Continuous Glucose Monitoring in the Self-management of Type 2 Diabetes: A Paradigm Shift

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e-LETTERS – OBSERVATIONS

Conventional lifestyle modification for type 2 diabetes involves 7% weight loss, avoidance of high-energy foods, and 150 min of weekly exercise (1). Although effective at preventing and treating diabetes, this approach is not appropriate for those who do not need or want to lose weight or who cannot maintain weight loss. An alternative may be to target postprandial glucose (PPG), the major contributor to HbA_{1c} (2). The GEM (glycemic load, exercise, and monitoring blood glucose) lifestyle modification program focuses on diminishing PPG by selecting low–glycemic load foods to prevent PPG spikes, reducing PPG with postprandial exercise, and using systematic self-monitoring of blood glucose (SMBG). Systematic SMBG educates individuals about the impact of different foods and physical activities on PPG, activates them to take action when glucose is outside of desired limits, and motivates them to repeat choices that produce desired glucose levels. Therefore, GEM represents a paradigm shift from reducing insulin resistance through reducing fat to reducing PPG.

In a preliminary study, GEM effectively lowered HbA_{1c} and promoted positive but not negative side effects (3). Despite the potential benefits of systematic SMBG to educate, activate, and motivate positive choices, GEM subjects in the preliminary study (3) did not perform more SMBG than subjects undergoing routine care, despite receiving free SMBG supplies. We hypothesized that more robust glucose feedback might be advantageous.

Here reported is the feasibility and efficacy of replacing systematic SMBG with continuous glucose monitoring (CGM) to increase qualitative and quantitative feedback. By simply glancing at a CGM device, individuals can immediately access their current glucose level and its direction and rate of change.

Six recently diagnosed adults followed the GEM program and monitored glucose with CGM. Two dropped out because of psychiatric issues. Participants read five GEM manual chapters at home and then discussed their relevance to their daily routine during each of five group sessions (see preliminary study for details [3]). Participants were assessed at baseline and at 3-month follow-up. Subject demographics and results are shown in Table 1; the last column shows results from the GEM/SMBG preliminary study.

All subjects substantially lowered their HbA_{1c}. Mean HbA_{1c} fell from 7.8 to 6.7%, which is a greater improvement than the reduction from 8.4 to 7.4% in the preliminary study. Participants also consumed fewer high–glycemic load foods, fewer grams of carbohydrate, and fewer calories without increasing fat intake.

Unlike those who used SMBG, subjects who monitored glucose with CGM during GEM reported eating more low–glycemic load foods in their daily routine and more frequently chose low–glycemic load foods in a behavioral challenge during pre/post assessments. Additionally, subjects who used CGM appeared to perform more SMBG and have fewer diabetes-associated problems (evaluated via Problem Areas in Diabetes [PAID] questionnaire) at follow-up.

This pilot study affirms the efficacy of GEM and further suggests that GEM intervention may be augmented with CGM to provide continuous and immediate feedback about the consequences of one's food and activity choices on glucose levels. These CGM benefits are consistent with the findings of Vigersky et al. (4). Studies that highlight the central role of glucose feedback in GEM, to educate, activate, and motivate individuals, contrast with research that found little benefit of nonsystematic use of SMBG by individuals managing

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Table 1—Demographics and pre/post measurements of subjects in the CGM/GEM study and group means for the earlier SMBG/GEM study

	Assessment	Subjects				Mean	SD	SMBG/GEM (group mean)
		GEM052	GEM053	GEM054	GEM055			
Demographics								
Age (years)		60	57	66	65	62.0	4.2	55.3
Sex		F	F	M	F			
BMI (kg/m^2)		55.5	30.6	26	38.1	37.6	13.0	
Duration of type 2 diabetes (years)		4.5	2	1	3	2.6	1.5	2.1
Medication (0 = none, 1 = metformin, 2 = two medications)		2	1	1	1	1.3	0.5	1
Metabolic control								
HbA$_{1c}$ (%) (Labs_HbA$_{1c}$)	Pre	8	8	7	8	7.8	0.5	8.4
	Post	6.3	7.2	6.1	7	6.7	0.5	7.4
Self-regulatory behaviors								
Knowledge (Qz total score)	Pre	17	10	18	13	14.5	3.7	15.5
	Post	15	18	20	22	18.8	3.0	16.9
High–glycemic load foods (Qx_HGL)	Pre	21	25	18	45	27.3	12.2	30.7
	Post	9	7	0	9	6.3	4.3	14.9
Low–glycemic load foods (Qx_LGL)	Pre	22	36	47	34	34.8	10.2	42.4
	Post	42	22	76	89	57.3	30.7	37.5
Behavioral challenge (1 = low, 0 = high)	Pre	0	0	1	0	0.25		0.41
	Post	1	1	1	1	1		0.54
Total carbohydrates (ASA24_CARB)	Pre	328	328	126	191	243.3	101.4	223.3
	Post	215	161	110	116	150.5	48.7	131.1
Total fiber (ASA24_FIBE)	Pre	43	15	20	18	24.0	12.8	19.9
	Post	17	11	26	12	16.5	6.9	15.9
Total fat (ASA24_TFAT)	Pre	122	192	64	142	130.0	52.9	93.6
	Post	119	108	89	54	92.5	28.5	77.7
Saturated fat (ASA24_SFAT)	Pre	30	63	15	41	37.3	20.2	28.9
	Post	37	46	36	29	37.0	7.0	23.9
Protein (ASA24_PROT)	Pre	87	129	119	161	124.0	30.5	88.8
	Post	122	111	127	29	97.3	46.0	83.3
Calories (ASA24_KCAL)	Pre	2,698	3,557	1,510	2,956	2,680.3	859.2	2,085
	Post	2,399	2,059	1,708	1,019	1,796.3	590.0	1,545
Pedometer (steps)	Pre	1.67	0	32	0	8.4	15.7	20.1
	Post	13	0	39	0	13.0	18.4	35.4
#SMBG (SMBG/day)	Pre	4	4	5	5	4.3	0.6	2.9
	Post	2	2	3	2	2.4	0.4	1.6
Physical measurements								
SBP (Stats_Sys)	Pre	123	128	139	149	134.8	11.6	124.4
	Post	119	138	146	129	133.0	11.6	128.4
DBP (Stats_Dia)	Pre	70	70	90	77	76.8	9.4	79.1
	Post	79	85	98	84	86.5	8.1	81.4
Weight (Stats_Wt)	Pre	347	180	182	214	230.8	79.1	221
	Post	315	173	169	202	214.8	68.4	213.2
Blood tests								
HDL (Labs_HDL)	Pre	40	69	44	53	51.5	12.9	38.8
	Post	44	75	45	48	53.0	14.8	41.8
LDL (Labs_LDL)	Pre	85	176	122	128	127.8	37.4	101.9
	Post	69	176	134	152	132.8	45.9	110
Triglycerides (Labs_Tri)	Pre	130	180	96	153	139.8	35.6	161.9
	Post	82	71	83	192	107.0	56.9	175
Psychological measurements								
PAID-5	Pre	13	0	2	11	6.5	6.5	7.9
	Post	2	0	0	11	3.3	5.3	5.8

See the preliminary study (3) for more details on SMBG/GEM study data. DBP, diastolic blood pressure; F, female; M, male; SBP, systolic blood pressure.

type 2 diabetes (5). The small sample size and two dropouts limit extrapolation of findings.

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References

1. Inzucchi SE, Bergenstal RM, Buse JB, et al.; American Diabetes Association (ADA); European Association for the Study of Diabetes (EASD). Management of hyperglycemia in type 2 diabetes: a patient-centered approach: position statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care 2012;35:1364–1379

2. Monnier L, Lapinski H, Colette C. Contributions of fasting and postprandial plasma glucose increments to the overall diurnal hyperglycemia of type 2 diabetic patients: variations with increasing levels of HbA_{1c}. Diabetes Care 2003;26:881–885

3. Cox DJ, Taylor AG, Singh H, et al. Glycemic load, exercise, and monitoring blood glucose (GEM): a paradigm shift in the treatment of type 2 diabetes mellitus. Diabetes Res Clin Pract 2016;111:28–35

4. Vigersky RA, Fonda SJ, Chellappa M, Walker MS, Ehrhardt NM. Short- and long-term effects of real-time continuous glucose monitoring in patients with type 2 diabetes. Diabetes Care 2012;35:32–38

5. Farmer AJ, Perera R, Ward A, et al. Meta-analysis of individual patient data in randomised trials of self monitoring of blood glucose in people with non-insulin treated type 2 diabetes. BMJ 2012;344:e486