Letters to the Editor

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Case Report: Patient with Ketoacidosis and Impaired Insulin Secretion

Original Article: Diabetes Mellitus: Diagnosis and Screening

Issue Date: April 1, 2010

Available at: http://www.aafp.org/afp/2010/0401/p863.html

Original Article: Latent Autoimmune Diabetes in Adults [Editorial]

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TO THE EDITOR: We read with interest the article by Drs. Patel and Macerollo on diabetes mellitus, and the accompanying editorial by Dr. Unger discussing latent autoimmune diabetes in adults (LADA). LADA is one of several distinct syndromes now classified as ketosis-prone diabetes.[1] We recently cared for a patient with newly diagnosed diabetes and ketoacidosis whose clinical and laboratory profile was not typical for type 1 diabetes, type 2 diabetes, or LADA. The 37-year-old man had no notable medical history and no primary care physician when he presented to the emergency department after several days of low-grade fever, nausea, and polyuria. Laboratory evaluation revealed severe ketoacidosis (venous pH, 6.99; plasma glucose, 416 mg per dL [23.09 mmol per L]; serum bicarbonate, 6 mEq per L [6.00 mmol per L]; anion gap, 22 mEq per L; serum ketones, positive). The patient was treated with hydration and intravenous insulin, and, after resolution of the acidosis, with subcutaneous insulin.

The patient was overweight but not obese (height, 6 ft, 0 in [183 cm]; weight, 217.78 lb [98 kg], body mass index, 29.4 kg per m^2). During his hospitalization, the patient's blood pressure was below 140/80 mm Hg except for a single elevated reading on presentation to the emergency department. His mother had previously been diagnosed with diabetes. His A1C level was 15.7 percent, suggesting a period of asymptomatic hyperglycemia before his acute presentation. His C-peptide level was 0.6 ng per mL (0.20 nmol per L; normal range, 0.8 to 3.5 ng per mL [0.26 to 1.16 nmol per L]). Testing was negative for antibodies to glutamic acid decarboxylase, tyrosine kinase, insulin, and islet-cell. Blood cultures were sterile, and a urine culture grew *Staphylococcus epidermidis, Klebsiella pneumoniae,* and Group B streptococci, probably representing contamination.

The patient had unequivocal ketoacidosis and impaired insulin secretion without evidence of autoimmunity. Although chronic hyperglycemia in the setting of insulin resistance can cause beta cell exhaustion in the absence of autoimmunity,[2] common manifestations of insulin resistance such as hypertension and obesity were absent in this patient. Alternatively, he may have had an atypical presentation of LADA in which the initial insulin-independent phase did not come to clinical attention. Although glutamic acid decarboxylase (GAD-65) autoantibody positivity is a sensitive predictor of insulin dependence,[3] novel autoantibodies have also been associated with beta cell destruction.[4] This case illustrates the heterogeneous nature of ketosis-prone diabetes, and the need for more research on this subject.

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