Diabetic ketoacidosis, severe hypertriglyceridemia, and acute pancreatitis in type 2 diabetes mellitus: a two-patient case series

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ABSTRACT

The coexistence of diabetic ketoacidosis (DKA) complicated by severe hypertriglyceridemia and acute pancreatitis is a rare, but a life-threatening condition that poses considerable diagnostic and management difficulties. In this case series, we describe two cases of young men with type 2 diabetes mellitus (T2D) with profound DKA, marked hypertriglyceridemia, and acute pancreatitis. Intensive treatment with intravenous insulin, aggressive fluid replacement, and supportive measures led to rapid resolution of ketoacidosis, substantial reduction in triglyceride levels, and clinical improvement. These cases highlight the intricate metabolic relationship between insulin deficiency, severe hypertriglyceridemia, and pancreatic injury in T2DM, emphasizing the importance of early recognition, insulin-based management, and close metabolic follow-up to guide long-term glycemic and lipid control.

Keywords: Diabetic ketoacidosis, severe hypertriglyceridemia, acute pancreatitis, insulin treatment, case series.

Type of Article: CASE SERIES **Specialty:** Internal Medicine

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Introduction

Diabetic ketoacidosis (DKA) is an acute and potentially life-threatening metabolic complication characterized by hyperglycemia, metabolic acidosis with a high anion gap, and ketosis. The incidence of DKA in type 1 diabetes (T1D) is substantially higher than in type 2, with reported rates of 178.6 and 20 cases per 10,000 person-years, respectively [1]. DKA in T1D manifests due to an absolute insulin deficiency, but it may also occur in T2D under conditions of relative insulin deficiency, often triggered by stressors such as infection, acute pancreatitis, trauma, alcohol use, and SGLT2 inhibitor use [2].

Acute pancreatitis (AP) is an acute inflammatory disorder of the pancreas associated with sudden onset of abdominal pain, especially in the epigastrium and radiating to the back, accompanied by the elevation of serum pancreatic enzymes (amylase and lipase). The most common etiologies are biliary stones and alcohol use. Hypertriglyceridemia is a relatively uncommon cause of AP, responsible for 1%-4% of cases, and is most commonly implicated when serum triglyceride (TRG) levels exceed 11.3 mmol/l [3,4].

AP is an established and relatively rare associated and contributory factor in DKA, and the coexistence of both entities creates diagnostic and management challenges. This case series describes two young men presenting with DKA, severe hypertriglyceridemia, and acute pancreatitis. It aims to underline the complex interplay of these conditions, highlight the importance of early recognition, and discuss keys to consider in diagnosis and management to improve clinical outcomes in this high-risk presentation.

Case Presentation

Case 1

A 36-year-old male presented with acute left upper abdominal pain radiating to the back for the last 2 days, accompanied by vomiting. He had been diagnosed with type 2 diabetes 5 months earlier. Initial examinations revealed hyperglycemia, moderate-to-severe hypertriglyceridemia, and negative anti-insulin antibodies (Table 1). Despite clinical recommendations to initiate both anti-diabetic (linagliptin) and hypolipidemic (atorvastatin/ezetimibe) treatments, the patient exhibited non-adherence and did not follow the prescribed regimens. He reported social alcohol consumption and no tobacco use. His family history included paternal coronary artery disease.

On physical examination, he was tachypneic with a fruity breath odor and mild tenderness of the left hypochondrium. He was hemodynamically stable and fully

Table 1. Biochemical values before admission, at admission and during outpatient follow-up.

Parameter	Reference range	Case 1 5 months Before Admission	Case 1 Admission	Case 1 Discharge	Case 1 2-month follow-up	Case 2 Admission	Case 2 Discharge	Case 2 1-month follow-up	Case 2 2-month follow-up	Case 2 4-month follow-up
Glucose, mg/dl (mmol/l)	74–106 (4.1–5.88)	167 (9.2)	447 (24.8)	105 (5.82)	97 (5.38)	307 (17.04)	225 (12.49)	130 (7.22)	118 (6.55)	95 (5.27)
Triglycerides, mg/dl (mmol/l)	<150 (<1.69)	569 (6.43)	5,010 (56.56)	192 (2.16)	139 (1.57)	3,501 (39.56)	667 (7.54)	177 (2.00)	112 (1.27)	111 (1.25)
Serumamylase, U/l	22–80	–	444	49	–	233	197	173	80	–
HbA1c, % (mmol/mol)	4.2–6.2 (22–44)	6.9 (52)	9.3 (78)	–	–	10.6 (92)	–	–	–	5.6 (38)
C-peptide, ng/ml (nmol/l)	1.1–4.4 (0.36–1.46)	5.37 (1.77)	–	4.96[1] (1.64)	–	–	2.61[2] (0.86)	–	–	–

Table of laboratory test results (Glucose, Triglycerides, Serum Amylase, HbA1c, C-peptide) 5 months prior to admission for the first case, as well as at admission, at discharge, and during the subsequent months of outpatient follow-up for case 1 and case 2.

Abbreviations: HbA1c, glycated hemoglobin.

[1]Measured with concomitant plasma glucose of 180 mg/dl (9.9 mmol/l).

[2]Measured with concomitant plasma glucose of 250 mg/dl (13.8 mmol/l).

oriented. Obesity (BMI 43.8 kg/m^2, waist circumference 142 cm) and insulin resistance features (hyperpigmentation of the neck, axilla) were present. No xanthoma or xanthelasma or features of familial partial lipodystrophy (FPLD) or Cushing syndrome (CS) were observed. Laboratory evaluation revealed hyperglycemia, high anion gap metabolic acidosis, ketonemia, ketonuria (+++), and severe hypertriglyceridemia. High serum and urine amylase levels (unavailable lipase level measurement) and inflammation markers were also noted (Tables 1 and 2).

Based on the Revised Atlanta Classification, the patient was classified as having moderate acute pancreatitis, taking into account the renal dysfunction (eGFR = 59 ml/min/1.73 m^2) for <48 hours. An acute necrotic collection measuring 2.2 x 2.3 cm between the tail and the body of the pancreas was detected in abdominal computerized tomography (Figure 1). No cholelithiasis or biliary dilation was detected; however, hepatic fatty infiltration was observed.

Intravenous hydration and insulin were administered immediately for the resolution of DKA and severe hypertriglyceridemia. At least 100 units of rapid-acting insulin per day were administered intravenously during the first 2 days of hospitalization for the resolution of DKA (Table 3). The administration of fibrates and plasmapheresis could be considered if severe hypertriglyceridemia failed to resolve with insulin treatment. Despite international recommendations regarding antibiotic use in acute pancreatitis, the administration of meropenem was chosen due to a strong suspicion of infection associated with documented necrosis. Pethidine (rather than fentanyl or morphine) was selected as the analgesic, based on our clinic's experience with similar cases of acute pancreatitis, and low molecular weight heparin for thromboprophylaxis, while the patient remained fasting. Gradually, the symptoms subsided within 1 week, and the laboratory findings improved. A multiple daily injection insulin regimen (insulin glargine as basal and rapid-acting insulin as bolus) was initiated when the patient began a liquid diet on 10th day of hospitalization. The total daily dose of insulin therapy was ~50-60 units per day, which was then reduced alongside the patient's clinical improvement.

The patient was discharged asymptomatic after 21 days of hospitalization with insulin degludec 16 units once a day (with titration instructions) and a low-fat diet. Glucagon was prescribed to reverse a possible episode of severe hypoglycemia. Fibrates were not prescribed, as a dramatic reduction in triglyceride levels—from 5,010 to 192 mg/dl—was observed following insulin administration. As an outpatient, the glycemic and lipidemic control was on target on insulin monotherapy (14 units of insulin degludec) until his last visit 2 months post-discharge, while a further reduction in triglycerides to 139 mg/dl was noted on an outpatient basis with basal insulin monotherapy (Table 1). He had no recurrence of pancreatitis or DKA. He was lost

Table 2. *Arterial blood gas values in admission.*

PARAMETERS	CASE 1	CASE 2	REFERENCE RANGES
Ph	7.14	7.17	7.35-7.45
pCO_2	10	15	35-45 mmHg
HCO_3-	3.4	5.5	21-28 mmol/l
K+	6	4.7	3.5-5.1 mmol/l
Anion gap	23.6	18.5	4-12 mmol/l
Serum ketones	5.4	4.8	<0.6 mmol/l
Glucose	447 mg/dl (24.8 mmol/l)	307 mg/dl (17.04mmol/l)	74–106 mg/dl (4.11–5.88 mmol/l)

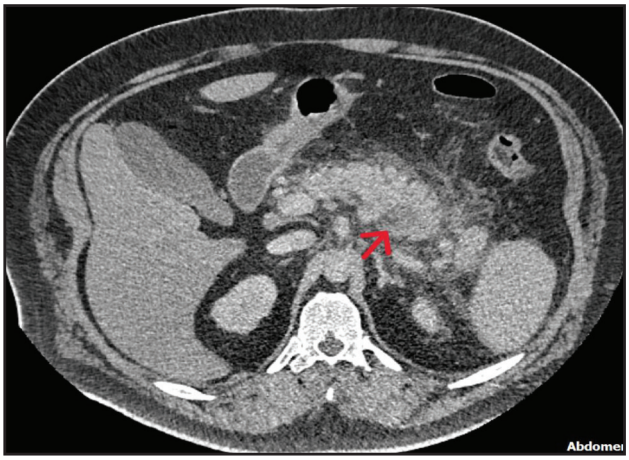

Figure 1. *Abdominal computerized tomography. Acute necrotic collection measuring 2.2 x 2.3 cm between the tail and the body of the pancreas (red arrow) and peripancreatic fat opacity.*

to follow-up, while Magnetic Resonance Imaging (MRI) of the upper abdomen was pending, for the exclusion of pancreatic cancer. A concise timeline for case 1 is provided in Table 4.

Case 2

A 30-year-old man presented with acute epigastric pain and vomiting for 24 hours. He had no known medical history but reported alcohol consumption of 168 units per week during the preceding 8 months and 45 pack-years of tobacco smoking. Family history was positive for T2D.

He was hemodynamically stable with mild epigastric tenderness. He did not present features of FPLD or CS. There was no evidence of xanthoma or xanthelasma. BMI was 28.3 kg/m^2 and waist circumference was 98 cm. Laboratory findings confirmed DKA with hyperglycemia, ketonemia, ketonuria (+++), and metabolic acidosis, along with severe hypertriglyceridemia and elevated serum amylase (unavailable lipase level measurement) (Tables 1 and 2). HbA1c was elevated, with preserved C-peptide levels and negative anti-β-cell antibodies, supporting a diagnosis of previously unrecognized T2D. Screening for illicit substances was negative.

Based on the Revised Atlanta Classification, the patient was classified as having moderate acute pancreatitis, due to renal impairment (eGFR = 49 ml/min/1.73 m²) for <48 hours. Imaging studies demonstrated mild pancreatic enlargement with peripancreatic fat opacity (Figure 2), without bile duct dilation or cholelithiasis. Fatty liver infiltration was also documented.

Intravenous hydration and insulin were administered immediately for the resolution of DKA and severe hypertriglyceridemia. At least 50 units of rapid-acting insulin were administered for the resolution of DKA within 10 hours (Table 3). Pethidine was chosen as an analgesic. Clinical symptoms and laboratory parameters gradually improved, while he remained fasting for the first 2 days of hospitalization. Alcohol withdrawal occurred without adverse effects.

He was discharged early from the hospital at his own request after 6 days, following a low-fat diet and 14 units of insulin degludec once a day with titration instructions. Fibrates were not prescribed during his hospitalization or outpatient follow-up, as triglyceride levels decreased with insulin therapy—from 3,501 to 112 mg/dl within 2 months. He is still regularly monitored as an outpatient. He has good glucose and triglyceride control with 12-14 units of basal insulin regimen therapy (Table 1). No recurrence of pancreatitis, DKA, or hypertriglyceridemia has been observed. A concise timeline for case 2 is provided in Table 4. Metformin may be added in case of poor glycemic control. GLP-1 RAs and DPP-4 inhibitors should be avoided due to the history of an acute pancreatitis episode, while SGLT2 inhibitors should be avoided due to a DKA episode. As far as the hypolipidemic management and cardiovascular protection, statin monotherapy or in combination with icosapent ethyl may be added in case of insufficient lipidemic control. No pancreatic pathology was identified on upper abdominal MRI.

Discussion

In DKA, insulin deficiency results in lipolysis and protein degradation, liberating a large number of free fatty acids (FFAs) and amino acids. Concomitantly, hypersecretion of counter-regulatory hormones (glucagon, cortisol, catecholamines, and growth hormone) enhances hepatic glycogenolysis and gluconeogenesis. Excessive hepatic uptake of FFAs results in an overproduction of very-low-density lipoproteins (VLDL), thereby increasing hypertriglyceridemia. Insulin deficiency further decreases the activity of lipoprotein lipase in peripheral tissues, thereby

Table 3. *Arterial blood gas values on resolution of DKA.*

PARAMETERS	CASE 1	CASE 2	REFERENCE RANGES
Ph	7.37	7.36	7.35-7.45
pCO_2	35	33	35-45 mmHg
HCO_3-	20	18	21-28 mmol/l
K+	3.6	3.8	3.5-5.1 mmol/l
Anion gap	10	11.9	4-12 mmol/l
Serum ketones	–	–	<0.6 mmol/l
Glucose	185mg/dl (10.27mmol/l)	160mg/dl (8.88mmol/l)	74-106mg/dl (4.11-5.88mmol/l)

Table 4. *Clinical timeline for each case.*

Case 1

TIME POINT	CLINICAL COURSE
2 days before admission	Symptoms of acute left upper abdominal pain radiating to the back, accompanied by vomiting.
~48 hours after admission	Resolution of diabetic ketoacidosis.
1 week	Symptoms resolution.
Hospital day 10	Introduction of a liquid diet.
Hospital day 21	Discharge.
2 month follow-up	Glycemic and lipidemic control with insulin monotherapy (14 units of insulin degludec). No recurrence of pancreatitis or diabetic ketoacidosis.

Case 2

TIME POINT	CLINICAL COURSE
24 hours before admission	Symptoms of acute epigastric pain and vomiting.
10 hours after admission	Resolution of diabetic ketoacidosis.
Hospital day 2	Diet reintroduction.
Hospital day 6	Early discharge at his own request.
2 month follow-up	Normalization of triglycerides. No recurrence of pancreatitis or diabetic ketoacidosis.
Regular follow-up	Glucose and triglyceride control with 12-14 units of basal insulin regimen therapy. No recurrence of pancreatitis or diabetic ketoacidosis.

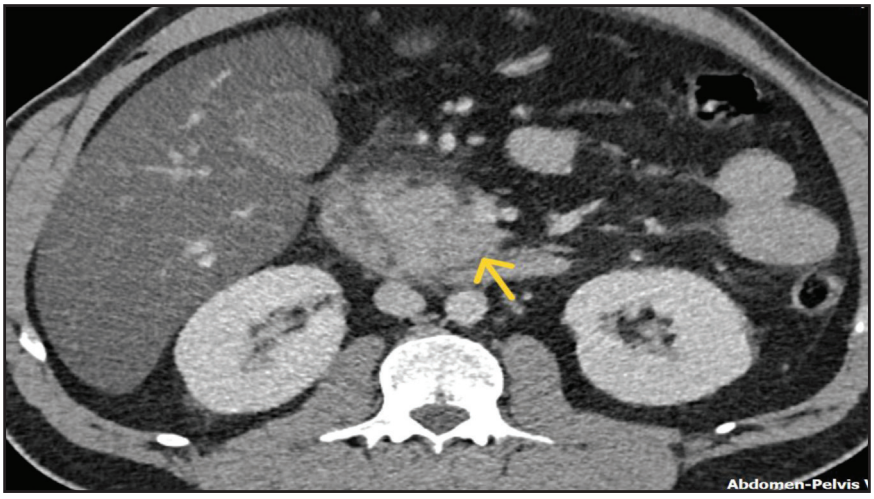

Figure 2. *Abdominal computerized tomography. Mild enlargement of the pancreas with peripancreatic fat opacity (yellow arrow).*

impairing VLDL clearance from the circulation. The aforementioned mechanisms increase TRG levels, which are hydrolyzed by pancreatic lipase into toxic fatty acids. Pancreatic injury manifests as acute pancreatitis [2,4-6].

DKA, acute pancreatitis, and severe hypertriglyceridemia have been reported in the literature. Case reports include individuals with a known history of T1D [4] and T2D [5,6]. DKA was resolved by intravenous insulin and

hydration, where hypertriglyceridemia was controlled with insulin administration [4]. In other cases, fenofibrate was prescribed for TRG level control, diminishing the risk of recurrence of acute pancreatitis [4,6]. However, concomitant presence of DKA and AP secondary to hypertriglyceridemia could result in multiorgan failure with fatal consequences [5].

Retrospective studies examined the clinical characteristics and outcomes of DKA episodes with and without acute pancreatitis [1,7]. Khan et al. [1] found a 9% prevalence of DKA and concurrent AP in T1D and T2D patients. Older age, male gender, Asian ethnicity, and T2D were associated with more severe DKA episodes, inflammation, kidney injury, longer hospitalization, and ICU admission rate. Ma et al. [7] reported that the prevalence of AP in DKA patients was 15.53% in T2D. Severe hypertriglyceridemia (TRG level > 10.52 mmol/l) was found to have the highest predicted value for acute pancreatitis onset in DKA patients. "Enigmatic triangle" has been documented since 1997, indicating the triple sign of DKA, hypertriglyceridemia, and acute pancreatitis. However, it is still unclear whether DKA is the leading cause or a complication of AP [5,7].

In our cases, the sequence of events is unclear, as all three acute conditions—DKA, acute pancreatitis, and severe hypertriglyceridemia—were present at admission. We hypothesize that hypertriglyceridemia was the primary trigger of acute pancreatitis in the first case, given the documented triglyceride levels >500 mg/dl 5 months earlier in the absence of lipid-lowering therapy, whereas excessive alcohol consumption was the likely trigger in the second case. In both patients, acute pancreatitis was subsequently complicated by DKA and severe hypertriglyceridemia secondary to relative insulin deficiency. Severe hypertriglyceridemia resolved with insulin monotherapy in both cases, supporting the concept that, such as DKA, it represents a manifestation of insulin deficiency. Therefore, insulin therapy should be considered the cornerstone of both acute and long-term management, whereas plasmapheresis and fibrates could be considered as last options.

In addition to the acute metabolic interactions between hypertriglyceridemia, DKA, and acute pancreatitis, longer-term pancreatic sequelae should also be considered. Most reviews and studies analyze the pathogenesis of pancreatic diabetes or type 3c diabetes, which is defined as new-onset diabetes after severe acute pancreatitis, excluding those with pre-existing DM. The estimated prevalence of the overall incidence of DM after AP is 23% [8]. However, there is no published study that specifically examines C-peptide levels before and after an acute pancreatitis episode in patients with pre-existing T2D. Duggan et al. [9] reported lower C-peptide levels during a 2-hour oral glucose tolerance test in patients with post-pancreatitis diabetes mellitus (type 3c) compared with diabetic controls. Further research is needed to determine the clinical utility of C-peptide measurement in guiding antihyperglycemic therapy.

Insulin therapy (basal or multiple injection regimen) is the most common post-acute pancreatitis antihyperglycemic treatment. Metformin may be added, while GLP-1 RAs and DPP-4 inhibitors should be avoided in post-acute pancreatitis cases. SGLT2 inhibitors are not advised to be prescribed in patients with a DKA episode [10-12]. It is noteworthy that the impaired glucagon secretion from α-cells predisposes to severe hypoglycemia, so clinicians should be aware of its prevention [3,12]. Lipid management with statins is needed for the initial management of hypertriglyceridemia. Icosapent ethyl, in addition to a statin, is necessary in high-risk or very high-risk patients with elevated triglyceride levels (fasting TRG level 135-499 mg/dl or 1.52-5.63 mmol/l) in order to reduce the risk of cardiovascular events [13]. Fibrates are used for the prevention of acute pancreatitis when TRG > 500 mg/dl (SI: 5.65 mmol/l) [14].

Finally, other possible causes of ketoacidosis should be considered, such as ketosis-prone diabetes (KPD) and alcoholic ketoacidosis (AKA) [10,15]. Ketosis-prone T2D is defined as newly diagnosed diabetes with an unprovoked DKA episode in certain ethnic groups (e.g., African American and Hispanic adults) with clinical features of T2D and a strong family history of diabetes. T1D autoimmunity is lacking, and β-cell function is preserved. Usually, after a short course of insulin therapy for DKA remission, individuals remain euglycemic with medical nutrition or non-insulin agents because of restoration of β-cell secretion and insulin sensitivity [2,16]. Our cases, particularly the second, are unlikely to represent KPD, as DKA was provoked by acute pancreatitis and insulin therapy remained necessary after resolution of the acute episode and during outpatient follow-up. Alcohol ketoacidosis manifests after alcohol cessation, which is rarely accompanied by hyperglycemia. Glucose administration resolves the ketoacidosis [15,17]. The aforementioned argue against this diagnosis in our cases.

Conclusion

The coexistence of DKA, severe hypertriglyceridemia, and acute pancreatitis is a rare, but potentially life-threatening metabolic emergency in patients with diabetes mellitus. This case series emphasizes the importance of early recognition of this "enigmatic triangle," and prompt insulin and fluid treatment is necessary for the resolution of the emergency. Fibrates and plasmapheresis may be considered as last options for the management of severe hypertriglyceridemia. Close insulin titration is required due to hypoglycemic vulnerability, especially following an episode of acute necrotizing pancreatitis. Diabetes type classification is challenging, and clinicians

should also consider other concomitant syndromes that may affect medical management (e.g., FPLD, CS, KPD, and AKA).

What's new?

"Enigmatic Triad" of diabetic ketoacidosis, severe hypertriglyceridemia, and acute pancreatitis is a well-known, but rare emergency in diabetes mellitus. Insulin monotherapy and fluid administration resolved the emergency without the addition of lipid-lowering agents or plasmapheresis. Diagnostic challenges in the classification of diabetes, recognition of other coexisting metabolic disorders, and medical care in the inpatient and outpatient settings are also described.

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Lists of Abbreviations

DM	Diabetes mellitus
DPP-4inh	Dipeptidyl peptidase 4 inhibitors
GLP-1 RAs	Glucagon-like peptide 1 receptor agonists
SGLT2inh	Sodium–Glucose Cotransporter 2 inhibitors

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Informed patient consent for publication

Signed informed consent could not be obtained from the patients or a proxy. However, the manuscript was reviewed and approved by the institutional ethics committee, and all identifying information was removed.

Ethical approval

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Summary of the case 1

1	Patient (gender, age)	Male, 36 years old
2	Final diagnosis	Diabetic ketoacidosis, severe hypertriglyceridemia, acute necrotizing pancreatitis, diabetes mellitus 2
3	Symptoms	Abdominal pain, vomiting
4	Medications	Intravenous insulin and hydration, pethidin, meropenem, thromboprophylaxis for resolution of the emergency, subcutaneous insulin (MDI initially, basal insulin as monotherapy)
5	Clinical procedure	Conservative treatment
6	Specialty	Internal medicine, endocrinology, gastroenterology

Summary of the case 2

1	Patient (gender, age)	Male, 30 years old
2	Final diagnosis	Diabetic ketoacidosis, severe hypertriglyceridemia, acute pancreatitis, diabetes mellitus 2
3	Symptoms	Abdominal pain, vomiting
4	Medications	Intravenous insulin and hydration, pethidin, thromboprophylaxis for resolution of the emergency, subcutaneous insulin (MDI initially, basal insulin as monotherapy)
5	Clinical procedure	Conservative treatment
6	Specialty	Internal medicine, endocrinology, gastroenterology