

Incretin Response In Patients With Postprandial Hyperinsulinemic Hypoglycemia Following Gastric Bypass Surgery With Or Without Extended Distal Pancreatectomy

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BACKGROUND and AIMS

Postprandial hyperinsulinemic hypoglycemia (pHH) is an increasingly recognized metabolic complication after bariatric surgery. The underlying mechanisms of pHH remain poorly understood and severe cases require extended distal pancreatectomy (DP) as a salvage therapy for recurrent, medically refractory hypoglycemia.

METHODS

Patients who had DP for pHH (DP-pHH) after Roux-en-Y gastric bypass (RYGB) were identified. An oral glucose tolerance test (OGTT) was performed and glucose, insulin, C-peptide and incretins (active GLP-1 and GIP) were measured every 30 minutes. Results were compared with OGTT performed in pHH patients without DP matched for BMI, age and sex.

RESULTS

Demographics

	DP-pHH (n=3)	pHH (n=3)
Males	2	2
Females	1	1
Mean age, Years	50 (44 - 59)	44 (33 - 50)
Mean BMI, kg/m ²	23.6 (22.1 - 28)	23.5 (23.4 - 34.9)
Mean time from RYGB to pancreatectomy, Years	7.8 (7 - 11)	-

pHH: Postprandial hyperinsulinemic hypoglycemia without pancreatectomy, DP-pHH: distal pancreatectomy for pHH. Values reported as mean (range)

Insulin and C-peptide responses to an OGTT were lower and glucose response was higher in DP-pHH compared to pHH patients. All three DP-pHH patients had elevated GLP-1 levels following the OGTT compared to pHH (median 113.9 vs 47.9 pmol/l). Also, maximal GIP level was higher in DP-pHH (median 104.2 vs 61.2 pmol/l). Time courses of glucose and measured hormones are shown in Figure 1.

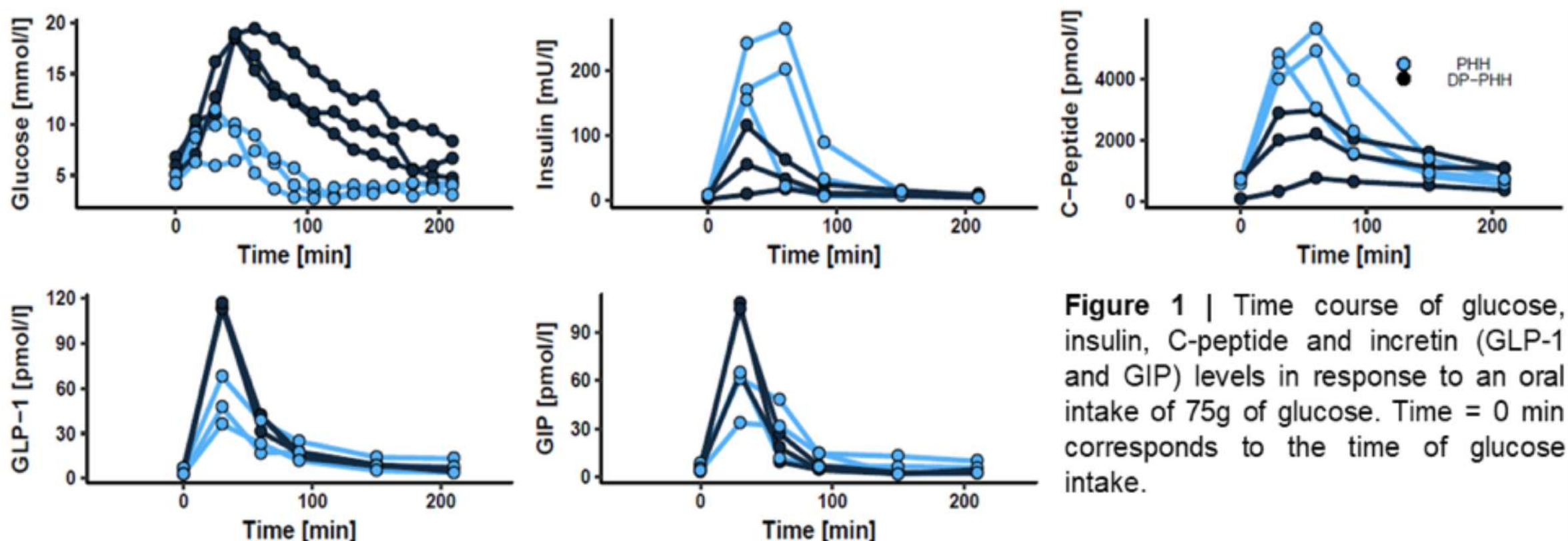


Figure 1 | Time course of glucose, insulin, C-peptide and incretin (GLP-1 and GIP) levels in response to an oral intake of 75g of glucose. Time = 0 min corresponds to the time of glucose intake.

CONCLUSIONS

We observed elevated incretin levels in DP-pHH patients compared to pHH patients with intact pancreas. These preliminary results point to a compensatory incretin response following decrease in pancreatic β -cell mass and support the important role of GLP-1 in the maintenance of residual β -cell function.