



RESEARCH ARTICLE

SEVERE DIABETIC KETOACIDOSIS UNMASKING LATENT AUTOIMMUNE DIABETES IN ADULTS (LADA): A CASE REPORT

¹Dr. Sanjay P Khare and ²Dr. Minal Chaudhari

¹Head-Academics and Clinical Training: Apollo Hospital Navi Mumbai; ²DNB Student, Apollo Hospital Navi Mumbai

ARTICLE INFO

Article History:

Received 18th June, 2026

Received in revised form

24th July, 2026

Accepted 26th August, 2026

Published online 30th September, 2026

Key words:

Diabetic ketoacidosis, Severe metabolic acidosis, Latent Autoimmune Diabetes in Adults, Anti-insulin antibody, C-peptide, Renal calculus, Sepsis.

*Corresponding author:

Dr. Sanjay P Khare

ABSTRACT

Diabetic ketoacidosis (DKA) is a dire emergency in patients with DM, but its severity and the triggering factors are not always plain and straightforward. We present the case of a middle aged man with known diabetes mellitus who presented with profound, life-threatening DKA with pH as low as 6.89, in the absence of an initially identifiable trigger. Beyond the acute resuscitation, the unusually severe presentation and the extraordinarily high insulin requirement during recovery raised clinical suspicion of an underlying autoimmune process, ultimately confirmed as Latent Autoimmune Diabetes in Adults (LADA) ¹. This case underscores the importance of looking beyond the obvious, both in the search for an underlying aetiology and in reconsidering an existing diagnosis when the clinical course does not fit the expected pattern.

Copyright©2023, Sanjay P Khare and Minal Chaudhari. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Dr. Sanjay P Khare and Dr. Minal Chaudhari. 2023. "Severe diabetic ketoacidosis unmasking latent autoimmune diabetes in adults (LADA): A case report". *International Journal of Current Research*, 19, (09), 38294-38296.

INTRODUCTION

A 41-year-old male, a known case of diabetes mellitus managed on OPD basis with insulin and oral hypoglycaemic agents (OHAs), presented with complaints of fatigue, polydipsia and weakness for two days. The symptoms were severe enough to render him unable to stand unsupported with two to three episodes of vomiting associated with abdominal pain. There was a history of one missed dose of insulin prior to the onset of symptoms. He unwisely took lots of electrical powder in water to quench his thirst which worsened his symptoms. Finally he was brought to ER in early dawn in a precarious condition. On presentation, the patient was drowsy and disoriented with hypotension, tachycardia, and tachypnoea. He had Kussmaul breathing with a classic fruity odour on his breath, raising immediate suspicion of severe diabetic ketoacidosis. Capillary blood glucose was so high that it could not be recorded on the glucometer. (Subsequently His blood sugar were reported to be >1200 mg/dl by lab). Blood ketone was markedly elevated at 4.7 mmol/L. A venous blood gas (VBG) performed on presentation confirmed severe high-anion-gap metabolic acidosis with severe acidaemia (pH 6.89), severe hyperkalaemia, hyponatraemia, and inadequate respiratory compensation with a concurrent respiratory acidosis component — a picture most consistent with severe DKA with impending respiratory fatigue.

Notably, the blood glucose on VBG could not be reported, as it exceeded the upper limit of the analyser's measurable range (highest recordable reading: 1080 mg/dL).

INVESTIGATIONS

MANAGEMENT

Immediate intravenous fluid resuscitation was initiated. It was one of those rare cases where, in view of the severity of acidosis, intravenous sodium bicarbonate had to be administered. An insulin infusion was started and carefully titrated to achieve a gradual reduction in blood glucose. Once sugars dropped to 300, vehicle was changed to half strength DNS in order to avoid cerebral oedema and other complications associated with rapid osmotic decline. Serum potassium was monitored closely; as expected, levels fell with correction of acidosis and initiation of insulin therapy, and intravenous potassium correction was subsequently started to treat the resultant hypokalaemia. In view of a raised total leucocyte count and elevated procalcitonin, empirical antibiotic therapy with cefoperazone-sulbactam was started, which was later escalated to meropenem in view of evolving sepsis. No definite focus of sepsis was identified at this stage. Inotropic support was instituted for persistent hypotension that was unresponsive to fluid resuscitation alone. Ultrasonography

Table 1. Serial Laboratory Investigations

Test	Day 1	Day 2	Day 3	Day 4	Day 6	Day 8
WBC	30.5k	15.34k	9.73k	5.57k	—	7.88k
CRP	74	—	—	—	—	7.6
PCT	10.47	—	—	—	—	0.13
Na / K	111 / 6.2	132 / 3.7	139 / 3.6	139 / 3.8	—	132 / 5.2
Vit D	19.9	—	—	—	—	—
Calcium	7.3	—	—	—	—	—
Creatinine	2.56	1.6	1.0	0.75	—	1.02
Urine Ketones	+	+	+	+	—	Trace

Table 2. Serial ABG / VBG Results

Test	Day 1					Day 2
	7 am	8 am	10 am	2 pm	8 pm	9 am
pH	6.891	6.901	7.027	7.306	7.335	7.452
pCO ₂	27.3	20.8	14.8	20.9	33.1	31.6
pO ₂	34.2	45.2	10.8	40.1	62.8	83.2
HCO ₃	5.0	5.5	6.8	13.5	18.7	23.5
Lac	2.9	1.9	1.2	2.4	1.0	1.2
Glu	77	417	769	373	248	72
Na	113	115	122	130	131	136
K	6.9	6.1	4.8	3.1	3.6	3.4

of the abdomen revealed a left renal calculus. Contrast-enhanced CT (CECT) of the abdomen performed for further evaluation demonstrated a left staghorn calculus with moderate left hydronephrosis. Urine R was normal and culture (urine+blood) yielded no pathogen. Retrograde intrarenal surgery (RIRS) with DJ stenting was planned but deferred in view of the patient's fluctuating blood glucose levels and overall clinical instability. The insulin infusion rate was gradually tapered as the patient stabilised, and urine ketones were monitored daily until they turned negative.

FURTHER EVALUATION AND DIAGNOSIS OF LADA

The severity of the DKA and the markedly elevated (“mega”) doses of insulin required to achieve glycaemic control, in a middle aged patient with no clearly identifiable precipitant, raised clinical suspicion of Latent Autoimmune Diabetes in Adults (LADA). Anti-insulin antibody testing returned positive, and serum C-peptide was found to be low, both findings complementing the clinical suspicion/ diagnosis of LADA. Subsequently, in latter days, urine routine examination revealed the presence of pus cells, raising the possibility that an underlying urinary tract infection may have been the focus of sepsis and the inciting event for DKA. The patient was accordingly continued on meropenam.

OUTCOME AND FOLLOW-UP

The patient showed gradual and sustained clinical improvement with correction of acidosis, electrolyte derangements, and resolution of sepsis-related haemodynamic instability. Inotropic support was weaned off, and once his oral intake was satisfactory, the insulin infusion was successfully transitioned to a combination of subcutaneous insulin and OHAs. He was discharged in a haemodynamically stable condition on this combination regimen, with advice for close glycaemic monitoring and outpatient urology follow-up for definitive management of the left staghorn calculus with RIRS and DJ stenting once feasible. He returned after 10 days in a fairly euglycemic state. All his parameters were normal. As planned, his RIRS was done. Immediately post removal of the obstructive calculus, there was clinically noticeable fungal discharge in urine. Clinically fluconazole was started and dose augmented once cultures returned positive.

He subsequently completed a 14 day course of oral fluconazole. As of last follow up, he was doing well with well controlled sugars and a normal urine report. His DJ stent removal was uneventful.

DISCUSSION

Diabetic ketoacidosis is a triad of hyperglycaemia, metabolic acidosis, and ketosis resulting from absolute or relative insulin deficiency. It is most often precipitated by infection, missed (or inadequate) insulin therapy, acute illness, or new-onset diabetes. The severity of acidosis in this patient, with a pH of 6.89, is at the extreme end of the spectrum and is associated with significant risk of cardiovascular collapse, arrhythmia, and death if not managed aggressively and promptly. Kussmaul breathing and a fruity (acetone) odour on the breath are classic clinical clues to underlying ketoacidosis and should prompt immediate point-of-care testing for blood glucose and ketones, even before laboratory confirmation is available. In this patient, the inability to record blood glucose on the glucometer (exceeding 1080 mg/dL) was itself a marker of the severity of the metabolic derangement. A notable feature of this case was the initial absence of an identifiable inciting cause. While a single missed dose of insulin was elicited on history, this alone would not typically be expected to produce such profound decompensation, prompting a search for an additional contributory factor. The eventual finding of pyuria on urine routine examination raised the possibility of an occult urinary tract infection as a covert trigger, illustrating the importance of a systematic search for infection even when no localising symptoms were initially apparent. Post RIRS (for the massive staghorn calculus) the fungal infection became apparent raising the doubt about it always being present but masked anatomically. As to why he responded initially in first admission with only meropenam and no antifungals, this remains a mystery. Equally important was the recognition of LADA in this patient. LADA is a form of autoimmune diabetes that shares features of both type 1 and type 2 diabetes mellitus, typically presenting in adulthood with a slower initial decline in beta-cell function but eventual progression to absolute insulin dependence.² The unusually severe DKA and the requirement for very high insulin doses to regain control, in a patient previously thought to have straightforward type 2

diabetes managed partly with OHAs and insulin, was the clinical trigger for further evaluation. Positive anti-insulin antibodies along with low C-peptide levels confirmed an autoimmune, insulin-deficient process, consolidating the diagnosis of LADA. This distinction is clinically important, as it has implications for long-term management, the limited or absent role of OHAs that depend on residual beta-cell function, and the need for aggressive/individualised/ target driven insulin-based therapy going forward.⁴ The incidental finding of a left staghorn calculus with hydronephrosis added a further layer of complexity to this case. Although deferred during the acute illness, it represented a significant finding that required definitive urological management and could itself have been a covert source of infection contributing to the initial presentation.

CONCLUSION

This case highlights that severe, life-threatening DKA can occasionally present without an immediately apparent underlying cause, and a systematic, persistent search for one — including occult infection — is essential. It further underscores that unusually severe DKA or unexpectedly high insulin requirements during recovery should prompt reconsideration of the underlying diabetes subtype, with a high index of suspicion for LADA in the appropriate clinical context.⁵ Early recognition and aggressive, protocol-driven management of severe DKA, along with careful electrolyte monitoring and judicious correction, are essential to achieving a favourable outcome.

LEARNING POINTS

- Severe DKA with profound acidaemia (pH < 7.0) is a medical emergency requiring aggressive fluid resuscitation, judicious insulin titration, and close electrolyte monitoring to avoid complications such as cerebral oedema.
- Absence of an obvious inciting event on initial assessment should prompt a thorough and ongoing search, including for occult infection, rather than premature closure of the diagnostic process. The cause might well be unmasked in subsequent tests.

- Unusually severe DKA and/or unexpectedly high insulin requirements in a middle aged patient with diabetes mellitus should raise suspicion for LADA.
- Anti-insulin antibody positivity together with low C-peptide levels supports the diagnosis of LADA and has implications for long-term therapy.³
- Incidental findings such as renal calculi identified during acute illness must be noted and addressed electively once the patient is stable, as they may represent both a long-term risk and a potential occult source of infection.

It is a good idea not to settle for a single, convenient explanation, especially in a complex or unusually severe presentation. A persistent, multidimensional approach to diagnosis often uncovers additional, clinically important findings.

REFERENCES

- Xenou M, Zoupas I, Lygnos D, Fousteris E. Diabetic ketoacidosis as first presentation of latent autoimmune diabetes in adults in a patient with hashitoxicosis as first presentation of Hashimoto's thyroiditis: a case report. *J Med Case Rep.* 2022; 16:PMC9347147. Available from: <https://doi.org/10.1186/s13256-022-03523-3>
- Buzzetti R, Tuomi T, Mauricio D, Pietropaolo M, Zhou Z, Pozzilli P, et al. Management of Latent Autoimmune Diabetes in Adults: A Consensus Statement From an International Expert Panel. *Diabetes.* 2020; 69(10):2037-2047. Available from: <https://doi.org/10.2337/dbi20-0017>
- Pieralice S, Pozzilli P. Latent Autoimmune Diabetes in Adults: A Review on Clinical Implications and Management. *Diabetes Metab J.* 2018; 42(6):451-464. Available from: <https://doi.org/10.4093/dmj.2018.0190>
- O'Neal KS, Johnson JL, Panak RL. Recognizing and Appropriately Treating Latent Autoimmune Diabetes in Adults. *Diabetes Spectr.* 2016; 29(4):249-252. Available from: <https://doi.org/10.2337/ds15-0047>
- Maraschin JF, Murussi N, Witter V, Silveiro SP. Classificação do diabetes mellitus [Diabetes Mellitus Classification]. *Arq Bras Cardiol.* 2010;95(2):40-46. Portuguese. Available from: <https://doi.org/10.1590/s0066-782x2010001200025>
