LIRAGLUTIDE IMPROVES BETA-CELL FUNCTION, MEASURED BY THE C-PEPTIDE/GLUCOSE RATIO, IN OBESE PATIENTS WITH TYPE 2 DIABETES.

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INTRODUCTION

Beta-cell function declines progressively in patients with type 2 diabetes (T2D), the fasting C-peptide/glucose ratio (Cp/G) is used for its evaluation. The GLP-1 receptor agonist liraglutide improves glucose and weight control, presumably due to improvement of beta-cell function and/or mass. This study evaluates the effect of a 6-months’ treatment with liraglutide in beta-cell function, measured by Cp/G, in patients with obesity and T2D.

PATIENTS AND METHODS

We performed an observational retrospective and prospective analysis of a cohort of 43 patients (24 women) with orally-treated T2D and obesity, to whom liraglutide 1.2 mg/day was added. At 3 months, dosage of liraglutide was increased to 1.8 mg/day in those cases in which HbA1c or weight goals were not fully achieved. We evaluated clinical and analytical data before and after 6 months of treatment. Statistical analysis with IBM SPSS Statistics Inc., version 21.0, using Cp/G as a surrogate marker of beta-cell function.

RESULTS

Patients’ basal characteristics prior to initiating liraglutide are shown in table 1. Mean T2D duration was 6.7 ± 3.8 years. Twenty six (60.5%) patients previously received one oral hypoglycemic agent (OHA) (metformin), and the rest of them were on two OHA. At 3 months, dosage was increased to 1.8 mg/day in 24 (55.8%) cases; these patients had higher pre-treatment HbA1c levels (7.8 ± 0.9 vs 7.3 ± 0.7%, p=0.025) and lower Cp/G values (0.0235 ± 0.0099 vs 0.0295 ± 0.0101, p=0.057), in comparison to those who remained on 1.2 mg/day. After 6 months of liraglutide, percentage weight loss (Δ%WL) was 5.2 ± 4.8 kg. BMI, FG, HOMA and HbA1c were significantly decreased (table 1). Mean HbA1c reduction was -0.79 ± 0.92% (which meant a reduction of 10.6 ± 11.5%). Cp/G values at 6 months increased 15.4 ± 36.6%, reaching 0.0296 ± 0.0148. Amelioration occurred regardless of pretreatment HbA1c or final dose of liraglutide. Basal Cp/G values correlated with Δ%WL and HbA1c at 6 months (figure 1). Decrease of HbA1c levels and Δ%WL were similar regardless of pretreatment HbA1c, BMI or Cp/G (figures 2-4).

Patients who were previously on only one OHA showed higher Cp/G at 6 months (0.0341 vs 0.0226, p<0.05) (figure 5). Individuals with pre-treatment Cp/G within the lower quartile achieved 6-month HbA1c levels < 7% less frequently (figure 6). Patients to whom liraglutide was increased to 1.8 mg/day achieved lower Δ%WL (7.1 ± 5.7 vs 3.6 ± 3.1, p=0.013) (figure 7), but there were no differences in the variation of HbA1c or Cp/G values.

CONCLUSIONS

Liraglutide seems to improve beta-cell function, measured by Cp/G, after 6 months, regardless of pre-treatment BMI, HbA1c or Cp/G. A lower basal pre-treatment Cp/G is associated to lower rates of optimal glucose metabolism control after 6 months of treatment with GLP-1 agonists.

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