied. During the examination, the electrocardiogram of the patient was monitored on the computer screen. On the basis of the calculated R-R interval, the parameters of autonomic neuropathy were calculated and subsequently compared with age- and sex-specific standard values. Autonomic neuropathy was diagnosed if the results of 2 of 4 tests were abnormal.

**Assessment of small intestinal bacterial overgrowth** All patients were studied after an overnight fast (for 8 hours). They were requested not to smoke 12 hours before and during the test. Patients drank the test solution (20 g of lactulose suspended in 200 ml of water). Hydrogen concentrations were measured with a breath-hydrogen analyzer (Gastro+Gastrolyzer, Bedfont Scientific Ltd, Maidstone, United Kingdom). Measurements were taken at 15-minute intervals in the first hour and at 30-minute intervals in the second hour. SIBO was defined as an elevated fasting

**TABLE 2** Clinical characteristics of patients with type 1 diabetes with a positive and negative test result for the presence of small intestinal bacterial overgrowth

| Variables | Negative result (n = 92) | Positive result (n = 56) | *P* value |
|---|---|---|---|
| age, y | 45 (35.5–54.5) | 44 (34–53) | 0.8 |
| sex, female/male, n | 33/59 | 21/35 | 0.86 |
| duration of diabetes, y | 20 (13.5–29) | 20.5 (13.5–27.5) | 0.74 |
| smoking, n (%) | 20 (21.7) | 16 (28.6) | 0.43 |
| hypertension, n (%) | 43 (46.7) | 28 (50) | 0.74 |
| clinical symptoms, n (%) | 38 (41.3) | 20 (35.7) | 1.0 |
| BMI, kg/m$^2$ | 24.9 (22.9–28.6) | 24.2 (22.7–27.8) | 0.43 |
| waist circumference, m | 0.91 (0.8–1.0) | 0.90 (0.76–1.00) | 0.61 |
| $HbA_{1c}$, % | 7.7 (7.1–8.7) | 7.7 (7.05–8.8) | 0.86 |
| hs-CRP, mg/l | 1.13 (0.52–2.28) | 1.16 (0.66–2.48) | 0.45 |
| TG, mmol/l | 1.03 (0.77–1.4) | 1.01 (0.73–1.49) | 0.91 |
| LDL cholesterol, mmol/l | 2.73 (2.3–3.4) | 2.69 (2.07–3.26) | 0.46 |
| HDL cholesterol, mmol/l | 1.8 (1.51–2.1) | 1.72 (1.36–2.11) | 0.39 |
| creatinine, mg/dl | 0.87 (0.79–1.01) | 0.91 (0.8–1.02) | 0.43 |
| eGFR (MDRD), ml/min/m$^2$ | 117.3 (100.8–139.2) | 113.5 (100.5–139.7) | 0.7 |
| peripheral neuropathy, n (%) | 32 (34.8) | 18 (32.1) | 0.74 |
| autonomic neuropathy, n (%) | 8 (8.7) | 6 (10.7) | 0.66 |

Data are presented as median (interquartile range) unless otherwise stated.

The Mann–Whitney test was used to compare continuous variables, and the Fisher exact test—to compare categorical variables.

Abbreviations: see TABLE 1

$H_2$ level of 20 parts per million (ppm) or higher or by the presence of a peak $H_2$ level of more than 12 ppm occurring in less than 60 minutes of the test. An example of the test result is presented in FIGURE 1.

**Statistical analysis** The results were analyzed with Statistica 10 software (Stat-Soft, Tulsa, Oklahoma, United States). Values were given as medians and interquartile ranges or as numbers and percentages. Normality of the distribution of quantitative data was evaluated with the Shapiro–Wilk test. Since the distribution of the vast majority of variables was skewed, the Mann–Whitney test was used in comparative analyses. Comparisons of categorical variables were conducted using the exact Fisher test. In order to simultaneously assess the potential factors associated with the occurrence of SIBO, a multivariate logistic regression model was used.

**RESULTS** The presence of SIBO was found in 56 patients with type 1 diabetes (37.8%) and in 30 healthy volunteers (73%) ($P$ = 0.006). In the logistic regression model, the presence of diabetes was associated with a lower incidence of SIBO, regardless of age, sex, body mass index (BMI), cigarette smoking, hs-CRP levels, and eGFR (odds ratio, 0.26; 95% confidence interval, 0.10–0.68; $P$ = 0.006). The comparison of type 1 diabetes patients with and without the evidence of SIBO is presented in TABLE 2. There was no association between SIBO symptoms and positive hydrogen test results, but the absence of symptoms did not exclude the presence of SIBO. The intestinal bacterial overgrowth may be asymptomatic.[1]

**DISCUSSION** Intestinal microflora is a complex and dynamic ecosystem. A qualitative analysis of this ecosystem is challenging, mainly because of the difficulties in performing in vitro studies. Sequencing of the *16S rRNA* gene (from amplification of bacterial genetic material from stool samples) allowed for a more accurate assessment of the intestinal flora.[9,10] Eckburg et al[11] conducted a study of intestinal microflora and found that 90% are Bacterioides and Firmicutes. Research using molecular biology methods shows that the same types of bacteria are present in the different parts of the intestine, but depending on the location, different subtypes predominate.[12]

SIBO is defined as bacterial overgrowth (increase in the number of and changes in the type of bacteria) in the small intestine.[1] The characteristics of microflora, not only gastrointestinal but also that in the skin or genital and urinary systems, have been examined by the international Human Microbiome Project. One of the most important factors influencing the composition of intestinal microflora is diet.[13] Other important factors include age, sex, and genetic and environmental factors.[14] Significant differences in the flora composition were observed between people living in different regions of the world.

It is currently believed that the modification of intestinal microflora can have a positive effect on

the metabolism of the host. In their meta-analysis, Kasińska and Drzewoski[15] showed that supplementation with probiotics significantly reduced $HbA_{1c}$ levels and insulin resistance in patients with type 2 diabetes. However, there have been no studies investigating these effects in patients with type 1 diabetes.

A possible limitation of our study is the lack of detailed nutritional data obtained using, for example, dietary questionnaires. However, this method is difficult and time-consuming for patients (which can limit the size of the study group). It is also subjective. Numerous authors recognize the direct aspiration duodenal fluid as the gold standard.[3] However, the method is invasive and expensive, which limits its wide application. In contrast, the $H_2$-breath test is noninvasive, easily accessible, and acceptable by most patients.

The $H_2$-breath test is based on the mechanism via which part of the gas produced by bacteria fermentation diffuse into the blood and is rapidly exhaled by breath, where it can be measured. In patients with diabetes, lactulose is more appropriate because it does not increase glycemia. The lactulose $H_2$-breath test was used by Mendoza et al,[16] who validated the test by comparison with bacterial cultures from duodenal aspirate. The reported sensitivity of the $H_2$-breath test of 85.7% and specificity of 90.9% show a high concordance between $H_2$ and lactulose breath tests. These results indicate the possible use of the lactulose $H_2$-breath test as a screening tool for SIBO.[16]

There are scarce data on SIBO in patients with type 1 diabetes. Faria et al,[17] in a group of 28 patients with long-standing (>10 years) type 1 diabetes, assessed the orocecal transit time and the presence of SIBO with the lactulose $H_2$-breath test. The analysis showed that there was no significant association between the prolonged orocecal transit time and SIBO.[17]

Virally-Monod et al[18] reported the presence of SIBO in 43% of patients with diabetes; therefore, it should be suspected in the case of chronic diarrhea in patients with diabetes. In our study, the prevalence of SIBO among patients with type 1 diabetes was 37.8%.

We showed that the incidence of SIBO was independent of BMI. This is in contrast to the results of a study in obese asymptomatic patients, in which SIBO was found in 17% of obese and 2.5% of nonobese patients.[19] The discrepancy may result from the low prevalence of obesity in our study group.

Zieth et al[20] reported that SIBO was associated with cardiovascular autonomic neuropathy in one-third of the patients with diabetes. Contrary to this finding, our study did not confirm the association between diabetic autonomic neuropathy and SIBO (by means of the ProsciCard III program).[20]

An appropriate therapy in patients with SIBO allows permanent or temporary removal of the causes of intestinal complaints. However, recommendations on the treatment of SIBO are inconclusive. Shah et al[21] performed a systematic review and meta-analysis comparing the effectiveness of antibiotic therapies in the treatment of patients with SIBO. The most studied antibiotic was rifaximin (8 of 10 studies).[21] According to Lauritano et al,[22] rifaximin showed a higher SIBO decontamination rate than metronidazole at the tested doses (the glucose breath test was reassessed 1 month after). Moroever, the prevalence of adverse events was significantly lower in patients receiving rifaximin in comparison with those receiving metronidazole.[22] Furnari et al[23] reported that 87% of patients treated with an oral antibiotic showed normal results of hydrogen breath tests. In a pilot study conducted by Khalighi et al[24] with the aim to evaluate the efficacy of a probiotic with lactic acid bacteria in the treatment of SIBO, patients receiving the probiotic demonstrated a better response than the control group (negative $H_2$-breath test result in 93.3% of those receiving probiotic compared with 66.7% of the control group). In all patients receiving the probiotic, abdominal pain completely subsided.[24]

To date, no studies have been published concerning the treatment recommendations specifically for patients with type 1 diabetes and SIBO. In addition, no posttreatment hydrogen respiratory tests have been conducted so far in patients with diabetes and SIBO. Once data from prospective studies become available, we will be able to draw conclusions with clinical implications.

In summary, the incidence of SIBO is lower in diabetic patients than in healthy subjects, but further research is needed to explain the association between the two conditions.

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ARTYKUŁ ORYGINALNY

# Zespół rozrostu bakteryjnego jelita cienkiego u dorosłych z cukrzycą typu 1: częstość występowania oraz związek z wyrównaniem metabolicznym cukrzycy i obecnością przewlekłych powikłań choroby

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**SŁOWA KLUCZOWE**

cukrzyca typu 1, powikłania jelitowe w cukrzycy, wodorowe testy oddechowe, zespół rozrostu bakteryjnego jelita cienkiego

**STRESZCZENIE**

**WPROWADZENIE** Dolegliwości ze strony przewodu pokarmowego mogą występować u 50–70% osób z cukrzycą.

**CELE** Celem pracy była ocena częstości występowania zespołu rozrostu bakteryjnego jelita cienkiego (*small intestinal bacterial overgrowth* – SIBO) u pacjentów z cukrzycą, a także związek występowania SIBO z wyrównaniem metabolicznym cukrzycy i obecnością przewlekłych powikłań choroby.

**PACJENCI I METODY** Grupę badaną stanowiło 148 osób z cukrzycą typu 1 leczonych w latach 2013–2015. Grupę kontrolną stanowiło 41 zdrowych ochotników. Obecność SIBO oceniano za pomocą nieinwazyjnego testu oddechowego z wykorzystaniem 20 g laktulozy rozpuszczonej w 200 ml wody z oceną stężenia wodoru w wydychanym powietrzu. Pomiary wykonano w 15-minutowych odstępach w czasie pierwszej godziny i 30-minutowych odstępach w trakcie drugiej godziny badania. Za wynik dodatni testu uznano: wyjściową wartość wydychanego wodoru ≥20 części na milion (*parts per milion* – ppm) lub wzrost wartości wyjściowej wydychanego wodoru o 12 ppm w czasie pierwszych 60 minut badania.

**WYNIKI** Obserwowano mniejszą częstość SIBO w grupie badanej w porównaniu z grupą kontrolną (56 pacjentów [37,8%] w porównaniu z 30 zdrowymi ochotnikami [73%]; p = 0,006]. W modelu regresji logistycznej ten związek był niezależny od wieku, płci, wskaźnika masy ciała, palenia papierosów, stężenia białka C-reaktywnego w surowicy oraz wielkości przesączania kłębuszkowego (OR 0,26; 95% CI: 0.10–0.68; p = 0,006).

**WNIOSKI** U pacjentów z cukrzycą typu 1 częstość występowania SIBO jest niższa niż u osób zdrowych. Jedną z możliwych przyczyn stwierdzonego związku może być korzystny wpływ leczenia żywieniowego u osób z cukrzycą.

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