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Latent autoimmune diabetes in an adult male presenting with diabetic ketoacidosis precipitated by sepsis

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Abstract
Latent autoimmune diabetes in adults (LADA) is a relatively common form of diabetes, but it remains under-researched. It shares characteristics of both type 1 and type 2 diabetes mellitus (T1D and T2D). While autoimmunity is a recognised underlying cause of LADA, certain environmental factors such as obesity, lack of physical activity, and smoking may also contribute to its development. Due to its overlapping features with T2D, LADA is often misdiagnosed as type 2 diabetes and initially managed with oral hypoglycaemic agents, which can delay the timely initiation of insulin therapy. Although rare, there are reports of patients with LADA presenting for the first time with diabetic ketoacidosis (DKA). We describe here a case of an adult male with latent autoimmune diabetes and sepsis who presented with DKA at onset.

Keywords: Latent autoimmune diabetes in adults, type 1 diabetes, type 2 diabetes, diabetic ketoacidosis, sepsis

Introduction
Diabetes mellitus (DM) is a group of metabolic diseases characterised by hyperglycaemia arising from defects in insulin secretion, insulin action, or both. It remains a leading non-communicable disease worldwide. The World Health Organization estimates that adult diabetes prevalence will double between 2000 and 2025.

Diabetes is an umbrella term for disorders that share the hallmark of persistent hyperglycaemia. Latent autoimmune diabetes in adults (LADA) sits between type 1 and type 2: it carries auto-antibodies like the former yet often masquerades as the latter. Global figures suggest the burden of diabetes will double in a quarter-century, and up to ten per cent of adults first labelled with type 2 eventually prove to have LADA. Progressive β-cell loss is usually slow, but stressors such as infection can tip the balance, triggering diabetic ketoacidosis.

Latent autoimmune diabetes in adults (LADA) is a slowly progressive form of autoimmune diabetes mellitus identified by pancreatic auto-antibodies and an initial lack of absolute insulin dependence. While early β-cell function is relatively preserved, LADA demonstrates a rapid decline that ultimately necessitates intensive insulin therapy.

Diabetic ketoacidosis (DKA) is a decompensated metabolic state characterised by hyperglycaemia, ketosis and acidosis. Serum glucose >250 mg/dL, arterial pH <7.3 and bicarbonate <18 mEq/L with elevated serum ketones define the condition. Absolute or relative insulin deficiency-often precipitated by infection-remains the central aetiology.

Case Presentation
A 36-year-old male presented on 19 June 2025 to MGM Hospital & Research Centre, Vashi, with a five-day history of fever, malaise and a rapidly enlarging painful swelling over the left upper thigh. He denied previous insulin therapy but reported sporadic metformin use for hyperglycaemia.

Vital signs revealed temperature 38.9 °C, pulse 104 bpm, blood pressure 150/90 mmHg and moderate dehydration. The left lateral thigh harboured a 7×6 cm fluctuant abscess.

Point-of-care glucose was 472 mg/dL; arterial blood gas demonstrated pH 7.09, bicarbonate 9 mEq/L and an anion gap of 31 mEq/L. Serum β-hydroxybutyrate measured 5.2 mmol/L, and urine dipsticks showed ketones 4+, confirming DKA.

Management commenced with 0.9 % saline boluses, potassium supplementation and an intravenous insulin infusion. Empirical piperacillin-tazobactam was started. Surgical incision and drainage released 80 mL purulent material growing methicillin-sensitive Staphylococcus aureus. Anti-GAD65 antibodies were positive and fasting C-peptide was 0.18 ng/mL, consistent with LADA.

The ketoacidosis resolved over 48 h; he transitioned to a basal-bolus insulin regimen (glargine 22 U nightly plus aspart with meals) and was discharged on hospital day 5 with endocrinology follow-up.

Clinical and Biochemical Findings

Parameter	Finding
Temperature	38.9 °C
Pulse	104 bpm
Blood Pressure	150/90 mmHg
SpO_2	96 % (Room air)
Capillary Glucose	472 mg/dL
β-Hydroxybutyrate	5.2 mmol/L
Urine Ketones	4+ (positive)
pH / HCO_3^-	7.09 / 9 mEq/L
Anion Gap	31 mEq/L
HbA1c	11.3 %
CRP	> 90 mg/L
Potassium	2.9 mEq/L
Chloride	90 mEq/L
Creatinine	1.2 mg/dL

Discussion

This case highlights how sepsis can unmask or precipitate severe insulinopenia in LADA, resulting in DKA. Early recognition, rapid source control and continuous insulin are critical to reduce morbidity. Identification of autoimmune aetiology mandates lifelong insulin and careful monitoring for associated autoimmune disease.

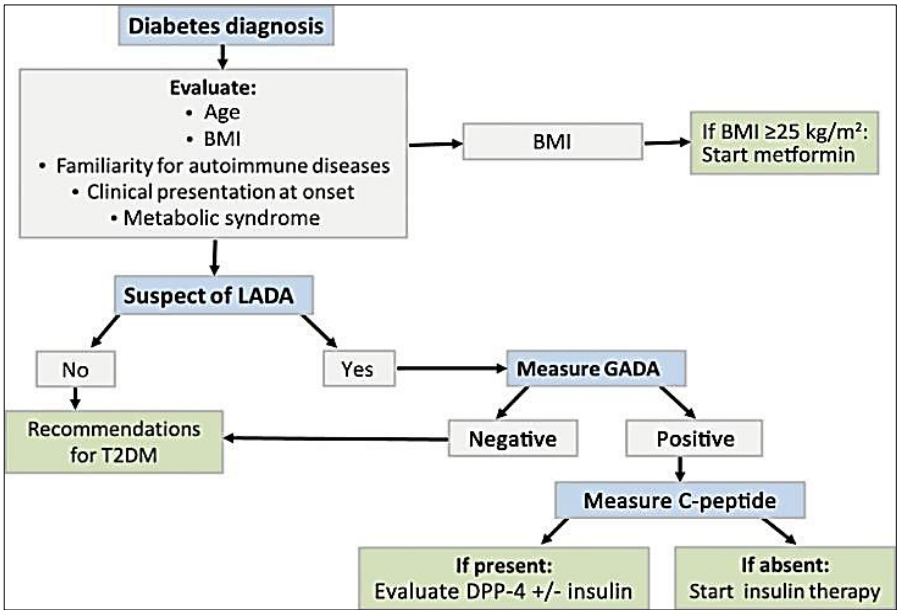
LADA combines autoimmune β-cell destruction with insulin resistance. Approximately 10 % of adults over 35 years phenotypically diagnosed with type 2 diabetes harbour islet antibodies. Progressive β-cell failure is most rapid in those with multiple antibodies, yet even single-antibody positivity predicts insulin requirement within five years.

Acute infection drives counter-regulatory hormone surges (glucagon, catecholamines, cortisol, growth hormone) that enhance lipolysis, ketogenesis and hepatic glucose output-key mechanisms precipitating DKA in LADA. In our case, a deep thigh abscess acted as the infectious nidus.

Management principles include prompt fluid resuscitation, continuous intravenous insulin, aggressive electrolyte replacement and early source control. Lifelong basal-bolus insulin and structured follow-up with endocrinology are essential for long-term glycaemic stability in LADA.

Emerging cohort data further delineate heterogeneity within LADA. The Action LADA study showed that individuals with high-titre anti-GAD65 antibodies progress to insulin dependency nearly three times faster than those with low titres, underscoring the value of quantitative antibody measurement. Finnish registries reveal that concomitant thyroid-peroxidase or trans-glutaminase antibodies are present in up to 30 % of LADA patients, supporting routine screening for autoimmune thyroid and coeliac disease. Metabolic phenotype also modulates risk: higher BMI and metabolic-syndrome features confer greater insulin resistance, often necessitating early combination therapy with insulin sensitisers such as metformin or GLP-1 analogues.

From a pathophysiological standpoint, sepsis accelerates β-cell apoptosis via pro-inflammatory cytokines (IL-1β, TNF-α) and oxidative stress. Experimental models suggest that timely IL-1 blockade may preserve residual β-cell mass, although clinical utility remains investigational. Recent consensus statements advocate meticulous inpatient transition from intravenous to subcutaneous insulin, guided by daily basal requirements calculated as 0.5-0.7 U/kg, to avert rebound hyperketonaemia. Finally, structured education on sick-day rules, glucose-ketone monitoring and early medical contact during infection is pivotal to preventing DKA recurrence.



Conclusions

A first episode of DKA in an adult with 'type 2-like' diabetes should raise the eyebrow for an autoimmune process. Getting the antibody panel early changes the long-game plan, helping patients accept lifelong insulin before another crisis hits. Rolling discharge plans into vaccination updates, infection surveillance and structured lifestyle support keeps people like our patient on the rails. This case also flags system-level issues. Antibody testing ought to be a standing order for adult DKA. Post-discharge reviews that weave in dietetics, psychology and continuous glucose monitoring give patients better odds of staying out of hospital. Finally, tightening antimicrobial stewardship and vaccination coverage may curb the very infections that drag susceptible patients into metabolic free-fall.

Sepsis-precipitated DKA in latent autoimmune diabetes requires fastidious acute care and durable long-term strategies. Timely surgical drainage, insulin therapy and electrolyte management reversed ketoacidosis, while autoimmune status compelled basal-bolus insulin for secondary prevention. Heightened clinical suspicion for LADA in adult DKA presentations may improve outcomes.

Beyond the immediate episode, this case highlights broader practice implications. First, antibody screening should be integrated into adult DKA workflows-particularly where phenotype and family history do not fully align with classical type 1 diabetes. Early serological confirmation facilitates tailored follow-up and patient counselling. Second, multidisciplinary discharge planning-bridging endocrinology, infectious-disease specialists, diabetes educators and dietitians-improves adherence and metabolic control. Third, vaccination status (influenza, pneumococcal, hepatitis-B) must be assessed, given the heightened infection risk in hyperglycaemic states. Fourth, structured lifestyle intervention addressing weight management, sleep hygiene and stress reduction synergises with pharmacotherapy to preserve β-cell function. Finally, digital health solutions-including continuous glucose monitoring (CGM) and app-based ketone logging-empower patients to detect metabolic deterioration early. Implementing these evidence-based measures promises to curtail hospital readmissions, reduce healthcare costs and ultimately enhance quality of life for individuals navigating the dual challenges of autoimmune diabetes and sepsis-triggered metabolic emergencies.

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