INTRODUCTION

Currently, type 1 diabetes (T1DM) is defined by the autoimmune destruction of the pancreatic β cells that culminates in dependence on exogenous insulin, typically 1 to 3 years after diagnosis. This ability to maintain a residual function of pancreatic β cells is, however, heterogeneous, appearing to be worst if the disease is early diagnosed. Recently, it was demonstrated that many type 1 diabetic patients produce small amounts of insulin decades after diagnosis.

CLINICAL CASES

CASE 1

42 years-old man
T1DM (positive anti-GAD antibodies) diagnosed 18 years ago
- reasonable metabolic control
- no known complications
- insulin pump (0.6u/kg/day)
Hypertension and dyslipidemia (lisinopril and rosvastatin)
Family history of diabetes (parents and brother)

elective laparoscopic cholecystectomy

nil per os for 7 days during the postoperative period under glycosylated fluids (150gr/daily)

without exogenous insulin administration for 7 days controlled blood glucose levels without acidemia and ketonemia

CASE 2

32 years-old female
Diagnosed with diabetes at age 19.
- chronic poor metabolic control
- irregular treatment with oral antidiabetic agents (metformin, sitagliptin and gliclazide)
Family history of diabetes (mother and uncle)

referred to Endocrinology department
- poor metabolic control (HbA1c 14,8%)
  - lack of therapeutic adhesion

positive anti-GAD antibodies
(120,0 U/ml ; N<10)

measurable C-peptide
(0,39 ng/ml; N: 0,8 – 6,0)

insulin therapy / lack of therapeutic adhesion

recent admission for DKA (first known episode)

DISCUSSION

The residual insulin production, detectable by the assay of C-peptide and its functional and clinical significance have been recently discussed. According to recent evidence, these cases show us that insulin production in patients with T1DM can be kept for many years after diagnosis and that the end of the “honey-moon phase” does not necessarily lead to the absence of insulin production.

BIBLIOGRAPHY:
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