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HYBRID DIABETES MELLITUS – A CASE REPORT

General Medicine

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ABSTRACT

Diabetes mellitus represents a group of common metabolic disorders unified by the presence of hyperglycemia. According to the WHO 2019 revised classification, diabetes is categorized into six major groups: Type 1 diabetes (insulin-dependent), Type 2 diabetes (insulin-resistant), Hybrid forms of diabetes such as Latent Autoimmune Diabetes in Adults (LADA), other specific types including monogenic and pancreatic diseases, unclassified types, and hyperglycemia first detected during pregnancy.

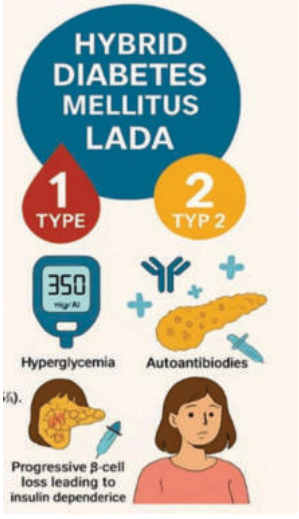
KEYWORDS

Hybrid Diabetes Mellitus, LADA, autoimmune Diabetes.

INTRODUCTION

Hybrid forms like LADA are slowly progressive autoimmune diabetes occurring in adults, initially resembling Type 2 diabetes clinically but ultimately evolving into insulin dependence due to autoimmune β-cell destruction. LADA should be suspected in patients diagnosed as Type 2 diabetes who present with normal weight, a personal or family history of autoimmune diseases, or unexplained deterioration of glycemic control despite oral agents and lifestyle measures.

Clinical factors suggesting LADA include age <50 years, BMI <25 kg/m², symptoms of hyperglycemia, presence of autoimmune diseases, and a family history of autoimmunity. The most reliable autoantibodies used for diagnosis are GAD-65, ICA, IA-2A, ZnT8A, and islet cell autoantibodies. Patients with suspected LADA should undergo GAD-65 antibody testing, and if mild risk features are present, C-peptide levels assist in classification. Sustained hyperglycemia and the need for insulin for at least six months after diagnosis are typical. These features collectively help differentiate LADA from classical Type 1 or Type 2 diabetes and guide long-term management. surrounding matrix, and that may result in higher compressive strength.



Sources: www.googleimages .com/ LADA
Figure 1: Hybrid Diabetes Mellitus LADA

CASE STUDY

A 24-year-old female with a two-year history of diabetes mellitus on regular premixed insulin presented with tiredness, continuous vomiting, and abdominal pain. On examination, she was conscious, with a pulse of 116 bpm, blood pressure 100/60 mmHg, respiratory rate 26/min, and oxygen saturation 92–98% on room air. Abdominal tenderness was present. Investigations showed blood glucose of 350 mg/dL, urine ketones positive, and ABG consistent with acute metabolic acidosis. Laboratory results revealed Hb 13.9 g/dL, total leukocyte count 15,320, platelets 3.05 lakh, blood urea 23 mg/dL, creatinine 0.6 mg/dL, bilirubin 0.8 mg/dL, SGPT 20 IU, SGOT 38 IU, sodium 136 mEq/L, and potassium 5.8 mEq/L.

She had two prior admissions for diabetic ketoacidosis and had been formally diagnosed with diabetes 20 months earlier. She also had Hashimoto's thyroiditis since 2020, with ultrasonography showing coarse echotexture and hypoechoic nodules. Fine-needle aspiration revealed features consistent with thyroiditis. She was not on treatment for thyroid disease. Due to poor glycemic control, further evaluation showed markedly elevated GAD-65 antibodies (85.8 IU/mL), anti-thyroperoxidase antibodies (>1300 IU/mL), and anti-thyroglobulin antibodies (>500 IU/mL). Her HbA1c remained consistently high at 14.5%. There was no family history of diabetes or autoimmune disorders. She was diagnosed with diabetic ketoacidosis and managed according to standard protocols.

DISCUSSION

LADA typically presents after the age of 30, but younger patients can also be affected, particularly those displaying autoimmune features and early insulin requirement. Phenotypically resembling Type 1 diabetes, LADA patients often have normal BMI, rapid loss of glycemic control, and a significant reduction in β-cell reserve despite oral antidiabetic therapy. The presented case demonstrates classic features of LADA, including young onset, low BMI, coexistence of autoimmune thyroid disease, persistent hyperglycemia, and markedly elevated autoantibodies (GAD-65, anti-TPO, anti-TG). Her multiple admissions with DKA highlight the progressive β-cell decline commonly seen in autoimmune diabetes.

CONCLUSION

Management should prioritize early initiation of insulin to preserve remaining β-cell function and prevent recurrent ketoacidosis. Oral agents such as DPP-4 inhibitors and GLP-1 receptor agonists show potential benefits in slowing β-cell loss, although evidence is evolving. Recognizing LADA early is essential to avoid misclassification as Type 2 diabetes, which often leads to delayed insulin therapy, worsening hyperglycemia, and repeated metabolic decompensation.

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