

First Identification of Flatbush Diabetes in Patients of Indian Origin

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Ketosis-prone diabetes is an emerging heterogeneous syndrome that encompasses the biochemical, clinical, genetic, and pathophysiological features of diabetes. This allows for a more comprehensive stratification of the disease vis-à-vis the traditional type 1 diabetes and type 2 diabetes subdivision (1). Flatbush diabetes, a subset of ketosis-prone diabetes, was first described in African Americans. Following this, it has been characterized in Hispanic, Chinese, and Japanese cohorts (2,3). A thorough literature search using MEDLINE/PubMed and the Cochrane Library databases (MeSH terms: Indian, ketogenic diabetes, ketosis prone diabetes, and Flatbush diabetes mellitus) determined that Flatbush diabetes has never been described in patients of Indian origin.

We identified a 39-year-old man who was admitted with mild diabetic ketoacidosis (based on his ketone status, serum bicarbonate level, and blood pH). Biochemistry revealed a random blood glucose of 800 mg/dL, 3+ urinary ketones, a pH of 7.25, and serum bicarbonate of 13 mmol/L. No underlying trigger or infective etiology was found. He was initially managed with insulin and subsequently transitioned onto oral hypoglycemic agents. Following this, with lifestyle modification, he was able to maintain an HbA_{1c} of 6.1% (43 mmol/mol) without any oral medications. His GAD65 autoantibody titer was 2.29 IU/mL (<10.00), IA2 autoantibody titer was <0.8 units/mL (<0.8), and nonstimulated C-peptide was 1.00 ng/mL (1.1–4.4). At the 4-year follow-up, he still does well on diet alone. The patient's results were categorized according to the A β classification scheme, as this seems to be the most accurate method for the classification of atypical diabetes (1,2).

To the best of our knowledge, Flatbush diabetes has never been described in adult patients of Indian origin. Our case represents the first of these data and highlights the crucial fact that patients with atypical diabetes exist in yet another ethnic group. We feel that our patient fits the diagnostic criteria of Flatbush diabetes (A- β +), as he shows a lack of β -cell autoimmunity and preserved β-cell function. This is confirmed by the lack of antibodies and detectable C-peptide levels. Prior data have shown that testing both GAD65 autoantibodies and IA2 autoantibodies in combination gives a sensitivity of up to 98% and a specificity of 98–100% for the diagnosis of type 1 diabetes (4). C-peptide levels are documented markers of pancreatic β -cell reserve (5).

There are several limitations to our study. First of all, the case only

identifies that this subset of diabetes exists in patients of Indian origin. We have yet to follow a large cohort longitudinally and monitor clinical outcomes. Due to financial constraints, it was not possible to perform a genetic analysis to determine what genetic mutations or predisposing polymorphisms exist in our index case. Further studies are needed to delineate the true prevalence of Flatbush diabetes in the Indian population. However, one must consider this diagnosis when dealing with patients who present with ketosis and either have a history of type 2 diabetes or show remarkable improvement in their glycemic parameters following the resolution of their glucotoxicity. This will aid in transitioning these patients off insulin, as they may do well with oral therapy.

Duality of Interest. No potential conflicts of interest relevant to this article were reported. **Author Contributions.** S.J. contributed to the discussion and reviewed and edited the manuscript. S.W. and K.S. performed the literature search and helped to identify the index case. S.S. researched the data and helped to organize the necessary biochemical tests. A.B. researched data and wrote the manuscript. A.B. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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