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Suresh Kishanrao *

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Case Report

Managing Latent Autoimmune Diabetes in Adults (LADA] in Smaller Settings

Suresh Kishanrao

Suresh Kishanrao, Family Physician & Public Health Consultant, Bengaluru.

***Corresponding Author:** Suresh Kishanrao, Family Physician & Public Health Consultant, Bengaluru.

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Abstract

Diabetes mellitus (DM) is a disease spectrum ranging from classic insulin deficiency of type 1 diabetes (T1DM) to insulin-resistant type 2 diabetes (T2DM). Latent autoimmune diabetes in adults (LADA) is a type of DM with clinical characteristics of both T1DM and T2DM, necessitating the creation of a new subtype of disease, type 1.5. Most of LADA patients are males aged above 30-35 years with a mean body mass index of around 22.5-23.0 kg/m², have uncontrolled blood sugar and Hb1Ac and both are not amenable to oral hypoglycaemic drugs, and everyone has positive anti-glutamic acid decarboxylase (GAD) antibody. Complications seen among these patients are diabetic Ketoacidosis. Other complication like mild non-proliferative diabetic retinopathy, peripheral neuropathy and combined moderate non-proliferative diabetic retinopathy and peripheral neuropathy are in the ratio of 4:2:1 respectively. LADA remains a diagnostic challenge, as its phenotypic picture resembles T2DM, while immunologically it resembles T1DM.

Materials and Methods: This article is based managing a few cases of LADA in 2025, quoting 2 atypical cases managed as out-patient and inpatient respectively. This is intended to guide primary care physicians to handle diabetic cases who respond initially for about 6 months with oral anti-diabetic drugs like Sulfonylureas and suddenly start deteriorating as indicated high Fasting Blood sugar and Hb1Ac.

Outcomes: Both the anecdotal cases are doing well after initiating Insulin therapy.

Keywords: diabetes mellitus (dm); type 1 diabetes (t1dm); type 2 diabetes (t2dm); latent autoimmune diabetes in adults (lada); fasting blood sugar (fbs); random blood sugar (rbs); glycated or glycosylated haemoglobin (haemoglobin a1c, or hb1ac); Micro & macro vascular complications; diabetic ketoacidosis (dka); diabetic retinopathy; nephropathy and neuropathy

Introduction

Diabetes mellitus (DM) is a disease spectrum ranging from classic insulin deficiency of type 1 diabetes (T1DM) to insulin-resistant type 2 diabetes (T2DM). Latent autoimmune diabetes in adults (LADA) is a type of DM with clinical characteristics of both T1DM and T2DM. Despite its clinical significance, LADA remains underdiagnosed and mismanaged due to overlapping characteristics with other diabetes types [1,3]. These patients have uncontrolled diabetes not controlled with oral hypoglycaemic drugs, mostly males aged above 30 years with a mean body mass index of 22.6 kg/m², and everyone had positive anti-glutamic acid decarboxylase (GAD) antibody. Complications seen among these patients are mild non-proliferative diabetic retinopathy, peripheral neuropathy and combined moderate non-proliferative diabetic retinopathy and peripheral neuropathy in the ratio of 4:2:1 respectively. LADA remains a diagnostic challenge, as its phenotypic picture resembles T2DM, while immunologically it resembles T1DM. Research on LADA in India indicates a high prevalence among diabetes patients initially diagnosed with type 2, often misdiagnosed due to adult-onset over age 30. Studies highlight that GAD65-positive Indian patients have higher HbA1c, & fasting blood sugar levels compared to GAD65-negative, suggesting the need for antibody testing. Key epidemiological factors of LADA in India include i) Prevalence of LADA is about 52% among certain cohorts. It is frequently misdiagnosed as Type 2 diabetes because patients often do not require insulin for at least the first 6 months after diagnosis iii) However, the presence of antibodies, GAD65 (glutamic acid decarboxylase), is critical for diagnosis, iv) and GAD65-positive patients in India often present with higher HbA1c, indicating faster progression to insulin dependence [3]. Clinically patients exhibit a phenotype that straddles Type 1 and Type 2, with slower destruction of islet beta cells compared to typical Type 1 diabetes. The condition often remains underdiagnosed in India, requiring a higher index of suspicion to prevent early complications. Key Complications of LADA include i) Microvascular Complications like High prevalence of retinopathy, nephropathy and neuropathy due to long-term hyperglycaemia ii) macrovascular diseases lead to cardiovascular disease, stroke, and atherosclerosis iii) As LADA involves a progressive loss of insulin-producing cells, patients are at risk of Diabetic Ketoacidosis (DKA), a life-threatening condition, especially if not treated with insulin early iv) LADA patients have a higher prevalence of

accompanying autoimmune thyroid diseases [1,4] If misdiagnosed as Type 2 and treated with sulfonylureas, beta-cell function can decline faster, leading to quicker insulin dependency, diabetic ketoacidosis a life-threatening condition, especially if not treated with insulin early. This article is based managing a few cases of LADA in 2025 and intended to guide primary care physicians to handle such cases.

Case Reports:

Case Study 1: LADA with Severe Complications: A 35-year-old male with severe weight loss (BMI 15) and peripheral neuropathy reported my clinic in January 2025. The patient's HbA1c of 16%, highlighting the severe catabolic state. His main complaints were wasting over last 2 years and being on oral anti-diabetic drugs for T2D since last 2 years. For the first 6 months his fasting blood sugar and HbA1c seemed reducing and under control with Tablet Amaryl 3 mg before meals and Tab Metformin 1000mg after each main meal. In the last 18 months his weight reduced from 70kg to 54 kgs. In November 2025 Tablet Gibtulio 25 mg was added before each meal. Suspecting LADA I got done an antibody test to differentiates Type 1 from Type 2 diabetes by detecting immune-mediated destruction of pancreatic beta cells. The sample was collected from home, and the results were available in3 days, which turned positive for GAD65. HIs glutamic acid decarboxylase antibody values 0.05 nmol/L whereas values in patients who have type 1 diabetes without a polyendocrine or autoimmune neurologic syndrome are usually 0.02 nmol/L or below. He was put on Insulin and in last 3 months has gained about 5 kg weight and Hb1Ac is around 9% and FBS around 150mg/Dl. In January 2026 his Hb1 Ac is 7% and FBS around 110. He has recovered weight up to 65 Kg.

Case Study 2: Diabetic Ketoacidosis (DKA): In July 2025 documented a 36-year-old male diagnosed Type 2 diabetes and on oral anti-diabetics presented symptoms of extreme thirst, frequent urination, abdominal pain, nausea/vomiting and deep/rapid breathing. On examination he had fruity-smelling breath, partially confused. His laboratory test indicated Random blood sugar was 400 mg/dL and ketones in urine. His attendant gave an history of a wound in the thigh with (thigh abscess) resulting from an injury after a fall from a two - wheeler getting infected. He had high anti-GAD65 antibodies and low fasting C-peptide (0.14 ng/mL). His DKA was managed in a private hospital following standard protocol of:

i) Fluid Resuscitation: Beginning with Normal Sodium Chloride to restore volume, followed by fluids containing 5-10% dextrose to avoid hypoglycaemia.

ii) Insulin Therapy: Start intravenous insulin at 5-10 U/hour, maintaining infusion until DKA resolves (pH calculated anion gap normal).

iii) He was put on appropriate IV antibiotic to address thigh abscess and switched to oral antibiotics after 2 days before discharge

iv) Monitored Potassium and replaced it potassium throughout, as insulin drives it into cells, adding 30 mEq K in each liter of IV fluid.

v) Measured Blood Glucose hourly, and electrolytes/pH every 2-4 hours.

vi) He was Transitioned to Subcutaneous Insulin, after the patient started eating and DKA resolved. subcutaneous insulin was initiated 2 hours before stopping the IV drip to prevent rebound ketosis.

He revered over 36 hours and was discharged after 3 days on Subcutaneous Insulin.

Discussions:

Latent autoimmune diabetes in adults (LADA) was first described by Zimmet as a subcategory of adult patients with phenotypic type 2 diabetes (T2DM) with autoantibodies against glutamic acid decarboxylase 65 (GAD 65). LADA Patients fail sulfonylurea treatment and require insulin therapy earlier in the course of the disease. In 1974, the adults were found to have the same autoantibodies against pancreatic islet cells as those diagnosed with type 1 diabetes (T1DM), necessitating the creation of a new subtype of disease, type 1.5. LADA causes chronic hyperglycaemia leading to microvascular damage. The retinal blood vessels are a common target for the deleterious effects of hyperglycaemia leading to diabetic retinopathy. The Immunology of Diabetes Society has defined strict criteria to classify LADA including i) An age of onset of at least 30 years of age ii) Lack of dependence on insulin within the first six months after diagnosis iii) Seropositivity of at least one related autoantibody. The most common autoantibodies implicated in both T1DM and LADA are GAD65 and islet-cell antibodies (ICA). It has been demonstrated that LADA shares genetic features of both T1D and T2D. The autoimmune inflammation induced by autoantibodies promotes destruction of the beta cells in the pancreas; therefore, eliminating their ability to create insulin. Without exogenous insulin therapy, these patients will enter a state of ketoacidosis resulting in serious complications including seizure, coma, and even death.[1]

Aetiology: LADA accounts for 2-12% of all cases of diabetes. The typical LADA patient is nonobese, older than 35 years, with diabetes initially controlled with lifestyle modifications that eventually progresses to the point of full insulin dependence. A multicentre study in Europe, Asia, and North America demonstrated that 4% to 14% of patients diagnosed with T2D were positive for diabetes-related autoantibodies [2]. A look at risk factor characteristics suggest that LADA is a hybrid of T1DM and T2DM. The interaction of physiologic stressors exacerbates the ongoing autoimmune processes in LADA patients, for disease onset [1]

Pathophysiology: As LADA shares characteristics of both T1DM and T2DM therefore there are two separate pathophysiologic processes at work. In patients who are genetically susceptible to developing T1DM, certain immunological factors can spark an autoimmune process causing islet cell destruction in the pancreas by autoantibodies. This leads to insulin deficiency as beta cells go through apoptosis. In contrast, obese individuals who share genetic susceptibility to T2DM develop low grade inflammation from visceral adiposity which trigger an autoimmune process specifically marked by IA-2 antibody positivity, causing beta cell dysfunction and decreased insulin production as well.

LADA in India: In India, LADA commonly presents in adults over 30 (often 30–40 years) with a lean-to normal BMI, initially diagnosed with Type 2 Diabetes (T2D) but experiencing rapid, unexpected failure of oral hypoglycaemic agents within 6-12 months, leading to insulin dependence. Key presentations include hyperglycaemic symptoms of polyuria, polydipsia, and weight loss despite treatment, often with higher C-peptide levels than Type 1 but lower than T2D, and positive GAD antibodies test. LADA often remains underdiagnosed or misdiagnosed as T2DM due to a lack of universally accepted diagnostic criteria, leading to a delay in initiating appropriate therapy, prolonged exposure to the deleterious effects of hyperglycaemia, and unwarranted exposure to ineffective oral hypoglycaemic agents. There is limited literature on the clinical spectrum of LADA in Indian adults. Some cases present as diabetic ketoacidosis (DKA) [3]. Managing diabetic ketoacidosis (DKA) is done in an intensive care unit during the first 24-48 hours always is advisable. When treating patients with DKA, the following points must be adhered. i) Correction of fluid loss with intravenous fluids ii) Correction of hyperglycaemia with insulin iii) Correction of electrolyte disturbances, particularly potassium loss iv)

Correction of acid-base balance v) Treatment of concurrent infection, if present as was done in our second case. Monitoring cerebrovascular accident, myocardial infarction, sepsis, or deep venous thrombosis and infections are equally important [7]. It is important to pay close attention to the correction of fluid and electrolyte loss during the first hour of treatment, followed by gradual correction of hyperglycaemia and acidosis. Correction of fluid loss makes the clinical picture clearer and may be sufficient to correct acidosis. The presence of even mild signs of dehydration indicates that at least 3 L of fluid has already been lost. Patients are discharged from the hospital only when they have been able to switch back to their daily insulin regimen without a recurrence of ketosis. When the condition is stable, pH exceeds 7.3, and bicarbonate is greater than 18 mEq/L, the patient is allowed to eat a meal preceded by a subcutaneous (SC) dose of regular insulin [7].

Management of LADA: There is no unique approach to treating diabetic complications like optic neuropathy, retinopathy associated with LADA. Treatment for diabetic retinopathy includes use of focal lasers and injection of drugs such as anti-VEGF therapies to reduce retinal swelling and oedema. In proliferative diabetic retinopathy, primary treatment is pan- retinal photocoagulation or anti-VEGF injection therapy as alternative treatment. Diabetic Ketoacidosis treatment follows standard protocol [7].

Medical therapy: The goal of medical therapy is to preserve beta-cell (B-cell) function, and maintain strict glycaemic control, through a personalized approach to therapeutics as LADA can present with different metabolic phenotypes, particularly the pre-existing level of insulin secretion. Patients with extremely low C-peptide (<0.3 nmol/L), a measure of insulin secretion, are considered equivalent to T1D and should follow insulin-based therapies. For patients with higher C-peptide levels, insulin-based therapy in combination with other hypoglycaemic agents like metformin, glucagon-like peptide-1 receptor agonists (GLP-1RAs), dipeptidyl peptidase IV (DPP4) inhibitors are recommended. Sulfonylureas have no place in treatment of LADA due to their effect of deteriorating B-cell function and maintaining poor glycaemic control. Sodium-glucose cotransporter-2 inhibitors (SGLT2) have not been studied in LADA yet to define their benefits. Lifestyle modifications such as weight loss and physical exercise, have no clear indication of improving outcomes in LADA.

Conclusion:

Latent autoimmune diabetes in adults (LADA) was first described by Zimmet as a subcategory of adult patients with phenotypic type 2 diabetes (T2DM) with autoantibodies against glutamic acid decarboxylase 65 (GAD 65). LADA Patients fail sulfonylurea treatment after about 6 months and require insulin therapy earlier in the course of the disease. LADA accounts for 2-12% of all cases of diabetes. The typical LADA patient is nonobese, older than 35 years, with diabetes initially controlled with lifestyle modifications and Sulfonylureas that eventually progresses to the point of full insulin dependence. There is no specific approach to treating diabetic complications like diabetic ketoacidosis, optic neuropathy, retinopathy associated with LADA. Patients with extremely low C-peptide (<0.3 nmol/L), a measure of insulin secretion, are considered to follow insulin-based therapies. For patients with higher C-peptide levels, insulin-based therapy in combination with other hypoglycaemic agents like metformin, glucagon-like peptide-1 receptor agonists (GLP-1RAs), dipeptidyl peptidase IV (DPP4) inhibitors are recommended. Sulfonylureas have no place in treatment of LADA.

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