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ORIGINAL ARTICLE

Postprandial Glucose Monitoring Further Improved Glycemia, Lipids, and Weight in Persons with Type 2 Diabetes Mellitus Who Had Already Reached Hemoglobin A1c Goal

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Abstract

Background:

Postprandial hyperglycemia contributes to poor glucose control and is associated with increased cardiovascular risk in type 2 diabetes mellitus (T2DM). The objective of the study was to determine the effect of postprandial self-monitoring of blood glucose (pp-SMBG) on glucose control, lipids, body weight, and cardiovascular events.

Method:

Subjects with T2DM hemoglobin A1c (A1C) between 6.5 to 7.0% were randomized into the study group (at least two pp-SMBG a day and dietary modification based on glucose readings) and control group (dietary modification based on glucose readings but no mandatory pp-SMBG) for a 6-month, observational study. Oral antidiabetic drugs or insulin regimen was unchanged in either group if A1C remained less than 7.0% during the study. End points included A1C, lipids, body weight, and cardiovascular events.

Results:

One hundred sixty-nine subjects, mean age 63 years, and body weight 88 kg were recruited. Hemoglobin A1c, weight, low-density lipoprotein (LDL), and triglycerides (TGs) were similar in the groups at baseline. By the end of 6 months, A1C (6.7 ± 0.1 to 6.4 ± 0.1%, $p < .05$), body weight (88.5 ± 7.3 to 85.2 ± 6.3 kg, $p < .05$), LDL (92.3 ± 2 8.4 to 81.1 ± 22.6 mg/dl, $p < .05$), and TGs (141 ± 21 to 96 ± 17 mg/dl, $p < .05$) decreased in the study group, but did not change in the control group. No cardiovascular events were observed in either group during the 6-month study period.

Conclusions:

In T2DM subjects who had already reached their A1C goal, pp-SMBG at least twice a day was associated with further improvement in glycemia, lipids, and weight, as well as exercise and dietary habit. We assume that lifestyle modification promoted by postprandial hyperglycemia awareness may underlie these findings. These results substantiate the importance of implementing pp-SMBG into lifestyle modification, and emphasize that pp-SMBG is critical in the control of T2DM.

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Abbreviations: (ADA) American Diabetes Association, (A1C) hemoglobin A1c, (LDL) low-density lipoprotein, (PPBG) postprandial blood glucose, (pp-SMBG) postprandial self-monitoring of blood glucose, (SD) standard deviation, (T2DM) type 2 diabetes mellitus, (TG) triglyceride

Keywords: lifestyle modification, postprandial glucose monitoring, postprandial hyperglycemia, type 2 diabetes mellituss

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Introduction

Postprandial hyperglycemia contributes to poor glucose control.[1–4] Furthermore, postprandial hyperglycemia has been implicated as a cardiovascular risk factor in patients with type 2 diabetes mellitus (T2DM).[5–7] Insulin deficiency, insulin resistance, and poor dietary habits (refined carbohydrates, large portions, high calorie intake) are the proposed causes of postprandial hyperglycemia.[8–11] The objective of this study was to determine the effect of postprandial self-monitoring of blood glucose (pp-SMBG) and self-dietary adjustment on glucose control, lipids, body weight, and cardiovascular events.

Methods

We performed a randomized, controlled trial on 169 T2DM patients with hemoglobin A1c (A1C) between 6.5 to 7.0%, selected from over 4500 T2DM patients in our clinic. Eighty-nine patients were randomly assigned to the study group (at least two pp-SMBG a day and self-dietary modification) and 80 patients were randomly assigned to the control group (self-dietary modification but no mandatory pp-SMBG) for a 6-month period. Baseline characteristics of T2DM patients in the study and control groups are given in (**Table 1**).

Sixty-two percent of patients in the study group and 65% of patients in the control groups were treated with oral antidiabetic drugs, including metformin, sulfonylurea, and thiazolidinediones. Three percent of patients in the study group and 1% of patients in the control group were treated with glargine insulin, respectively. Statins were used in 77% of patients in the study group and fenofibrates were used in 25% of patients in the study group. Statins were used in 81% of patients in the control group and fenofibrates were used in 29% of patients in the control group.

Table 1.
Clinical Characteristics of Study Participants

Variable	Study group (n = 89) 40 males/49 females	Control group (n = 80) 36 males/44 females	P
	Mean ± SD	Mean ± SD	
Age (year)	63.6 ± 6.4	62.3 ± 6.9	0.77
Weight (kg)	88.5 ± 7.3	87.5 ± 13.6	0.75
A1C (%)	6.7 ± 0.1	6.7 ± 0.1	0.83
LDL (mg/dl)	92.3 ± 28.4	92.4 ± 18.8	0.23
TG (mg/dl)	141.4 ± 21.3	140.8 ± 23.1	0.19

Both groups received advice on lifestyle modification prior and during the study period including regular exercise (at least 30 min duration, 5 times per week), weight reduction, glycemic load, and self-dietary modification targeting fasting blood glucose <110 mg/dl and 2 h postprandial blood glucose (PPBG) <140 mg/dl. A diet based on guidelines of the American Diabetes Association (ADA) and Joslin Diabetes Center was recommended to both groups including a meal plan of 3–5 servings of carbohydrate (or 45–75 g of carbohydrates), 2–4 protein (or 12–24 g of protein), and 1–2 fat (or 3.5 to 7 g of fat). Complex carbohydrates, monounsaturated fats, and vegetable proteins were recommended over processed refined carbohydrates, saturated fat, and animal proteins. Patients kept records on daily exercise, postprandial glycemia, calorie intake, and servings of food groups. No bolus insulin or oral antidiabetic drugs were applied based on PPBG during the study period. Oral antidiabetic drugs or insulin regimen was unchanged in either group if A1C remained less than 7.0% during the study period. Two-tailed paired comparisons of beginning and ending A1C, lipids, and body weight values were used to determine if pp-SMBG was effective. Results were reported as mean ± standard deviation (SD) and $p < .05$ was considered significant. One sample t-test was employed where percent variables were compared.

Results

Dietary records from patients in the study group revealed an association between postprandial hyperglycemia and high intake of refined carbohydrates, calories, saturated fat, and physical inactivity (**Table 2**). During the first month, pp-SMBG revealed postprandial hyperglycemia while 30% of the recorded meals were high in refined carbohydrates, calories, and saturated fat, and only 2% of the patients excised regularly. By the end of the sixth month, pp-SMBG revealed improved postprandial glycemia while only 6% of the meals were high in refined carbohydrates, calories, and saturated fat, and

10% of the patients exercised regularly. On the other hand, none of the patients in the control group had at least two pp-SMBG a day for the 6-month duration; their dietary records revealed a less desirable change at the end of the 6 months (**Table 2**).

By 6 months, there was a significant reduction in A1C (6.7 ± 0.1 to 6.4 ± 0.1%, $p < .05$), low-density lipoprotein (LDL) (92.3 ± 28.4 to 81.1 ± 22.6 mg/dl, $p < .05$), triglycerides (TG) (141.4 ± 21.3 to 96.7 ± 17.7 mg/dl, $p < .05$), and body weight (88.5 ± 7.3 to 85.2 ± 6.3 kg, $p < .05$) in the study group, but no significant reduction was observed in the control group (**Table 3** and **Figure 1**). No cardiovascular events were observed in either group during the 6-month study period.

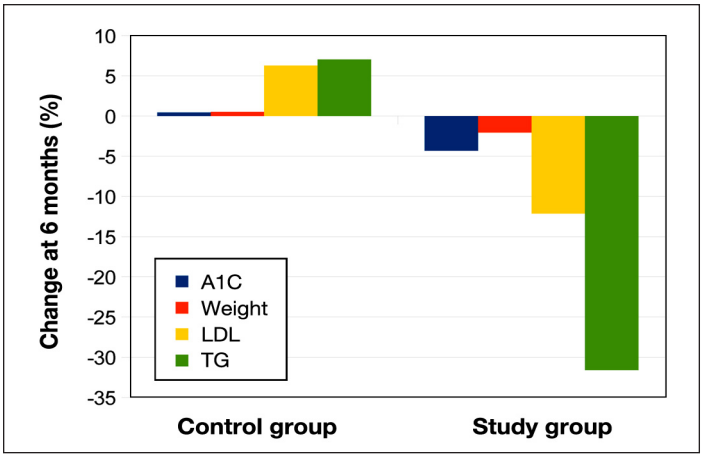

Figure 1. Percentage changes in A1C, weight, LDL, and TG over 6 months.

Table 2.
Changes in Selected Variables from Baseline in Study and Control Groups[a]

Variables	Study group (n = 89)			Control group (n = 80)		
	1st month	6th month	p	1st month	6th month	p
Total intake per day (calories)	2716 ± 816	2134 ± 368	<.05	2806 ± 1012	2522 ± 983	.09
Refined carbohydrate[b]						
No. of servings/day	13.6 ± 3	2.1 ± 0.4	<.05	15.1 ± 3	14.2 ± 4	0.8
% calories of total daily intake	30%	6%	<.05	34%	32%	0.8
High in saturated fat[c]						
No. of servings/day	4 ± 2	0.6 ± 0.3	<.05	4 ± 2	3 ± 2	0.1
% calories of total intake	30%	6%	<.05	29%	25%	0.6
% of individuals taking > 29 cal/kg/day[d]	30%	6%	<.05	32%	29%	0.7
% of individuals exercising regularly	2%	10%	<.05	3%	5%	0.5
Postprandial blood glucose (mg/dl)	148 ± 7	139 ± 5	<.05			

[a] Data are presented as mean ± SD except where otherwise indicated.
[b] White rice, white bread, refined sugar, pastries
[c] Sausage, rendered animal fat, whipped cream, hydrogenated oil
[d] 29 cal/kg/day is the recommended calorie intake to maintain a stable body weight in the clinic.

Table 3.
Changes in Selected Variables from Baseline in Study and Control Groups

Variable	Study group (n = 89) Mean ± SD			Control group (n = 80) Mean ± SD		
	baseline	6 month	P	baseline	6 month	P
Weight (kg)	88.5 ± 7.3	85.2 ± 6.3	<.05	87.5 ± 13.6	87.9 ± 13.1	0.19
A1C (%)	6.7 ± 0.1	6.4 ± 0.1	<.05	6.7 ± 0.1	6.7 ± 0.1	0.13
LDL (mg/dl)	92.3 ± 28.4	81.1 ± 22.6	<.05	92.4 ± 18.8	98.2 ± 56.8	0.34
TG (mg/dl)	141.4 ± 21.3	96.7 ± 17.7	<.05	140.7 ± 23.1	150.6 ± 25.9	0.43

Discussion

Interestingly, this study provides evidence that in patients with T2DM who had already reached the A1C goal, simple home-based pp-SMBG at least twice a day was associated with further improvement in glycemia, lipids, and body weight. We assume that self-dietary adjustment, and life style modification promoted by postprandial hyperglycemia awareness may underlie these findings. This assumption is based on review of dietary records where decreased intake of refined carbohydrates, calories, and saturated fat and increased physical activity were observed in the study group by 6 months. Our data support the notion that PPBG significantly contributes to overall glycemic exposure, and dietary modification aiming to improve postprandial hyperglycemia significantly lowers A1C, particularly when it is close to 7.0%.

Our dietary records revealed an association between postprandial hyperglycemia and high intake of rapidly digested/absorbed refined carbohydrates. Furthermore, the dietary records identified that those who ate refined carbohydrates were eaters of excessive amount of saturated fat, calories, and were physically inactive. This association points to the intriguing possibility that the combined factors may act synergistically to result in a larger postprandial glycemic excursions, exacerbated inflammatory processes, and increased cardiovascular risk.[11–16] Because of the limited time course of our study, we could not adequately evaluate clinical events.

All our patients had self-recorded daily calorie intake and numbers of servings related to refined carbohydrate and food high in saturated fat, however the records were by no means without flaws. In addition, only 76% of the records had complete recordings on other food groups, such as nuts, herbs, and fibers (data not shown). Therefore, we were not able to completely assess the effects of all food groups on the improved metabolic parameters.

Other possible biases/limitations of our data analysis also includes nonidentical numbers of patients using basal insulin and different classes of oral drugs between the two groups. However, we consider this possibility unlikely based on the fact that all patients, at the beginning of the study, had been on the same regimen with close-to-target A1C, lipids, and weight for more than 5 months.

Although the key question of whether postprandial hyperglycemia is a risk factor for cardiovascular disease is remains unanswered, our data indicate that implementing pp-SMBG strategy aiming to lower postprandial hyperglycemia is a good therapeutic choice because it improves lipids, weight, and A1C when it is already close to target. In addition, adherence to a healthier lifestyle promoted by postprandial hyperglycemia awareness is always very good for our patients with diabetes.

Conclusion

In T2DM patients who had already reached the A1C goal, pp-SMBG at least twice a day was associated with further improvement in glycemia, lipids, and weight, as well as diet and exercise habits. We presume that self-dietary adjustment and lifestyle modification promoted by postprandial hyperglycemia awareness underlie these findings. These results substantiate the importance of implementing pp-SMBG into lifestyle modification to achieve optimal control of T2DM.

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