INTRODUCTION

Currently, type 1 diabetes (T1D) 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 with 18 years of duration
  - reasonable metabolic control
  - no known complications
  - Insulin pump (0,6u/kg/day)
- Hypertension and dyslipidemia (linisopril and rosuvastatin)
- Family history of diabetes (parents and brother)

**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 uncles)

- positive anti-GAD antibodies (120,0 U/mL ; N:<10)
- measurable C-peptide (0,39 ng/mL; N: 0,8 – 6,0)
- 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 type 1 diabetic patients 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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