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Postprandial Plasma Glucose Response and Gastrointestinal Symptom Severity in Patients With Diabetic Gastroparesis

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Abstract

Background: Gastroparesis is a well-known diabetic complication. The pathogenesis is not fully understood. However, it is important to early diagnose these patients.

Method: This study evaluated the plasma glucose response after a test meal, and gastrointestinal (GI) symptom severity in patients with clinical suspicion of diabetic gastroparesis, and assessed its usefulness to predict gastroparesis. In all, 83 subjects with insulin-treated diabetes mellitus (DM) type 1 and 2 were included; 53 subjects had gastroparesis and 30 had normal gastric emptying determined by gastric scintigraphy. GI symptom severity during the preceding 2 weeks was evaluated with a validated questionnaire. The test meal consisted of 100 g meat, 40 g pasta, 150 g carrot, and 5 g oil. The subjects ingested the meal under fasting conditions, and plasma glucose was followed during 180 minutes.

Results: Patients with gastroparesis demonstrated a blunted plasma glucose response after a test meal versus patients with normal gastric emptying ($P < .005$), reflected by lower maximum increase in plasma glucose response and incremental area under the curve of the plasma glucose, but a similar time to the maximum plasma glucose level. All GI symptoms were more severe in patients with gastroparesis. GI symptom severity had the best discriminative value to identify patients with gastroparesis with an area under the receiver operating curve of 0.83 (optimal cutoff: sensitivity 87%, specificity 80%).

Conclusions: Patients with diabetic gastroparesis have a blunted postprandial plasma glucose response. Combining this information with the presence of GI symptoms can help clinicians identify diabetic patients with gastroparesis.

Keywords

diabetic gastroparesis, gastrointestinal symptom, postprandial glucose response, postprandial glucose, test meal.

Gastroparesis is defined as delayed gastric emptying in the absence of an obstruction to outflow from the stomach.[1] This is a well-known diabetic complication and occurs in both diabetes mellitus (DM) type 1 and type 2.[2] The pathogenesis of gastroparesis is not fully understood, but 1 important factor is GI autonomic neuropathy.[3,4] The prevalence is uncertain but studies have suggested 30-65% in patients with long-standing DM.[2] Many patients with diabetic gastroparesis (DGP) suffer from GI symptoms associated with decreased quality of life,[5,6] which sometimes contribute to poor nutritional status, caused by inadequate oral intake of nutrients, but also by losses from vomiting and/or diarrhoea.[7] The association between GI symptoms and the degree of delayed gastric emptying has varied between studies, but abdominal bloating and postprandial fullness[2] and abdominal pain[8] have been found to correlate significantly with the presence of gastroparesis.[2] Upper gastrointestinal motor function is a critical determinant of postprandial glucose concentration.[9-11] Many DGP have, therefore unstable plasma glucose and studies have also confirmed that glycosylated hemoglobin (HbA1c), is higher in DGP.[12] As good metabolic control is of importance to prevent further diabetic complications,[13] it is therefore important that patients with gastroparesis are properly diagnosed and receive adequate care.[13]

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A common perception is that gastroparesis is uncommon and often under-recognized, inadequately investigated and poorly managed.[14,15] Gastric scintigraphy is the gold standard to measure gastric emptying and to make a diagnosis of gastroparesis.[16] However, the method is expensive and not widely available.[17,18] Other measurements of gastric emptying, such as breath tests, a wireless motility capsule,[18] and gastric emptying of radiopaque markers[19] are also not easily accessible for all clinicians. Therefore it would be useful to have clinical parameters that would allow clinicians to suspect gastroparesis, and to function as an office-based screening tool. Moreover, a test that could be performed by the patient himself or herself would be even more advantageous. Available studies demonstrate that the postprandial plasma glucose response is affected in DGP, such as a reduced postprandial glucose increase,[20] a longer time to peak blood glucose after meal intake and an extended period of postprandial hyperglycemia.[21]

Based on the presence of abnormal postprandial glucose, as well as upper GI symptoms in DGP, we hypothesized that this information can be used clinically to raise a suspicion of gastroparesis. Therefore, in patients with insulin treated diabetes and a clinical suspicion of gastroparesis we aimed to thoroughly characterize the plasma glucose response after a test meal, and to assess if this together with a questionnaire-based assessment of the severity of GI symptoms during the preceding 2 weeks could help to predict gastroparesis.

Methods

Subjects

We included patients with insulin treated DM, age 18-70 years, who complained of GI symptoms and/or had poor glycemic control, leading to a clinical suspicion of gastroparesis. An upper GI endoscopy had recently been performed in the majority of the patients as part of the clinical management as decided by the treating physician. We did not include patients who had undergone GI surgery except for appendectomy, or patients with severe psychiatric disease, sequela after cerebrovascular disease, and untreated disease with a potential impact on gastric emptying or GI symptoms. Clinical information about the patients was obtained from chart review. HbA1c values were converted to DCCT standard levels using the formula: HbA1c (DCCT) = (0.923 × HbA1c (MonoS) + 1.345; R2 = 0.998.[22] This study was approved by the Radiation Safety Committee at Sahlgrenska University Hospital, Sahlgrenska Academy, University of Gothenburg, and the Regional Ethical Review Board in Gothenburg, Sweden. All participants received verbal and written information about the study and gave written informed consent before entering the study.

Test Meal and Postprandial Glucose Response

The composition of the test meal with food in large particle size has been described in greater detail in an earlier study[23] and is in accordance with the dietary recommendation for DM subjects in Europe.[24,25] The content of the test meal was 100 g meat, 40 g pasta, 150 g carrot and 5 g oil. Pasta was chosen because it has a low glycemic index.[26,27] The meal consisted of slices of smoked pork meat, pasta boiled for 14 minutes (as per factory recommendations) and grated raw carrots with canola oil. The nutrient content was 1.57 MJ (375 kcal), 26 g protein, 13 g fat (31% of the total energy), 38 g carbohydrates, and 4.8 g of fiber (3 g/MJ) as calculated from the Database Swedish National Food Composition Tables (National Food Agency, Uppsala, Sweden).[28]

The subjects arrived at the hospital at 8 am after a 10 hours overnight fast. Intake of beverages containing alcohol was prohibited 24 hours before the test meal, and smoking was not allowed during the day of the test meal. Pharmacological agents known to affect gastric emptying were not allowed before (48 hours) and during the study. Plasma glucose samples were taken immediately before the ingestion of the meal, t = 0 minutes, and 15, 30, 45, 60, 90, 120, 150, and 180 minutes from the beginning of the meal. The samples were immediately analyzed in an automated plasma glucose analyzer by a glucose oxidase method (Merck, Darmstadt, Germany) using HemoCue NAD-NADH (HemoCue AB, Ängelholm, Sweden). The plasma glucose was not allowed to be >10 mmol/L at the beginning of the meal because of the known adverse effects of hyperglycemia on gastric emptying.[29,30] The subjects took their ordinary dose of insulin for breakfast (Unit insulin/gram carbohydrate) and were instructed to ingest the test meal within 25 minutes. Patients who had plasma glucose <4 mmol/L during the 3-hour test were given glucose supplementation (glucose tablets).

Questionnaire Assessing GI Symptom Severity

Before intake of the test meal the subjects were asked to complete the validated questionnaire, Patient Assessment of Gastrointestinal Disorders–Symptom Severity Index (PAGI-SYM), evaluating the perceived severity of GI symptoms for the preceding 2 weeks. The PAGI-SYM consists of 20 items, and these are combined into 6 subscales: nausea/vomiting, fullness/early satiety, bloating, upper abdominal pain, lower abdominal pain, and heartburn/regurgitation. The subjects were asked to rate their symptoms in a 6-point Likert-type scale ranging from 0 (*no symptoms*) to 5 (*very severe symptoms*). Evaluation studies found this questionnaire to be valid, reliable and responsive to changes in patients with upper GI disorders.[31,32] Furthermore, a subset of this scale, the 9-item Gastroparesis Cardinal Symptom Index (GCSI), consisting of the 3 subscales nausea/vomiting, fullness/early satiety and bloating, has been validated in patients with gastroparesis and found to be responsive to changes in overall symptom severity.[33] We used a total score for GCSI to reflect total upper GI symptom burden/gastroparesis-related symptoms.

Gastric Scintigraphy

On a separate day, within 2 weeks from the test meal, gastric emptying was measured with a gamma camera (MAXI II General Electric, Hermes Nuclear Diagnostic AB, Milwaukee, WI) during 3 hours after intake of a 99mTc-labeled omelet (310 kcal), and in accordance with national recommendations and national reference values for this standardized test meal. For men the normal range for retention of the radioactivity in the stomach at 120 minutes after the finished meal (R120) was 0-51%, for women < 50 years old 9-66%, and for women > 50 years old 0-55%. This method has been described in detail elsewhere.[16,17,19]

Statistical Analysis

Statistical analyses were performed using the statistical software package SPSS/PC statistics 19 (IBM, Chicago, IL). For the analyses, patients were divided into 2 groups, 1 with normal gastric emptying (DNGP) and 1 with DGP according to reference values.[17] The postprandial glucose response after a test meal was defined in different ways; besides evaluating the entire plasma glucose response curve, both the maximum increase in plasma glucose (peak glucose response) and the time to reach the peak glucose value from the beginning of the meal intake, and the incremental area under the plasma glucose curve (IAUC), which was analyzed according to the trapezium rule.[34] Median and range were compared between the groups using the Mann–Whitney *U* test. A mixed between–within subjects analysis of variance (ANOVA) was conducted to assess the effect of delayed gastric emptying, on the postprandial glucose response across the 3-hour period from the beginning of the test meal. Correlations between GI symptom severity, postprandial glucose parameters (see above) and gastric scintigraphic retention were calculated using Spearman's correlation coefficients. Receiver operating characteristic (ROC) curves were used to determine the sensitivity and specificity of upper GI symptom severity (GCSI total score) and postprandial plasma glucose parameters to differentiate between DGP and DNGP. An area under the ROC curve (AUROC) of > 0.7 is considered fair, > 0.8 good, and > 0.9 excellent discriminating ability. The best cutoff values for discriminating DGP and DNGP were determined, and positive and negative predictive values were calculated. Two-tailed *P* values < .05 were accepted as statistically significant.

Results

Subjects

We included 83 subjects with insulin treated DM (48 men, 35 women; age 58, 27-69 years (median, range). The subjects were recruited from hospitals and primary care outpatient clinics in the western region of Sweden, and referred to our unit due to clinical suspicion of gastroparesis, based on the presence of upper GI symptoms and/or poor glycemic control. Fifty-five patients had DM type 1, 23 subjects had DM type 2, 3 subjects had DM Latent Autoimmune Diabetes in the Adult (LADA) and 2 subjects had secondary DM to cortisone medication. For the analyses the subjects were grouped in 2 main groups, DM type 1 and 2, where LADA was included in DM type 1 and secondary DM in DM type 2.

The subjects with DM type 1, LADA and secondary DM were treated with short-acting and long-acting insulin. The subjects with DM type 2 were treated with short-acting and long-acting insulin or mix insulin.

Gastroparesis was confirmed in 53 (64%) subjects, 29 (55%) of them were women and 38 of them were in the DM type 1 group. Clinical characteristics of the patients are shown in Table 1, as DGP (n = 53) and DNGP (n = 30). As can be seen, DGP were younger, but otherwise no significant differences between the groups were detected. Lag phase, defined as the time from finished meal until that time when 10% was emptied from the stomach, was mean ± SD (median, range) 35.5 ± 12.7 (30, 20-55) and 68.3 ± 27.9 (60, 30-140) minutes in 62 subjects with normal and delayed gastric emptying, respectively.

Plasma Glucose Response

The subjects tolerated the test meal and were able to ingest the meal during 25.0 (19-35) (median, range) minutes. All subjects completed the entire 3-hour test. There was no significant difference in the plasma glucose at the beginning of the meal between DGP and DNGP 7.5 (4.3-10.0) (median, range) mmol/L and 8.4 (4.9-10.0) mmol/ L, respectively ($P = .23$).

During the 3 hours following meal intake, 12 DGP and 1 DNGP needed glucose supplementation due to plasma glucose < 4.0 mmol/L (range 6-36 gram of glucose). Their measured plasma glucose value at 180 minutes was 4.6 mmol/L (2.9-8.7 mmol/L) and the plasma glucose for all subjects at 180 minutes was 7.4 mmol/L (2.9-17.5 mmol/L)respectively. The plasma glucose value before the supplementation of glucose was used in the remaining measurements of the study (last data carried forward). Glucose supplementation were done at mean ± SD, median (range) 92.5 ± 38.8, 105 (30-150) minutes. They needed 14.1 ± 7.4, 15 (6-36) g glucose.

A mixed between-within subjects ANOVA was performed to assess the impact of delayed versus normal gastric emptying on the plasma glucose response during the 3-hour test (Figure 1). There was no significant interaction between gastroparesis and the plasma glucose response over time, Wilks's lambda = .84, $F(8, 74) = 1.7$, $P = .11$, partial $\eta^2 = .15$. There was a substantial effect of time on the plasma glucose response, Wilks's lambda = .42, $F(8, 74) = 12.9$, $P < .0005$, partial $\eta^2 = .58$. The main effect comparing the 2 diagnostic groups was significant, $F(1) = 8.5$, $P = .005$, partial $\eta^2 = .094$, meaning that the plasma glucose response differed between DGP and DNGP, with a blunted plasma glucose in DGP (Figure 1). In line with these findings the peak glucose response and the IAUC for the glucose curve was lower in DGP, but the time to reach the peak glucose level did not differ between the groups (Table 2). Of these parameters, only the peak glucose response

Table 1. Demographic Characteristics of Diabetic Subjects With and Without Gastroparesis.

	Subjects with gastroparesis, n = 53		Subjects with normal gastric emptying, n = 30	
	Median	Range	Median	Range
Age, year	54*	27-69	62	29-69
Weight, kg	84.3	48.7-124.5	83.8	68.0-119.5
BMI, kg/m^2	27.0	19.00-40.9	27.0	21.9-37.0
Duration of diabetes, years	25	2-55	16	2-48
Insulin treated, years	23	1-55	15	2-48
U insulin/kg body weight/day	0.6	0.2-2.2	0.6	0.2-1.7
Number of insulin injections/day, n	4	1-8	5	2-7
HbA1c, %[a]	7.4	5.7-12.0	7.5	5.4-10.1
Gastric scintigraphic retention at 120 minutes, %	74	55-91	38	17-62
Creatinine, μmol/L	73	55-142	76	47-162
GFR, mL/min/1.73 m^2	85	43-128	88	37-141

BMI, body mass index; GFR, glomerular filtration rate; HbA1c, glycosylated hemoglobin.
[a]HbA1c = DCCT standard.
*P < .05.

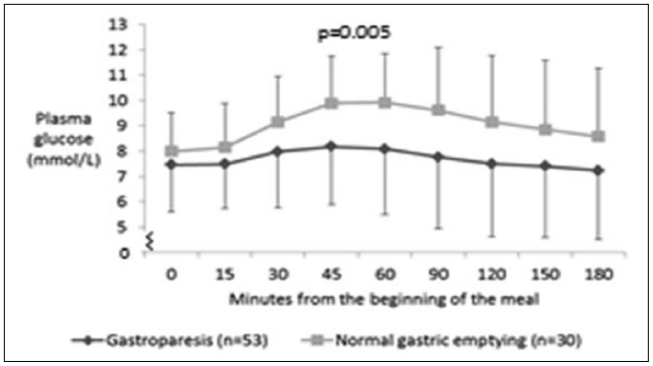

Figure 1. Postprandial glucose response (0-180 minutes) in patients with diabetes with and without gastroparesis. A mixed between-within subjects ANOVA demonstrated a significant main effect of the diagnostic group, that is, the postprandial glucose response differed between patients with and without gastroparesis, with a blunted glucose response in the gastroparetic group (P = .005).

correlated significantly, but weakly, with gastric scintigraphic retention at 2 hours (r = -0.23; P = .04).

GI Symptom Severity

DGP had more severe GI symptoms than DNGP for all the PAGI-SYM subscales (Table 2). Moreover, the GCSI total score, reflecting gastroparesis symptoms, was clearly higher in DGP than in DNGP (2.3 [0-4.8] vs 0.08 [0-3.5]; P < .0001). As can be seen in Table 3, GI symptom severity was correlated with gastric scintigraphic emptying (retention at 2 hours), and the plasma glucose response (IAUC and peak glucose response), but the association was stronger with gastric emptying. Among the different symptoms, the nonpainful upper GI symptoms demonstrated the strongest associations with gastric emptying.

Plasma Glucose Response and GI Symptom Severity to Discriminate Diabetic Patients With and Without Gastroparesis

GI symptom severity (GCSI total score) had the best discriminative validity to positively identify DGP (AUROC = 0.85), with the optimal cutoff being GCSI total score ≥ 0.8, yielding a sensitivity of 87% and a specificity of 80% (Figure 2A). The positive and negative predictive values were 88% and 77%, respectively. The plasma glucose response parameters were close to reaching fair discriminative validity to positively identify DGP, with the peak glucose response having the best AUROC value (0.66), and IAUC having AUROC = 0.64. The optimal cutoff for the peak glucose response increases to identify DGP was ≤ 1.8 mmol/L, yielding a sensitivity of 60% and a specificity of 70% (Figure 2B). Demanding a combination of GCSI total score ≥ 0.8 *and* a peak glucose increase ≤ 1.8 mmol/L resulted in a poor sensitivity (37%), but a specificity and a positive predictive value of 100%—all 20 patients, who had this combination had gastroparesis. However, the negative predictive value was only 47%. If patients were allowed to have GCSI total score ≥ 0.8 *and/or* a peak glucose increase ≤ 1.8 mmol/L, the sensitivity and specificity to identify gastroparesis were 87% and 67%, respectively.

Discussion

In this study we have demonstrated that DGP have a blunted plasma glucose response, as well as a substantial degree of GI symptoms in general and upper GI symptoms in

Table 2. Postprandial Glucose Response After Test Meal and GI Symptoms During 2 Weeks According to PAGI-SYM in Diabetic Patients With and Without Gastroparesis.

	Diabetes with gastroparesis, n = 53		Diabetes without gastroparesis, n = 30		
	Median	Range	Median	Range	*P* value
IAUC	58	0-722	196	0.6-1101	.018
Time to peak glucose level (min)	45	15-180	60	15-180	.30
Peak glucose response (mmol/L)	1.3	–2.1-5.8	2.4	–0.3-8.8	.011
PAGI-SYM: Nausea/vomiting[a]	1.00	(0.0-4.33)	0.0	(0.0-2.33)	.0001
PAGI-SYM: Fullness/early satiety[a]	2.25	(0.0-5.00)	0.25	(0.0-4.25)	.0001
PAGI-SYM: Bloating[a]	3.00	(0.0-5.00)	0.0	(0.0-4.50)	.0001
PAGI-SYM: Upper abdominal pain	2.00	(0.0-5.00)	0.0	(0.0-3.50)	.0001
PAGI-SYM: Lower abdominal pain	1.50	(0.0-5.00)	0.0	(0.0-4.00)	.0001
PAGI-SYM: Heartburn/regurgitation	1.00	(0.0-3.14)	0.0	(0.0-4.14)	.0001

IAUC, incremental area under the glucose curve; PAGI-SYM = Patient Assessment of Gastrointestinal Disorders–Symptom Severity Index.
[a]Makes up the Gastroparesis Cardinal Symptom Index (GCSI).

Table 3. Correlation Between Retention of the Isotope in the Stomach at 120 Minutes After Finished Meal, Incremental Area Under the Plasma Glucose Curve, Respectively, and GI Symptom Severity (PAGI-SYM Scores).

Diabetic subjects, n = 83	PAGI-SYM nausea/vomiting	PAGI-SYM fullness/early satiety	PAGI-SYM bloating	PAGI-SYM upper abdominal pain	PAGI-SYM lower abdominal pain	PAGI-SYM heartburn/ regurgitation
R_{120}	r = .60, P < .0001	r = .62, P < .0001	r = .63, P < .0001	r = .55, P < .0001	r = .49, P < .0001	r = .64, P < .0001
IAUC	r = –.32, P = .003	r = –.27, P = .012	r = –.36, P = .001	r = –.27, P = .015	r = –.42, P = .000	r = –.22, P = .048
Plasma glucose peak level	r = –.30, P = .006	r = –.19, P = .088	r = –.26, P = .019	r = –.20, P = .071	r = –.30, P = .007	r = –.12, P = .26

IAUC, incremental area under the glucose curve; PAGI-SYM, Patient Assessment of Gastrointestinal Disorders–Symptom Severity Index; R_{120}, retention of the isotope in the stomach at 120 minutes after finished meal.

particular. This information can be used in the clinical setting by the physician and the patient to raise the suspicion of gastroparesis. However, confirming the diagnosis gastric scintigraphy is still required.

Several studies have in different ways found an association between gastric emptying and the postprandial glucose.[9,11,20,21,23,35-40] In our study we found that the plasma glucose response was blunted in DGP, with a mean peak glucose level that was approximately 1 mmol/L less in DGP, even though there were no differences in the plasma glucose level before the meal between the DGP and DNGP. Contrary to our findings, Punkkinen et al found no significant differences in the postprandial glucose between DGP and DNGP; however in that study, there was a tendency toward group differences in the fasting glucose levels, which may have influenced the meal response.[36]

In our study 8 DGP had no increase in plasma glucose at all during the test, and 12 subjects needed glucose supplementation due to plasma glucose levels below 4 mmol/L. These subjects were not withdrawn because our results are in agreement with Lysy et al,[9] who demonstrated a significant association between hypoglycemia within 2 hours from the beginning of the meal and prolonged lag phase and gastric emptying time, which has also been confirmed in other studies.[23,37,38] Moreover, in line with some previous studies we found a blunted plasma glucose response overall in DGP.[9,20,23] The plasma glucose response pattern found in our study was a slight median increase in plasma glucose during 15-60 minutes and a decrease during 90-180 minutes from the beginning of the meal. However, the time to peak glucose response was the same in DGP and DNGP, which is in line with the study by Zeng et al.[20] In another study, by Ramzan et al, the postprandial glucose peak appeared (12 minutes) later in DGP than in DNGP.[21] This could be related to inclusion of type 2 DM only in that study, and not all of them were treated with insulin. It has been shown that in DGP the requirement of insulin is lower and not only being insulin-treated or not, but also the type of insulin, that is, short- versus long-acting insulin may affect the time to the peak glucose level. Furthermore, the balance between mealtime dose insulin and basal dose insulin, or mix of short- and long-acting insulin and number of injections influence glucose levels.[39,41,42]

The fact that we only measured plasma glucose every 15 minutes and 30 minutes during the first hour and second hour respectively, compared with continuous glucose monitoring

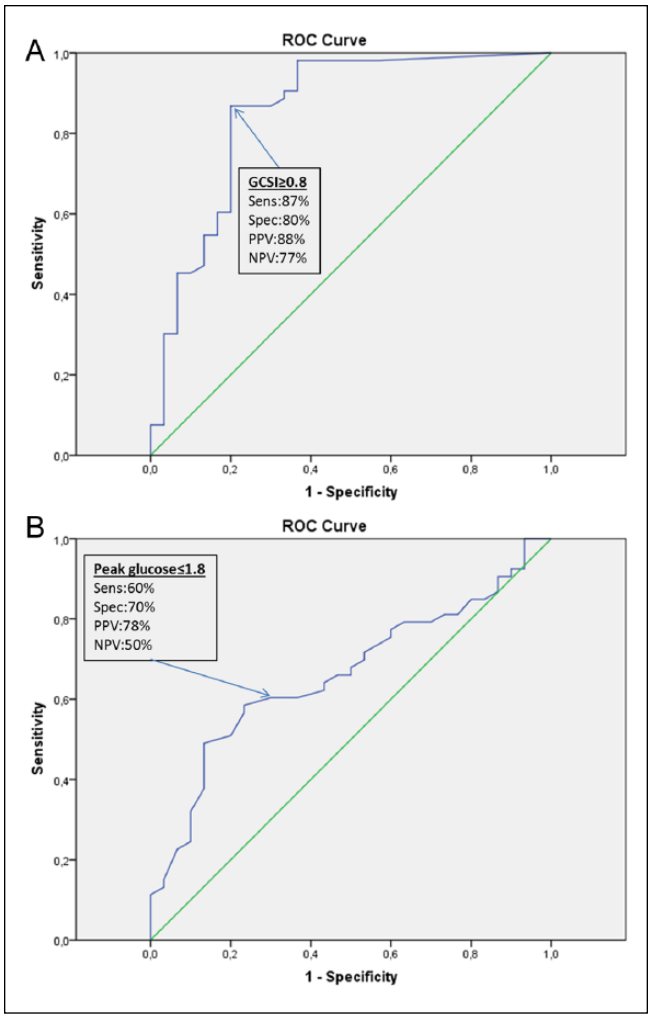

Figure 2. Receiver operating characteristic curves to assess the sensitivity and specificity of Gastroparesis Cardinal Symptom Severity Index (GCSI) total score (A) and the peak postprandial glucose response (B) in discriminating patients with diabetic gastroparesis (n = 53) from patients with diabetes and normal gastric emptying (n = 30).

(CGMS), in the study by Ramzan et al,[21] limit our possibility to detect more subtle differences in time to the peak glucose level. Also another study indicated a difference in the rise of the postprandial glucose level between DGP and DNGP, and demonstrated that the time from the start of the meal to the rise of plasma glucose > 1.0 mmol/L (blood glucose latency) seemed to be an excellent predictor of gastroparesis.[40] Taken together a blunted postprandial glucose response seems to be present in DGP, even though this has been defined and measured somewhat differently in different studies.

Regarding the possibility to predict the presence of gastroparesis in our group of patients with DM, we found fair discriminant ability of the peak postprandial glucose response, as well as of the IAUC. The optimal cutoff level for peak postprandial glucose response was ≤ 1.8 mmol/L over the baseline glucose level, with a specificity of 60% and sensitivity of 70%, making it insufficient to use this test on its own to indentify DGP. However, when combining this cutoff with a GCSI[33,43] total score ≥ 0.8, a 100% specificity was demonstrated, and all patients who had this combination actually had gastroparesis, which seem to be clinically useful information. However, the sensitivity using this combination was poor. Potentially, CGMS, which give glucose mean results every 5 minutes has the potential to improve the sensitivity and specificity,[20,21,39] even though the reproducibility seem to be an issue.[44]

Somewhat surprisingly, the best discriminant ability to predict gastroparesis was seen with upper GI symptom severity using the GCSI total score, and the optimal cutoff was found for a total score ≥ 0.8, which yielded sensitivity and specificity of 87% and 80%, respectively. Given the sometimes poor correlation between GI symptom severity and degree of gastric emptying,[2,4,45] this may seem surprising, but our group of patients was selected and included based on the clinical suspicion of gastroparesis, which may have influenced our results. However, we did not measure retention of the test meal with scintigraphy at 4 hours, which has been demonstrated to increase the detection rate of gastroparesis,[46] so potentially we may have misdiagnosed a small proportion of our patients as having normal gastric emptying.

Testing our findings prospectively in a larger unselected population seems necessary before recommending clinical use of plasma glucose response measurement in combination with questionnaire-based assessment of upper GI symptoms to identify DGP. However, the fact that this procedure is easy to administer and comparably is noninvasive makes the approach attractive for clinical decision making.

Conclusions

In the present study we have demonstrated that DGP have a blunted plasma glucose response, which may be related to an increased risk of hypoglycemic episodes and affect insulin dosing as well as the long-term glycemic control. Moreover, the plasma glucose response may be used together with questionnaire-based assessment of upper GI symptom severity as a decision-making tool, raising the suspicion of gastroparesis. However, there is still a need of investigation of gastric emptying by gastric scintigraphy to diagnose gastroparesis in patients with diabetes.

Abbreviations

DGP, patient with diabetic gastroparesis; DM, diabetes mellitus; DNGP, patient with diabetes and normal gastric emptying; GCSI, Gastroparesis Cardinal Symptom Index; GI, gastrointestinal; HbA1c, glycosylated hemoglobin; IAUC, incremental area under the plasma glucose curve; LADA, latent autoimmune diabetes in the adult; MJ, Mega Joule; PAGI-SYM, Patient Assessment of Gastrointestinal Disorders–Symptom Severity Index.

Declaration of Conflicting Interests

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