LIXISENATIDE CLINICAL EXPERIENCE ON PATIENTS WITH TYPE 2 DIABETES AND OBESITY IN ENDOCRINOLOGY OFFICES

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BACKGROUND

Some treatments of diabetes, such as Lixisenatide, improve global metabolic status beyond glycemic control.

AIM

To evaluate tolerance to Lixisenatide and its effects on weight and metabolic control in type 2 diabetes and obese patients attended in Endocrinology offices.

MATERIAL AND METHOD

A prospective study of patients with type 2 diabetes and obesity. In an intra-subject analysis, clinical and analytical data were evaluated at baseline and after Lixisenatide treatment.

RESULTS

We studied 104 patients (51% women) with type 2 diabetes and obesity. Average age was 58.4 ± 10.5 years, average duration of diabetes was 11.2 ± 6.7 years and 35.6% and 69.6% had family history of cardiovascular disease and diabetes, respectively. At baseline, 92.2% of the patients had oral hypoglycemic agents, 13.5% GLP-1 agonists and 67.3% insulin (44.2% basal insulin, 12.5% premixed insulin and 10.6% basal-bolus insulin). We re-evaluated the patients 3.8 ± 1.6 months after treatment with Lixisenatide.

We checked that blood pressure and lipid improvement were not due to hypertension and lipid treatment intensification. We analyzed patients treated and non treated separately. These improvements were still significant in the hypertension and lipid treated subgroups (SBP, p=0.001; DBP, p=0.005; Total-Chol, p<0.001, LDL-Chol, p=0.013 and TG p=0.014), while only the decrease of SBP (p = 0.036) remained significant in the subgroup of patients without hypertension or lipid-lowering therapy.

No changes in levels of amylase related to Lixisenatide treatment were observed (57.8 \pm 19.4 IU/L).

Regarding digestive tolerance to Lixisenatide, 7.9% of patients did not tolerate, 5% tolerated 10mcg per day and 85.1% tolerated 20mcg per day.

CONCLUSIONS

Table 1: Clinical and analytical variables pre- and post-treatment.

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Variable	Baseline	Lixisenatide	P
Weight (kg)	99.5 ± 19.2	96.2 ± 18.9	<0.001
BMI (kg/m2)	37.1 ± 6.4	35.8 ± 6.2	<0.001
WC (cm)	107.6 ± 14.1	104.7 ± 14.3	0.002
SBP (mmHg)	147.0 ± 18.6	136.3 ± 19.6	<0.001
DBP (mmHg)	84.1 ± 11.2	79.5 ± 10.3	0.001
HR (bpm)	80.9 ± 9.3	80.6 ± 9.3	0.849
Fasting glucose (mg/dL)	192.8 ± 67.3	154.0 ± 44.8	<0.001
HbA1c (%)	8.7 ± 1.2	7.8 ± 1.2	0.022
Total-Chol (mg/dL)	182 ± 38.1	164.4 ± 30.0	<0.001
LDL-Chol (mg/dL)	96.3 ± 30.0	87.5 ± 33.3	0.046
HDL-Chol (mg/dL)	45.7 ± 12.0	45.7 ± 11.5	0.940
TG (mg/dL)	216.7 ± 156.2	184.4 ± 104.9	0.020
GOT (UI/L)	28.4 ± 17.0	24.9 ± 11.5	0.024
GPT (UI/L)	35.9 ± 27.9	32.1 ± 15.7	0.109
GGT (UI/L)	48.6 ± 37.2	46.7 ± 33.7	0.426
AF (mU/mL)	75.1 ± 23.7	76.7 ± 25.0	0.680
Albumin/creatinine ratio (mg/L)	15.6 ± 17.1	16.0 ± 22.8	0.876
Albumin/creatinine ratio (mg/g)	43.6 ± 140.1	33.3 ± 97.2	0.246
Insulin units	37.6 ± 36.4	35.7 ± 32.2	0.195
Hypertension drugs (%)	76	82.7	<0.001
Lipids drugs (%)	72.1	82.7	<0.001

Significant improvement of anthropometric parameters and glycemic control in terms of fasting glucose and HbA1c, and significant decrease of blood pressure and lipid profile were observed. Lixisenatide was safety and well tolerated in most patients. In addition, we found a significant intensification of antihypertensive and lipid-lowering therapy, not only hypoglycemic, in our clinical practice with an overall metabolic approach of patients.

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