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Acute Appendicitis as a Precipitating Factor for Diabetic Ketosis in Newly Diagnosed Type 2 Diabetes Mellitus: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Funds Collection G

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Patient: Male, 47-year-old
Final Diagnosis: Diabetes mellitus type 2
Symptoms: Abdominal pain • diarrhea • dizziness • nausea • vomit
Clinical Procedure: —
Specialty: Metabolic Disorders and Diabetics

Objective: Unusual clinical course

Background: Infection is a well-established precipitant of hyperglycemic crisis in patients with diabetes. The concurrent presentation of acute appendicitis and diabetic ketosis (DK) poses a diagnostic challenge because of overlapping abdominal symptoms.

Case Report: A 47-year-old man was diagnosed with acute appendicitis based on characteristic symptoms and positive findings on ultrasound and computed tomography (CT). Laboratory test results revealed elevated serum glucose at 24.46 mmol/L, serum total CO_2 at 21.6 mmol/L, and an elevated serum beta-hydroxybutyrate level of 3178 µmol/L. The patient was diagnosed with new-onset type 2 diabetes mellitus (T2DM), presenting with DK triggered by acute appendicitis. Following the patient's refusal to undergo surgery, a conservative regimen consisting of intravenous antibiotics, fluid resuscitation, and intensive insulin therapy was successfully administered.

Conclusions: This case demonstrates that acute appendicitis, although an uncommon trigger, can serve as a critical precipitating factor for severe DK in adults with newly diagnosed T2DM. It highlights the underlying pathophysiological interplay between intra-abdominal infection, systemic inflammatory response, and acute metabolic decompensation. The case emphasizes the importance of maintaining a high index of suspicion for occult infections in patients presenting with DK, particularly when abdominal symptoms are prominent or atypical. Early imaging evaluation and comprehensive laboratory assessment are essential for accurate diagnosis. Moreover, this report illustrates that timely initiation of intensive insulin therapy, appropriate antimicrobial treatment, and supportive care can lead to favorable clinical outcomes. In selected patients who decline surgery, carefully monitored conservative management may represent a viable alternative.

Keywords: appendicitis • diabetes mellitus, type 2 • ketosis

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952909

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Introduction

Diabetic ketosis (DK) has traditionally been considered a hallmark complication of type 1 diabetes mellitus, but its incidence in patients with type 2 diabetes mellitus (T2DM) has increased substantially in recent decades [1,2]. Recent epidemiological data demonstrate that a large and growing proportion of DK hospitalizations now occur in patients with T2DM, particularly in the context of systemic infections, severe physiological stress, or iatrogenic factors. Among infectious precipitants, pneumonia and urinary tract infections predominate; however, intra-abdominal infections are increasingly being recognized as consequential triggers [3].

Crucially, acute appendicitis specifically is an uncommon trigger for DK, and its concurrent presentation with metabolic acidosis creates significant diagnostic challenges [4]. The shared symptomatology of nausea, vomiting, abdominal tenderness, and leukocytosis can easily obscure the identification of the primary infection source. Case reports have demonstrated that acute appendicitis can precipitate DK, particularly in patients with established type 1 diabetes [5]. More broadly, infection and acute inflammatory stress are recognized triggers of DK, whereas euglycemic DK has most frequently been associated with SGLT2 inhibitor use [6].

We present a rare instance of acute appendicitis-precipitated DK in a patient newly diagnosed with T2DM. This case adds significant clinical value by delineating the pathophysiology of inflammation-mediated metabolic derangement and providing an optimized workflow for differentiating DK from surgical acute abdomens.

Case Report

A 47-year-old man had no prior diagnosis of T2DM, but he reported a recent history of polydipsia, polyuria, and weight loss. He visited the emergency department for severe nausea, repeated vomiting, diffuse abdominal pain, diarrhea, and dizziness lasting for 5 hours. The patient reported consuming contaminated food prior to the onset of illness. He had a history of hepatic steatosis and no previous episodes of DK.

On assessment, the patient appeared dehydrated and tachypneic. Vital signs included a temperature of 38.5 °C, heart rate of 118 beats/min, blood pressure of 130/80 mm Hg, respiratory rate of 28 breaths/min with Kussmaul breathing, and oxygen saturation of 97% on room air. Physical examination revealed prominent tenderness in the right lower quadrant, accompanied by rebound tenderness and guarding. Cardiac and pulmonary examination results were unremarkable.

Given that the severity and localization of the abdominal pain were disproportionate to typical DK-related discomfort, abdominal imaging was promptly performed after admission. Ultrasound (**Figure 1**) and CT scans (**Figure 2**) revealed an enlarged appendix, fluid accumulation, and periappendiceal exudation indicative of acute appendicitis.

Laboratory testing (**Table 1**) revealed an elevated serum glucose level of 24.46 mmol/L, an elevated glycated serum protein level of 5.9 mg/L, and a markedly elevated serum beta-hydroxybutyrate level of 3178 µmol/L. An infectious workup revealed elevated inflammatory markers, consistent with a bacterial infection. Further workup ruled out alcoholic and starvation ketosis. Although arterial blood gas analysis was unavailable, the updated 2024 international consensus report on

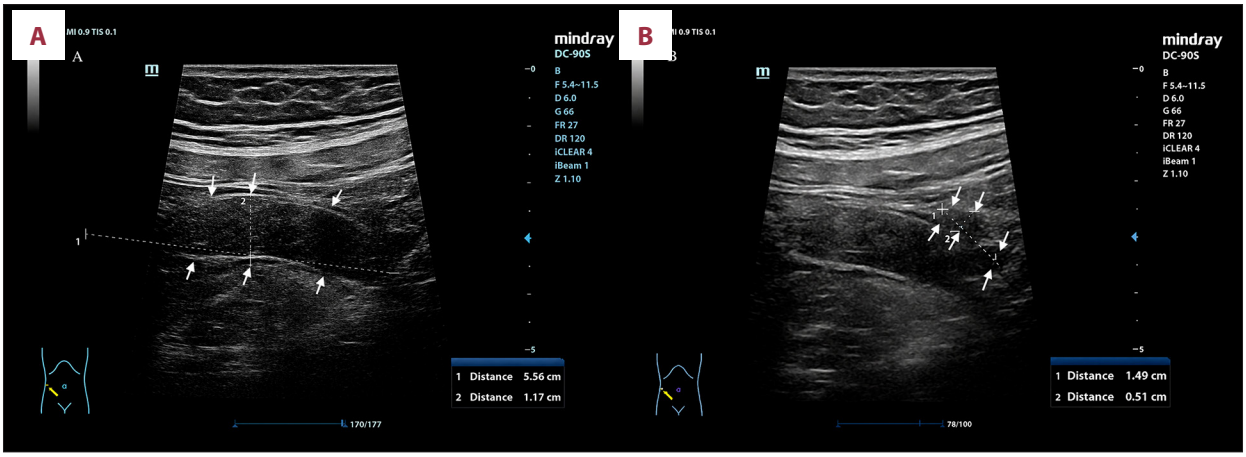

Figure 1. Ultrasound image. (**A**) Ultrasound shows an enlarged appendix (indicated by the white arrows, measuring approximately 5.56 cm in length and 1.17 cm in diameter) with decreased echogenicity, anechoic lumen, and poor acoustic transmission. (**B**) Ultrasound shows an indistinct contour of the appendiceal tip (indicated by the white arrows), with an approximately 1.56 × 0.51 cm irregular anechoic area adjacent to the appendix. Findings are suggestive of acute appendicitis.

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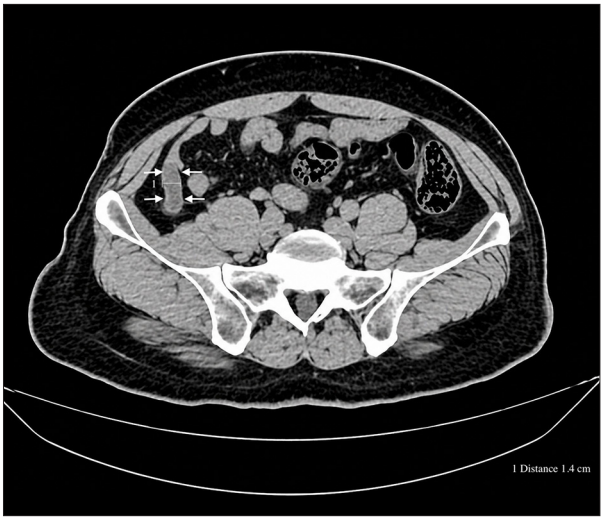

Figure 2. Computed tomography image. Computed tomography shows an enlarged appendix (indicated by the white arrows, measuring approximately 1.4 cm in diameter), fluid accumulation, and periappendiceal exudation, consistent with acute appendicitis.

hyperglycemic crises [7] explicitly eliminates the strict requirement for arterial blood gas, instead endorsing quantitative beta-hydroxybutyrate measurement for diagnosing ketosis. In accordance with these current standards, our patient's markedly elevated serum beta-hydroxybutyrate level, alongside a mildly reduced serum total CO_2 (21.6 mmol/L), without severe acidotic decompensation, supported the diagnosis of DK rather than overt diabetic ketoacidosis (DKA). The final diagnosis was new-onset T2DM with DK triggered by acute appendicitis.

However, the patient refused any surgical intervention. This refusal necessitated a rigorous, monitored, and conservative management strategy utilizing antibiotics and intensive insulin therapy. Given the shared pathophysiological mechanisms between DK and DKA, management followed standard DKA protocols [3]: (1) initiate immediate resuscitation with intravenous isotonic saline; (2) commence continuous intravenous insulin infusion at 0.1 U/kg/h with glucose monitoring; (3) perform frequent electrolyte monitoring and administer potassium replacement as required; and (4) administer empirical broadspectrum antibiotics due to suspected intraabdominal infection and local severity.

Infection control led to a significant reduction in insulin requirements, prompting a switch to oral hypoglycemic agents, and the patient was discharged on day 6. The patient remained symptom-free at the 2-month follow-up.

Discussion

This case highlights an underrecognized interaction between acute intra-abdominal infection and metabolic decompensation in patients with T2DM. While infection is a well-established precipitant of DK [3], acute appendicitis as the initial trigger—particularly in newly diagnosed T2DM—is rare and poses significant diagnostic challenges.

From a mechanistic perspective, this case reflects inflammation-driven metabolic dysregulation. Acute appendicitis induces a systemic inflammatory response characterized by elevated cytokines such as interleukin 6 and tumor necrosis factor α

Table 1. Laboratory findings on admission with reference ranges.

Laboratory test	Value	Reference range
Blood glucose level (mmol/L)	24.46	3.9-6.1
Serum β – hydroxy butyrate (umol/L)	3178	0-280
Glycated serum protein (mg/L)	5.9	1.4-2.95
Serum total CO_2	21.6	22-29
Sodium	139	135-145
Potassium	4.53	3.5-5.5
Creatinine	58	54-133
Glycosuria	2+	Negative
Urinary ketones	4+	Negative
White blood cell count (/L)	16.45×10^9	$3.5\text{-}9.5 \times 10^9$
Neutrophil count (/L)	14.89×10^9	$1.8\text{-}6.3 \times 10^9$
Neutrophil ratio	90.5%	40%-75%
Blood amylase (U/L)	23	35-135

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[8], which impair insulin signaling through inhibition of insulin receptor substrate pathways [9]. This process is further amplified by counter-regulatory hormones, promoting lipolysis and ketogenesis [3]. Notably, such stress responses may unmask latent metabolic dysfunction, suggesting that DK can represent a dynamic response to acute physiological stress rather than solely a complication of established diabetes.

A major clinical challenge lies in the overlap of symptoms between DK and appendicitis. Abdominal pain, nausea, vomiting, and leukocytosis are common to both conditions [4,10], increasing the risk of diagnostic anchoring. Clinicians may misattribute abdominal findings to DK-related pseudo-peritonitis [11], delaying recognition of a surgical abdomen [12]. In this case, localized right lower quadrant tenderness prompted timely imaging, which proved critical for diagnosis. These findings support the need for early imaging in patients with DK presenting with focal abdominal signs, rather than attributing symptoms exclusively to metabolic causes [10].

This case also reflects evolving diagnostic standards. The 2024 international consensus report emphasizes quantitative beta-hydroxybutyrate measurement over arterial blood gas analysis for diagnosing hyperglycemic crises [7]. Here, markedly elevated beta-hydroxybutyrate levels combined with mildly reduced serum total CO_2 were sufficient to establish DK, demonstrating the practical utility of updated biochemical criteria [7].

From a management perspective, although appendectomy remains the standard of care, this case illustrates that conservative treatment may be feasible in selected patients when surgery is declined. Intensive insulin therapy combined with targeted antimicrobial treatment effectively controlled both the metabolic and infectious processes, interrupting the cycle of inflammation and ketosis [3,13]. However, this approach should be applied cautiously given the potential risks associated with delayed surgical intervention [10].

In conclusion, acute appendicitis can act as a rare but important trigger of DK, even as the first manifestation of T2DM. This case underscores the importance of maintaining diagnostic vigilance, applying updated diagnostic criteria, and adopting an integrated approach that simultaneously addresses metabolic and infectious etiologies [3,7].

Conclusions

This case demonstrates that acute appendicitis, albeit uncommon, is a critical precipitating factor for severe DK in adults with newly diagnosed T2DM. Successful management depends on a high index of clinical suspicion to overcome diagnostic pitfalls, coupled with early radiographic evaluation and prompt antimicrobial therapy for effective source control. Clinicians must maintain a high level of vigilance for potential intra-abdominal sources of infection in diabetic patients presenting with persistent or localized abdominal tenderness. As evidenced by this patient's rapid stabilization, integrating the latest 2024 consensus guidelines for diagnosis and immediately initiating comprehensive medical and antimicrobial therapy is crucial for halting disease progression and securing optimal patient outcomes, even when surgical intervention is declined.

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Patient Consent

Informed consent for publication of this case report has been obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Dhatariya KK, Glaser NS, Codner E, Umpierrez GE. Diabetic ketoacidosis. Nat Rev Dis Primers. 2020;6(1):40
2. Umpierrez GE, Kitabchi AE. Diabetic ketoacidosis: Risk factors and management strategies. Treat Endocrinol. 2003;2(2):95-108
3. Fayfman M, Pasquel FJ, Umpierrez GE. Management of hyperglycemic crises: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. Med Clin North Am. 2017;101(3):587-606
4. Umpierrez G, Freire AX. Abdominal pain in patients with hyperglycemic crises. J Crit Care. 2002;17(1):63-67
5. Thompson BD, Kitchen A. Euglycemic diabetic ketoacidosis in type 1 diabetes on insulin pump, with acute appendicitis: A case report. Clin Pract Cases Emerg Med. 2021;5(3):136-38
6. Griffin TP, Dinneen SF. SGLT2 inhibitors increase risk for diabetic ketoacidosis in type 2 diabetes. Ann Intern Med. 2020;173(8):JC40
7. Umpierrez GE, Davis GM, ElSayed NA, et al. Hyperglycaemic crises in adults with diabetes: A consensus report. Diabetologia. 2024;67(8):1455-79
8. Hotamisligil GS, Erbay E. Nutrient sensing and inflammation in metabolic diseases. Nat Rev Immunol. 2008;8(12):923-34

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

9. Mottale R, Dupuis C, Szklarzewska S, Preiser JC. The metabolic response to stress in critical illness: Updated review on the pathophysiological mechanisms, consequences, and therapeutic implications. Ann Intensive Care. 2025;15(1):174
10. Macaluso CR, McNamara RM. Evaluation and management of acute abdominal pain in the emergency department. Int J Gen Med. 2012;5:789-97
11. Csomor J, Jirkovska J, Vedralova L, et al. Diabetic ketoacidosis with an acute abdomen as a first manifestation of type 1 diabetes mellitus. Acta Endocrinol (Buchar). 2017;13(4):509-11
12. Zhang M, Li J, Fan L, Liu H. Misdiagnosis of diabetic ketoacidosis: A case report. J Med Case Rep. 2025;19(1):409
13. American Diabetes Association. Standards of Care in Diabetes—2024. Diabetes Care. 2024;47(Suppl. 1):S1-S321

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