Case Report

When diabetes defies classification: A diagnostic crossroads between idiopathic type 1 diabetes and emerging type 5 diabetes

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ABSTRACT

Atypical forms of diabetes increasingly challenge conventional classification systems, particularly in young lean individuals presenting with insulin deficiency but lacking autoimmune markers. We report the case of a 22-year-old Indian male with poorly controlled diabetes diagnosed at 17 years of age who presented with fever, generalized weakness, burning micturition, hypotension, severe metabolic acidosis (arterial pH 6.9), ketonuria, and anemia. Immunological evaluation, including glutamic acid decarboxylase, insulin autoantibodies, insulinoma-associated antigen-2, zinc transporter 8, and islet cell antibodies, was negative. The patient was managed with intravenous fluids, insulin infusion, electrolyte correction, nutritional support, and antibiotics with gradual clinical improvement. The coexistence of profound insulin deficiency, ketosis, lean body habitus, and probable chronic undernutrition created a diagnostic dilemma between idiopathic type 1 diabetes (type 1B diabetes) and malnutrition-related diabetes mellitus, recently re-emphasized as type 5 diabetes. This report highlights the evolving spectrum of atypical diabetes phenotypes, the limitations of current classification systems, and the importance of recognizing malnutrition-associated diabetes in resource-limited settings.

Key words: Autoantibody-negative diabetes, Idiopathic type 1 diabetes, Ketosis-prone diabetes, Malnutrition-related diabetes mellitus, Type 1B diabetes, Type 5 diabetes

Diabetes mellitus is a heterogeneous metabolic disorder characterized by chronic hyperglycemia resulting from impaired insulin secretion, insulin action, or both. The conventional classification includes type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus, gestational diabetes mellitus, and specific secondary forms of diabetes [1]. However, increasing recognition of atypical diabetes phenotypes has exposed important limitations in this traditional framework. Idiopathic T1DM, also known as type 1B diabetes, represents a subgroup of insulin-dependent diabetes characterized by beta-cell dysfunction in the absence of autoimmune destruction [2,3]. Patients often present with ketosis or diabetic ketoacidosis despite negative pancreatic autoantibodies. The exact pathophysiology remains unclear, although genetic predisposition and non-autoimmune beta-cell dysfunction have been proposed [3]. In parallel, malnutrition-related diabetes mellitus (MRDM), historically described in undernourished populations of Asia and Africa, has regained scientific attention and has recently been re-emphasized as type 5 diabetes [4,5]. The condition predominantly affects young lean individuals with chronic undernutrition and impaired pancreatic beta-cell reserve. Emerging evidence suggests that chronic nutritional deprivation during childhood and adolescence may impair pancreatic development and insulin secretory capacity [6,7].

The rationale for reporting this case lies in the growing diagnostic uncertainty surrounding young lean patients with insulin-dependent diabetes but negative autoimmune markers. Such cases frequently overlap clinically with ketosis-prone diabetes, idiopathic type 1 diabetes, and type 5 diabetes, making accurate classification difficult. Furthermore, reports describing overlap between type 1B diabetes and emerging type 5 diabetes remain limited in the literature. This case highlights the diagnostic complexity and emphasizes the need for improved phenotyping and evolving classification systems for atypical diabetes in low- and middle-income countries.

CASE PRESENTATION

A 22-year-old Indian male presented to the emergency department of a tertiary care teaching hospital in Greater

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Noida with complaints of fever, generalized weakness, and burning micturition for 5 days. He was a known case of diabetes mellitus diagnosed at the age of 17 years and had a history of poor glycemic control with irregular treatment adherence. There was no significant family history of diabetes mellitus or autoimmune disease.

The patient appeared chronically undernourished and lean on examination. At presentation, he was drowsy and systemically ill. His blood pressure was 90/62 mmHg, pulse rate was 104 beats/min, respiratory rate was 28 breaths/min, and oxygen saturation was 96% on room air. Pallor was present. There was no icterus, cyanosis, clubbing, lymphadenopathy, or pedal edema. Bilateral coarse crepitations were heard on respiratory examination. Abdominal examination was soft and non-tender. Neurological examination revealed altered sensorium with neck rigidity. The sequence of major clinical events from diagnosis to discharge is summarized in Table 1.

Arterial blood gas analysis demonstrated severe metabolic acidosis with an arterial pH of 6.9 and a serum bicarbonate level of 5.9 mEq/L. The calculated anion gap was 15.2. Urinalysis revealed significant ketonuria. Random blood glucose at admission was 144 mg/dL. Hemoglobin was 6.8 g/dL, suggestive of severe anemia. Peripheral smear and biochemical evaluation were consistent with non-megaloblastic macrocytic anemia. Further evaluation for autoimmune diabetes was performed. Glutamic acid decarboxylase antibodies, insulin autoantibodies, insulinoma-associated antigen-2 antibodies, zinc transporter 8 antibodies, and islet cell antibodies were negative, suggesting a non-autoimmune etiology of insulin deficiency (Table 2). The differential diagnoses considered included idiopathic type 1 diabetes (type 1B diabetes), ketosis-prone diabetes mellitus, fibrocalculous pancreatic diabetes, and MRDM (type 5 diabetes). The presence of ketosis and severe metabolic acidosis favored profound insulin deficiency. However, the patient's lean body habitus, probable chronic undernutrition, and absence of autoimmune markers suggested overlap with malnutrition-related diabetes.

Due to the absence of pancreatic imaging abnormalities and lack of evidence of pancreatic calcification, fibrocalculous pancreatic diabetes was considered less likely. The final diagnosis was autoantibody-negative insulin-dependent diabetes with overlapping features of idiopathic type 1 diabetes and emerging type 5 diabetes.

The patient was managed aggressively in the intensive care setting with intravenous crystalloid fluid resuscitation, continuous intravenous insulin infusion, correction of electrolyte imbalance, broad-spectrum antibiotics, and supportive care. Close monitoring of blood glucose, acid-base status, urine output, and electrolytes was performed. Anemia was managed with supportive measures and nutritional optimization. Following stabilization and improvement in metabolic parameters, the patient was transitioned to a subcutaneous basal-bolus insulin regimen. Dietary counseling and nutritional rehabilitation were advised along with regular follow-up for glycemic monitoring.

Table 1: Timeline of clinical events

Time point	Clinical event
Age 17 years	Diagnosed with diabetes mellitus
Following years	Poor glycemic control with irregular treatment adherence
5 days before admission	Fever, generalized weakness, and burning micturition developed
Day 0	Hospital admission with hypotension, metabolic acidosis, and ketonuria
Day 1–3	Intensive care management with intravenous fluids, insulin infusion, electrolyte correction, antibiotics, and supportive care
Day 4 onward	Progressive clinical and biochemical improvement
Discharge	Transitioned to a basal-bolus insulin regimen with nutritional counseling

Table 2: Laboratory parameters at admission

Parameter	Value	Reference range
Random blood glucose	144 mg/dL	70–140 mg/dL
Arterial pH	6.9	7.35–7.45
Serum bicarbonate	5.9 mEq/L	22–26 mEq/L
Anion gap	15.2	8–16
Hemoglobin	6.8 g/dL	13–17 g/dL
Urine ketones	Positive	Negative
GAD65 antibodies	Negative	Negative
IA-2 antibodies	Negative	Negative
Insulin autoantibodies	Negative	Negative
ZnT8 antibodies	Negative	Negative
Islet cell antibodies	Negative	Negative

The patient demonstrated gradual clinical improvement with resolution of metabolic acidosis and stabilization of hemodynamic parameters. Sensorium improved significantly, and systemic symptoms subsided. He tolerated oral intake adequately and remained clinically stable at the time of discharge. The patient was discharged on a basal-bolus insulin regimen with advice regarding adherence, home glucose monitoring, nutritional optimization, and follow-up evaluation. Long-term follow-up and beta-cell reserve assessment could not be performed.

DISCUSSION

This case highlights the growing complexity of diabetes classification in young lean individuals presenting with severe insulin deficiency but lacking evidence of autoimmune beta-cell destruction. Distinguishing idiopathic type 1 diabetes from malnutrition-related diabetes remains clinically challenging, especially in resource-limited settings where advanced metabolic and genetic investigations are often unavailable.

Idiopathic type 1 diabetes (type 1B diabetes) is characterized by insulinopenia and susceptibility to ketoacidosis without detectable autoimmune

markers [2,3]. Unlike classical autoimmune type 1 diabetes, these patients do not demonstrate immune-mediated beta-cell destruction. Several studies suggest that type 1B diabetes may be more prevalent among Asian and African populations and may exhibit fluctuating insulin requirements [8].

MRDM, recently re-emphasized as type 5 diabetes, is increasingly recognized as a distinct entity affecting undernourished young adults in low- and middle-income countries [4,5]. Historically described in tropical countries, the condition is characterized by low body mass index, impaired insulin secretion, and chronic nutritional deprivation beginning in childhood [6,9]. Unlike type 2 diabetes, insulin resistance is not the predominant abnormality. Recent evidence suggests that pancreatic beta-cell dysfunction secondary to undernutrition and impaired pancreatic development plays a central role in disease pathogenesis [7,10].

The International Diabetes Federation's recognition of type 5 diabetes in 2025 renewed global interest in this neglected entity [11]. Emerging literature suggests that millions of individuals worldwide may remain undiagnosed or misclassified due to overlap with type 1 and type 2 diabetes phenotypes [11,12].

The present patient demonstrated features overlapping both type 1B diabetes and type 5 diabetes. Severe metabolic acidosis and ketonuria indicated marked insulin deficiency and predisposition to ketosis, findings more commonly associated with idiopathic type 1 diabetes [3,8]. However, the lean phenotype, probable chronic undernutrition, young age at onset, and absence of pancreatic autoantibodies supported possible malnutrition-related diabetes [4,5]. The distinguishing clinical characteristics of idiopathic type 1 diabetes and type 5 diabetes are summarized in Table 3 [13-15].

Similar diagnostic dilemmas have been described in previous studies involving lean young individuals with insulin-dependent diabetes lacking autoimmune markers.

Table 3: Comparison between idiopathic type 1 diabetes and type 5 diabetes

Feature	Idiopathic type 1 diabetes (type 1B)	Type 5 diabetes
Autoimmune markers	Absent	Absent
Age of onset	Young	Young
Body habitus	Lean or normal	Lean
Insulin deficiency	Severe	Moderate to severe
Ketoacidosis	Common	Rare
Nutritional association	Usually absent	Strong association
Beta-cell dysfunction	Presentw	Present
Insulin resistance	Minimal	Minimal
Long-term insulin requirement	Usually required	Variable
Geographic predominance	Worldwide	Low- and middle-income countries

Misra *et al*. described nutritionally compromised patients requiring insulin therapy despite the absence of classical autoimmune features [9]. Ramachandran *et al*. similarly documented malnutrition-related diabetes in South Indian patients presenting with low body mass index and impaired insulin secretion [16]. Earlier World Health Organization reports from tropical countries also recognized atypical diabetes phenotypes associated with chronic undernutrition and pancreatic dysfunction [15]. Taneera *et al*. emphasized reduced beta-cell reserve rather than insulin resistance as the dominant pathogenic mechanism in malnutrition-related diabetes [10]. Rangraze *et al*. further highlighted the growing public health relevance of type 5 diabetes in low- and middle-income countries [12]. These findings collectively support the possibility that chronic undernutrition contributes significantly to atypical insulin-dependent diabetes phenotypes.

Ketosis-prone diabetes was another important differential diagnosis in this patient. Balasubramanyam *et al*. and Maldonado *et al*. described ketosis-prone diabetes as a heterogeneous syndrome characterized by episodic ketoacidosis with variable long-term insulin dependence [8,14]. However, unlike classical ketosis-prone diabetes, the present patient had chronic undernutrition, young age at onset, and persistent insulin requirement, favoring overlap with type 1B diabetes or type 5 diabetes rather than transient beta-cell dysfunction alone.

The broader challenge of diabetes classification has also been emphasized in contemporary literature [17-19]. Leslie *et al*. highlighted that conventional diabetes categories frequently fail to account for overlapping metabolic phenotypes encountered in clinical practice [19]. Pozzilli and Pieralice discussed the diagnostic uncertainty that may arise in antibody-negative insulin-dependent diabetes states [18]. Furthermore, epidemiological studies from India continue to demonstrate the coexistence of undernutrition and diabetes within vulnerable populations [17]. These observations reinforce the need for revised classification systems incorporating nutritional status, beta-cell reserve, and metabolic heterogeneity. Global epidemiological transitions in diabetes further complicate accurate phenotyping in developing countries [20].

This case also demonstrates the limitations of current diagnostic approaches. Measurement of fasting and stimulated C-peptide levels could have provided additional insight regarding residual beta-cell reserve. Detailed anthropometric assessment, nutritional profiling, pancreatic imaging, and long-term follow-up would have further strengthened classification.

The strength of this report lies in highlighting an evolving and under-recognized clinical entity from a resource-limited setting. The case emphasizes the need for clinicians to consider malnutrition-related diabetes in young lean individuals presenting with atypical insulin-dependent diabetes. Increased awareness may improve

early recognition, appropriate insulin therapy, nutritional rehabilitation, and future epidemiological research.

CONCLUSION

This case underscores the diagnostic challenge of classifying autoantibody-negative diabetes in young lean individuals presenting with metabolic crises. The coexistence of ketosis, severe insulin deficiency, chronic undernutrition, and negative autoimmune markers created a diagnostic overlap between idiopathic type 1 diabetes and emerging type 5 diabetes.

Recognition of atypical diabetes phenotypes is particularly important in low- and middle-income countries where malnutrition remains prevalent. Current diabetes classification systems may not adequately capture such overlapping presentations. Further studies incorporating beta-cell reserve assessment, nutritional evaluation, genetics, and longitudinal follow-up are necessary to refine diagnostic criteria and improve understanding of type 5 diabetes.

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