WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 14, 935-940. Case Study ISSN 2277–7105

A CASE SERIES ON THE MANAGEMENT OF FLATBUSH DIABETES IN A 1000 BEDDED TERTIARY CARE TEACHING HOSPITAL

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Article Received on
29 May 2025,

Revised on 20 June 2025,
Accepted on 10 July 2025

DOI: 10.20959/wjpr202514-37614

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ABSTRACT

Background: Ketosis-prone diabetes or Flatbush diabetes is a widely recognized clinical entity which has become relatively common in sub-Saharan Africans, Asian and Indian populations, and Hispanic populations. Flatbush diabetes most commonly commences during middle age with no known precipitating cause. The condition is in contrast to type 1 and type 2 diabetes where the patients are insulin independent, do not have autoantibodies, and have well-functioning beta cells as evidenced by C-peptide levels. Patients initially exhibit severe hyperglycemia requiring insulin therapy along with oral hypoglycaemic agents which gets transitioned to dietary management or diet plus oral medication gradually with time. It is described as type 1.5 diabetes because patients lack auto-antibodies such as GAD, IAA, IA2 etc and have well preserved β –cell functioning. The mechanisms responsible for the development of flatbush diabetes as well its remission with time remains unknown. The study aims to emphasize the clinical presentation and importance of management of Flatbush diabetes in Indian population. **Case presentation:** A 59 year old female admitted with complaints of involuntary movements of right upper limb and lower limb with non-ketotic hyperglycemia associated with hemichorea with HbA_1c >18.5%. The patient was initially managed with IV fluids and insulin therapy followed by normoglycemia managed with lifestyle and healthy dietary management. A similar case was reported by a male patient of age 90 years hospital with the complaints of chest pain, breathing difficulty and vomiting. The patient also received basal bolus insulin therapy along with IV fluids with dose titrations in accordance to the blood glucose levels monitored. The patient was put off the insulin therapy/ antidiabetic agents following normoglycemia. The above two patients were advised to continue the same healthy lifestyle and dietary habits without the requirement of

antidiabetic agents. **Conclusion:** The above case study reports provides an insight to the clinicians to predict which patients with diabetes require temporary insulin treatment versus life-long insulin therapy.

KEYWORDS: Flatbush diabetes, ketosis, hyperglycemia, management.

BACKGROUND

Ketosis-prone type 2 diabetes is more common in male, middle-aged, overweight, or modestly obese (type 1 obesity); has a family history of type 2 diabetes; presents with new-onset severe hyperglycemia and ketosis or frank diabetic ketoacidosis; and is GAD and islet cell antibody negative. They require initial treatment with insulin and fluid and electrolyte replacement. Following several weeks to months of insulin treatment, their metabolic abnormalities improve, and they may be managed by diet alone or diet plus oral anti-diabetic agents.[1] We hypothesize this to be due to removal of a critical component of glucose or lipotoxicity at the level of the beta-cell and/or peripheral tissue.

The beta cells of patients with severe hyperglycemia with or without ketosis have lost the ability of exogenous or endogenous glucose to stimulate β-cell insulin secretion. The ability of glucose to stimulate insulin secretion begins to return after 2 weeks of normoglycemic treatment and maximizes by 8 to 12 weeks of normoglycemic treatment.[1] The prevalence of KPDM is also growing in the pediatric population with one study reporting that 17% of obese adolescents have clinical characteristics of KPDM in that they present with DKA but are able to discontinue insulin and maintain good glycemic control.[2] Ketosis-prone diabetes (KPD) comprises a group of diabetes syndromes characterized by severe beta cell dysfunction (manifested by presentation with DKA or unprovoked ketosis) and a variable clinical course. These syndromes do not fit the traditional categories of diabetes defined by the American Diabetes Association (ADA). There are four different classification schemes: the ADA system, a modified ADA system, a body mass index based system, and the Aß system (based on the presence or absence of autoantibodies and the presence or absence of beta cell functional reserve).[3] It is possible that sustained hyperglycemia per se before the development of DKA downregulates the β-cell insulin production capacity. The concept of "glucotoxicity" has been put forward to explain the contribution of toxic effects of hyperglycemia on β-cell function.[4] The goal of new classification schemes provides the clinicians a different aspect of managing special presentations of diabetes rather than the usual contemporary practice. This study aims to identify little differences in the clinical

characteristics and clinical outcomes in patients presenting with Flatbush diabetes when compared to those presenting with severe hyperglycemia with no ketoacidosis and the variability in their management strategies.

CASE PRESENTATION

Case No. 1.

We identified a 59 year old female who was admitted with complaints of involuntary movements of right upper limb and lower limb. ECHO was done and it shows sclerotic aortic valve, LV diastolic dysfunction, and tachycardia during the study. MRI of brain shows T1 hyperintensity in left lentiform nucleus, likely non-ketotic hyperglycemia associated hemichorea. CT scan of thorax revealed wall calcific plagues in aorta and in coronary arteries, collapse consolidation in right middle lobe, plate atelectasis in left lingular segment, and degenerative changes in spine. USG of abdomen showed chronic liver parenchymal disease. The biochemical values includes random blood glucose (185 mg/dl), HbA_1c (>18.5%), SGPT (121 U/L), SGOT (205 U/L), Prothrombin time (15.8 seconds), Haemoglobin (13.3 g/dl), Monocytes (6.2%), Eosinophils (6.5%). The patient was diagnosed to with Huntington's chorea, uncontrolled DM, and Systemic hypertension.

This patient received IV fluids throughout the stay in hospital. The patient received Tab. Oxcarbazepine 300mg TDS, Tab. Tetrabenzine 25mg BD, Tab. Sodium valproate 300mg TDS, Tab. Risperidone 2mg BD, Tab. Rosuvastatin plus Clopidogrel 75/10 mg OD, Tab. Cerebrolysin hydrolysate 90 mg BD, Tab. Ursodeoxycholic acid 300 mg BD, Tab. Rifaximin 400 mg BD during the stay in the hospital. The patient was initiated with a basal bolus insulin regimen plus oral hypoglycaemic agents for 8 days according to the blood glucose levels monitored at appropriate time intervals. The dose of insulin was titrated in accordance to the blood glucose levels as evidenced by routine blood glucose monitoring tests. The patient neither received insulin nor oral hypoglycaemic agents for 3 days since blood glucose levels were normal as evidenced by routine blood glucose monitoring tests. During discharge the patient had general condition and stable vital status. Following discharge, the patient was advised to monitor blood glucose regularly and manage with diet for glycemic control. After 2 weeks of follow-up, the patient turned up with a random blood sugar of 138 mg/dl, SHT of 115/120,132/109,106/153. HbA_1c was reduced to 13.6%. The patient had no hypoglycemic symptoms. On second review, the patient presented with no hypoglycemic symptoms had near normal blood glucose levels of108, 97 and 102 mg/dlmonitored using a standard blood

glucose monitor. After 1 month of follow up, the patient achieved complete remission of hyperglycemia to normal by regular monitoring of blood glucose levels with strict dietary and lifestyle habits. The patient was advised to continue the same dietary management, without the requirement of oral hypoglycemic agents/ insulin.

Case No. 2

A male patient of age 90 years was admitted in the hospital with the complaints of chest pain, breathing difficulty and vomiting. The patient had a past medication history of Coronary artery disease, myocardial infarction, left ventricular dysfunction, congestive cardiac failure and ejection fraction of 30%. The patient was not pallor, icteric, edematous, cyanotic and has no clubbing of fingers and clinically evident lymphadenopathy. The patient is neither smoker nor alcoholic. The patient was conscious, oriented with a pulse rate of 70 beats/minute, blood pressure of 140/80 mmHg. The patient had normal cardiovascular and respiratory functioning. The results of clinical investigations included Troponin I (Negative), ECG (Sinus tachycardia, LBBB), serum sodium (129mg/dl), TSH (3.3 m IU/ml), ALP (130mg/dl), haemoglobin (13.6 g/dl), HbA_1c (>18.5%). The patient had normal Chest X-ray and kidney functioning status. The patient was diagnosed with Ischemic heart disease, Left ventricular dysfunction, CCF, newly diagnosed Diabetes Mellitus. The patient was initially treated with IV fluids.The patient received Inj. Meropenem 1g IV TDS, Inj. Tramadol 50mg, Inj. Ondansetron 4mg BD, Inj. Levosulpiride 25mg BD, Tab. Tolvaptan 15mg BD, Tab. Gliclazide 500mg BD, Tab. Ivabradine 5mg BD, Tab. Atorvastatin plus Clopidogrel 20/75 mg OD during the period of stay in the hospital.

The patient received a basal bolus insulin regimen which included short acting insulin plus long acting insulin for 6 days with the titration of units in accordance with the blood glucose levels that are monitored at appropriate time intervals. The patient was put on insulin and oral hypoglycaemic agent free period for 6 days since the patient exhibited normoglycemia. During discharge, the patient had stable vitals and normal glycemic status and was advised for routine monitoring and recording of blood glucose levels at home. The patient was asked to manage his glycemic control with healthy dietary habits. The patient was followed up after 1 week and presented with no hypoglycemic symptoms. The patient was asked to continue the same dietary habits and no oral hypoglycemic agents for glycemic control.

DISCUSSION AND CONCLUSION

Our case report represents two classical cases of flat bush diabetes or ketosis prone diabetes. The presentation is rare in an endocrinology setting and is more commonly seen in African-American population rather than Indian populations. We strongly believe that the above two cases fits into the category of flat bush diabetes since the patients present with lack of insulin auto antibody and preserved ß-cell function. The diagnosis of such conditions must be considered only when patients present with either a history of type 2 diabetes or show remarkable improvement in their blood glucose parameters following the resolution of their glucotoxicity. This will aid in transitioning these patients off insulin, since they might benefit from simple lifestyle/ dietary management such as low fat/non-fat diet, high protein diet and low carbohydrate diet plan. Despite a recent history of severe hyperglycemia, patients with FBD in remission had basal and stimulated insulin secretion similar to those in healthy subjects.

List of abbreviations

GAD – Glutamic acid decarboxylase

IAA- Insulin autoantibody

IA2 – Islet antigen 2

ECG – Echocardiogram

TSH – Thyroid stimulating hormone

ALP – Alkaline phosphatase

HbA_1c – Glycated haemoglobin

KPD – Ketosis prone diabetes

DKA - Diabetic ketoacidosis

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