DIABETOLOGY & METABOLIC SYNDROME

MEETING ABSTRACT Open Access

The ketosis-prone diabetes diagnosis dilemma-a case report

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Background

Ketosis-prone diabetes (KPD) comprises diabetic ketoacidosis (DKA) without phenotypic characteristics of type 1 diabetes mellitus (T1D).

Objective

Discussion of KPD diagnosis through a case presentation.

Materials and methods

Clinical report and literature review.

Results

A 16-year-old African American male was brought to emergency with abdominal pain, vomiting, lack of appetite and loss of 13kg (regular weight 152Kg, BMI 42.5kg/m^2). Evaluation detected dehydration, blood glucose of 550mg/dL, acidosis and ketonemia. There was no previous diagnosis of diabetes. He received management for DKA, with fluid therapy and regular insulin via continuous intravenous infusion (around 100-140UI/day) for 4 days. Subcutaneous insulin regimen with NPH and regular at meals was initiated after correction of acidosis. He kept capillary glycemia around 250mg/dL despite increasing doses of insulin and exclusion of other pathologies. Detectable C-peptide (1.32; 0.78-5.19ng/mL), negative glutamic acid decarboxylase antibodies (anti-GAD), along with laboratory tests done a year ago showing fasting plasma glucose of 117mg/dL and glycated hemoglobin (A1c) of 6.2%, justified starting metformin 850mg/day. Patient evolved with marked improvement of glycemic control and was discharged. He returned at the diabetes clinic after 1 month, bringing his self-monitoring showing capillary blood glucose between 60-130mg/dL and A1c of 9.3%. Insulin was gradually reduced and metformin increased to maximum dose, leading to KPD hypothesis. Patient is currently receiving outpatient treatment and waiting result of HLA assessment.

Discussion

KPD presents with classical symptoms of DKA, but majority of patients are overweight/obese and middle-aged Afro-American males. Incidence is increasing in younger patients. KPD is classified into four Aβ groups, based on the presence or absence of autoimmunity (A+; A-) and pancreatic β-cell functional reserve (β+; β-). Negative anti-GAD and high C-peptide level classify the patient described above as A-β+ subgroup, which is characterized by DKA findings with clinical features of type 2 diabetes. Forty percent of these patients achieve insulin independence and glycemic control with oral agents only. Insulin withdrawal can be longer than 10-14 weeks.

Conclusion

KPD should be investigated in young obese patients presenting with DKA, since treatment and follow up differs from T1D. Reversible β-cell dysfunction and remission to near normoglycemia is possible.

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