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Diabetic Ketoacidosis Complicated by Hypertriglyceridemia-Induced Pancreatitis: A Case Report

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Abstract

Background: Diabetic ketoacidosis (DKA) is a severe complication of diabetes characterized by hyperglycemia, metabolic acidosis, and ketosis. It can lead to complications such as hypertriglyceridemia-induced pancreatitis due to increased lipolysis and triglyceride formation. **Case Presentations:** A 28-year-old female presented with symptoms consistent with diabetic ketoacidosis (DKA), including polyuria, polydipsia, polyphagia, and unintentional weight loss. Laboratory results revealed severe hyperglycemia (glucose 22.9 mmol/L, HbA1c 14.5%), metabolic acidosis (pH 7.15), and elevated beta-hydroxybutyrate (6.75 mmol/L). Further evaluation showed markedly elevated triglycerides (45 mmol/L) and lipase (2928 IU/L), indicating mild pancreatitis. Upon reviewing her clinical presentation and lab findings, poorly controlled diabetes was determined to be the primary cause, leading to DKA and secondary hypertriglyceridemia. The patient was managed in the ICU with insulin therapy, fluid resuscitation, and Fenofibrate to address hypertriglyceridemia. Identifying the primary precipitant through this comprehensive assessment is crucial for directing management toward aggressive control of hyperglycemia, ketosis, and triglyceride levels. **Conclusions:** This case underscores the complex interplay between DKA, hypertriglyceridemia, and pancreatitis. Accurate clinical evaluation is essential to tailor management strategies focusing on aggressive control of hyperglycemia and triglyceride levels to prevent complications and optimize patient outcomes.

Keywords

Diabetic Ketoacidosis, Hypertriglyceridemia, Pancreatitis, Insulin Therapy, Fenofibrate

1. Introduction

Diabetic ketoacidosis (DKA) is a serious complication of diabetes characterized by hyperglycemia, metabolic acidosis, and elevated ketone levels. It is commonly seen in patients with type 1 diabetes but can also occur in individuals with type 2 diabetes under certain conditions. [1] In recent years, increasing attention has been directed toward understanding the interplay between DKA and other metabolic complications, particularly hypertriglyceridemia and acute pancreatitis, as these associations are often under-recognized in clinical practice. The co-occurrence of these conditions can present significant diagnostic and therapeutic challenges.

DKA can cause hypertriglyceridemia due to increased lipolysis and release of free fatty acids converted into triglycerides. [2] Elevated triglyceride levels, exacerbated during DKA by increased lipolysis, can directly contribute to pancreatitis by promoting fat accumulation in pancreatic cells. Previous studies estimate that approximately 7% of acute pancreatitis cases are attributed to hypertriglyceridemia as a secondary cause. [3] Moreover, emerging evidence highlights the intricate bidirectional relationship between these conditions: DKA can predispose patients to hypertriglyceridemia and pancreatitis, while severe hypertriglyceridemia can worsen insulin resistance, potentially triggering or exacerbating DKA. [4]

Although the associations between these conditions are well-documented, the literature provides limited in-depth analysis of the temporal dynamics of how they interact. A better understanding of these interactions could significantly enhance clinicians' ability to diagnose and manage these conditions effectively, optimizing treatment strategies and improving patient outcomes. [4] [5] This case report presents a unique instance of the triad of DKA, hypertriglyceridemia, and pancreatitis, offering insights into their intricate interrelationships and management approaches.

2. Case Presentation

A 28-year-old female presented to the emergency department with symptoms of polyuria, polydipsia, polyphagia, and unintentional weight loss over the past few weeks. She had a significant history of feeling unwell for the past year, gaining approximately 20 pounds, and recently losing weight due to her catabolic state. Her past medical history included depression and an appendectomy, and her home medications included Venlafaxine, Aripiprazole, and Pantoprazole. She reported occasional cigarette smoking and occasional use of THC and alcohol but denied significant chronic alcohol use. Her family history was notable for type 2

diabetes in her mother.

Upon admission, her laboratory results were significant for severe hyperglycemia, with a random serum glucose on admission of 22.9 mmol/L (412 mg/dL), hemoglobin A1c (HbA1c) of 14.5%, beta-hydroxybutyrate (BHB) level of 6.75 mmol/L, and a pH of 7.15, consistent with DKA. Additionally, she had markedly elevated triglycerides at 45 mmol/L (4000 mg/dL) and lipase at 2928 U/L. A CT abdomen revealed mild pancreatitis, likely secondary to hypertriglyceridemia, and an abdominal ultrasound showed no evidence of kidney stones with a normal common bile duct size.

Serial laboratory monitoring demonstrated a significant improvement in both triglyceride and lipase levels throughout the treatment course. Triglyceride levels decreased from 45 mmol/L (4000 mg/dL) at admission to 5.5 mmol/L (487 mg/dL) by day 5, while lipase levels showed a corresponding reduction from 2928 U/L on admission to 134 U/L by day 5. This gradual normalization of lipase levels mirrored the resolution of acute pancreatitis (refer to Figure 1 for detailed trends in lipase and triglyceride levels on admission and throughout hospitalization). These trends highlight the success of the acute management strategy, which included intravenous insulin therapy, aggressive fluid resuscitation, and meticulous electrolyte correction. While Fenofibrate therapy was introduced for long-term triglyceride management, the immediate focus remained on correcting the metabolic disturbances associated with hypertriglyceridemia, DKA, and acute pancreatitis.

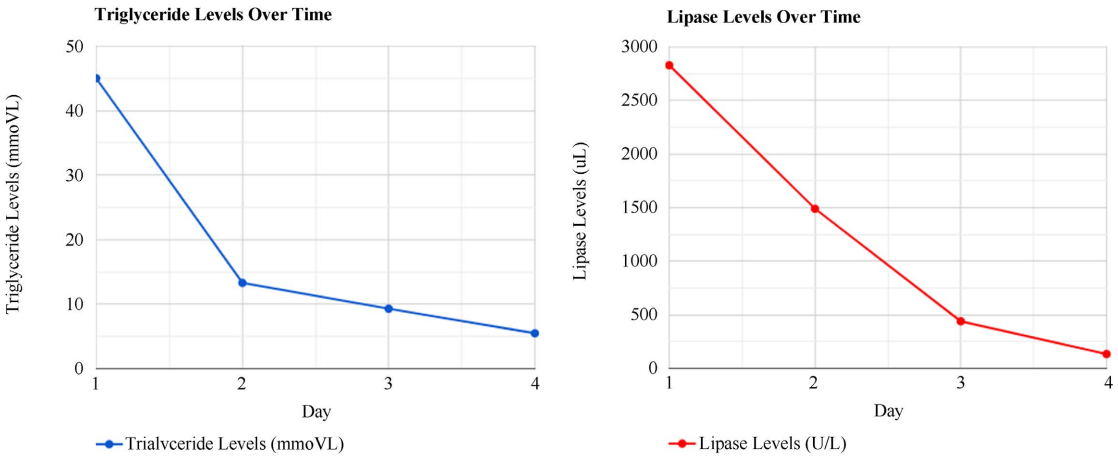

Figure 1. Trend of lipase and triglyceride levels during treatment.

3. Investigations

Initial laboratory investigations on admission revealed significant abnormalities. As mentioned previously, the patient's glucose level was markedly elevated at 25 mmol/L (450 mg/dL). Hemoglobin A1c (HbA1c) was 14.5%, indicating chronic hyperglycemia. Beta-hydroxybutyrate (BHOB) was 6.75 mmol/L (pending during initial examination), suggestive of significant ketonemia. Arterial blood gas analysis showed a pH of 7.15, a partial pressure of carbon dioxide (pCO2) of 23.8 mmHg, and a bicarbonate (HCO3) at 8.3 mmol/L consistent with metabolic acidosis.

Electrolyte imbalances included sodium at 125 mmol/L, potassium at 4.2 mmol/L, chloride at 84 mmol/L, and total carbon dioxide at 11 mmol/L. Additional laboratory findings included urea at 3.3 mmol/L (92.42 mg/d), creatinine at 87 μmol/L (0.98 mg/dL), and plasma lactate at 1.2 mmol/L. The anion gap was significantly elevated at 30 mmol/L, supporting the diagnosis of DKA.

As mentioned previously, the triglycerides were markedly elevated on admission at 45 mmol/L (4000 mg/dL) with a lipase 2928 U/L. Urinalysis showed 4+ ketones, negative nitrite, trace leukocytes, and a negative beta HCG. Given the patient presented with abdominal pain, a CT abdomen confirmed mild pancreatitis, and an ultrasound of the abdomen showed no evidence of kidney stones with a normal common bile duct size (refer to Table 1 for detailed laboratory and metabolic panel results on admission).

Table 1. Admission laboratory values and metabolic panel.

Laboratory Parameter	Upon Admission Results
Glucose	22.9 mmol/L (412 mg/dL) (4 - 11 mmo/L)
Hemoglobin A1c (HbA1c)	14.5% (4.8% - 5.9%)
Beta-hydroxybutyrate (BHOB)	6.75 mmol/L (<0.27 mmol/L)
Venous blood gas—pH	7.15 (7.33 - 7.46)
Venous blood gas—Partial Pressure of CO_2 (pCO_2)	23.8 mmHg (40 - 50 mmHg)
Venous blood gas—Bicarbonate (HCO_3)	8.3 mmol/L (22 - 27 mmol/L)
Sodium	125 mmol/L (136 - 144 mmol/L)
Potassium	4.2 mmol/L (3.5 - 5.1 mmol/L)
Chloride	84 mmol/L (98 - 107 mmol/L)
Total Carbon Dioxide (CO_2)	11 mmol/L (19 - 30 mmol/L)
Anion Gap	30 mmol/L (8 - 16 mmol/L)
Urea	3.3 mmol/L (92.42 mg/dL) (2.4 - 6.4 mmol/L)
Creatinine	87 μmol/L (0.98 mg/dL) (49 - 84 mmol/L)
Lipase	2928 U/L (14 - 85 U/L)
Plasma Lactate	1.2 mmol/L (0.7 - 2.1 mmol/L)
Triglycerides	45 mmol/L (4000 mg/dL) (<1.8 mmol/L)

4. Treatment

Upon admission to the ICU on admission day 1, the patient was treated according to the hospital's DKA protocol, which included intravenous insulin therapy, aggressive fluid resuscitation, and electrolyte correction to address severe hyperglycemia and metabolic derangements. Initial laboratory investigations revealed lipase at 2828 U/L and triglycerides at 45 mmol/L (4000 mg/dL), confirming acute pancreatitis secondary to severe hypertriglyceridemia. Insulin therapy was prioritized

to manage both hyperglycemia and hypertriglyceridemia, as insulin promotes triglyceride clearance by activating lipoprotein lipase.

By admission day 2, after 24 hours of intensive insulin therapy and fluid resuscitation, her triglyceride levels dropped markedly to 13.3 mmol/L (1176 mg/dL), and lipase decreased to 1489 U/L, suggesting partial resolution of hypertriglyceridemia and pancreatitis. Her management was transitioned to subcutaneous basal-bolus insulin therapy consisting of insulin glargine at bedtime and insulin aspart with meals as her DKA had resolved.

On admission day 3, with further improvement in laboratory parameters, her lipase dropped to 440 U/L, and triglyceride levels fell to 9.3 mmol/L (824 mg/dL). At this point, Fenofibrate was initiated to target hypertriglyceridemia directly and prevent recurrence. Her clinical condition continued to stabilize, and by admission day 4, her lipase had declined further to 134 U/L, and triglyceride levels were at 5.5 mmol/L (487 mg/dL). Given her elevated hemoglobin A1c of 14.5%, lack of a history of primary hypertriglyceridemia, and initial presentation with diabetic ketoacidosis (DKA), it was hypothesized that uncontrolled diabetes led to the onset of DKA. This, in turn, exacerbated hypertriglyceridemia, which likely contributed to the development of pancreatitis.

By admission day 4, the patient's clinical stability allowed her to transfer to the medical floor for further optimization of her insulin regimen. Normal results from a 24-hour urinary free cortisol test ruled out initial concerns of Cushing's syndrome due to central obesity. During her medical floor stay, her insulin glargine and aspart doses were titrated, and plans for dietitian consultation and diabetes education were arranged. At discharge, the patient remained on Fenofibrate to manage hypertriglyceridemia and her basal-bolus insulin regimen. She was provided detailed instructions for basal insulin titration and scheduled for a follow-up visit at the diabetes clinic two weeks post-discharge.

Notes from her follow-up in the hospital diabetes clinic two weeks post-discharge indicated that despite central obesity and negative anti-GAD antibodies, the most probable diagnosis remained type 1 diabetes. With risk factors for ongoing severe insulin deficiency, including family history, age at presentation, and marked hyperglycemia at her presentation for DKA, the patient was counselled to continue insulin therapy indefinitely. She remained on her basal/bolus insulin regimen and Fenofibrate, with plans for ongoing follow-up at the diabetes clinic.

At six months, her HbA1c had improved significantly from 14.5% at admission to 8.4%, reflecting better glycemic control on her discharged regimen. Her triglyceride levels at six months were not available during this visit but were being followed up by her primary care physician.

5. Discussion

Hypertriglyceridemia plays a pivotal role in the pathogenesis of acute pancreatitis, particularly when serum levels surpass 11.2 mmol/L (1000 mg/dl). [6] The elevated triglycerides undergo hydrolysis by pancreatic lipase into free fatty acids,

which form cytotoxic complexes with calcium ions, precipitating direct pancreatic injury through lipotoxicity, necrosis, and inflammation. This process is compounded by endothelial dysfunction, microvascular thrombosis, and ischemia, which further contribute to pancreatic injury. [7] Additionally, it is postulated that profound insulin deficiency exacerbates hypertriglyceridemia by upregulating lipolysis in adipocytes and suppressing lipoprotein lipase (LPL) activity. LPL normally hydrolyzes triglycerides in circulating chylomicrons and very low-density lipoproteins (VLDL); its suppression results in severe hypertriglyceridemia, amplifying the risk of acute pancreatitis. This interdependence underscores the pathophysiological synergy between DKA and hypertriglyceridemia, which potentiates pancreatic injury. [7] [8]

The convergence of DKA, AP, and hypertriglyceridemia represents a clinically challenging triad characterized by its rarity and significant morbidity and mortality. [9] A key challenge lies in delineating the primary driver of this interplay. Hyperglycemia and ketonemia in DKA trigger lipolysis, increasing plasma free fatty acids and hepatic VLDL production. The resultant hypertriglyceridemia further predisposes patients to acute pancreatitis through lipotoxic mechanisms. Conversely, acute pancreatitis-induced stress and systemic inflammation can provoke counter-regulatory hormone release, including catecholamines, glucagon, and cortisol, which collectively impair insulin action, precipitating DKA. This bidirectional relationship creates a vicious cycle where each condition worsens the other. [8]

The rationale for treatment in such cases involves addressing all components of the triad. Aggressive insulin therapy is pivotal for controlling hyperglycemia, halting lipolysis, and reducing triglyceride levels. In parallel, intravenous fluids correct dehydration and metabolic acidosis, improving pancreatic perfusion. For severe hypertriglyceridemia, plasmapheresis or therapeutic plasma exchange may be considered, particularly when triglyceride levels exceed 1000 mg/dL and are associated with ongoing pancreatitis. However, timely access to these therapies and the associated costs may limit their widespread administration. [10]

Determining whether DKA triggered hypertriglyceridemia leading to pancreatitis or if hypertriglyceridemia-induced pancreatitis precipitated DKA requires a careful evaluation of the clinical timeline. A key factor is the sequence of symptom onset and laboratory results. Whether hyperglycemia and ketonemia are present before abdominal pain and elevated pancreatic enzymes may help suggest that DKA was the primary event, which then led to hypertriglyceridemia and subsequent pancreatitis. On the other hand, if the patient initially presents with symptoms of acute pancreatitis (e.g., severe abdominal pain, nausea, vomiting) followed by hyperglycemia and ketonemia, hypertriglyceridemia-induced pancreatitis may be considered the primary trigger for DKA. [11] Additionally, the typical progression of these conditions can offer clues. DKA often develops rapidly within hours to days, whereas hypertriglyceridemia-induced pancreatitis may have a variable onset depending on the severity. [12] An elevated HbA1c further supports

DKA as the primary driver, reflecting a prolonged period of poorly controlled hyperglycemia. A thorough review of the patient's diabetes history, lipid levels, and prior episodes of DKA or pancreatitis is essential for establishing a clear timeline and identifying the primary precipitant. Importantly, these conditions can also have a bidirectional relationship, where one exacerbates the other, creating a vicious cycle that complicates management. [13]

In this particular case, our patient's presentation suggests a complex interplay of conditions, with DKA likely being the primary precipitant. As stated above, her symptoms of polyuria, polydipsia, polyphagia, and unintentional weight loss, along with severe hyperglycemia (HbA1c of 14.5%) and significant ketonemia (BHOB of 6.75 mmol/L), are hallmark features of DKA. The markedly elevated triglycerides of 45 mmol/L (4000 mg/dL) suggest secondary hypertriglyceridemia, which is a common complication of DKA due to increased lipolysis and free fatty acid conversion. This hypertriglyceridemia likely led to the development of mild pancreatitis, as confirmed by the elevated lipase levels and CT findings. To rule out other causes of pancreatitis, a review of her lab results showed no overt abnormalities. Her home medications of venlafaxine, aripiprazole, and pantoprazole were evaluated using the Naranjo Adverse Drug Reaction Probability Scale. [14] The scores indicated that these medications were unlikely to have significantly contributed to her pancreatitis, pointing instead to hypertriglyceridemia as the primary cause. Therefore, the sequence appears to be DKA causing hypertriglyceridemia, which subsequently induces pancreatitis. However, the bidirectional relationship means that while DKA initiated this cascade, the resultant hypertriglyceridemia and pancreatitis could further exacerbate her metabolic derangements, creating a cyclical worsening of her condition.

Our case reports have also documented managing this complex interplay between DKA, hypertriglyceridemia, and pancreatitis. [15]-[17] In all of these cases, prompt diagnosis and comprehensive management, including CT scans and lipid panels, were critical in achieving successful outcomes. This triad, while rare, underscores the necessity for heightened clinical suspicion and tailored therapeutic approaches to effectively manage these interconnected conditions.

There are limitations to this case report; most notably, the retrospective nature of the study may have impacted the thorough identification, reporting, and documentation of the patient's medical chart. The lack of genetic testing or a more comprehensive family history limits the understanding of potential hereditary contributions to the patient's condition. In addition, not all potential contributing factors (such as detailed lipid panel, autoantibodies, or inflammatory markers) were evaluated, potentially overlooking other underlying conditions. Finally, the documented short follow-up period post-discharge limits the assessment of long-term management efficacy and patient outcomes, including potential recurrences of DKA or pancreatitis.

This case underscores the critical importance of recognizing intricate bidirectional relationships: DKA can induce hypertriglyceridemia, leading to pancreatitis,

and severe hypertriglyceridemia can exacerbate DKA. Identifying the primary cause through detailed clinical evaluation is crucial for developing tailored management strategies, which should focus on aggressive control of hyperglycemia, ketosis, and triglyceride levels.

6. Conclusion

In summary, this case highlights the intricate relationship involving DKA, hypertriglyceridemia, and pancreatitis. The potential bidirectional exacerbation between DKA and hypertriglyceridemia emphasizes the necessity for a thorough clinical assessment to identify the underlying precipitant. Accurate identification is crucial for tailored therapeutic strategies, emphasizing comprehensive monitoring and long-term management to prevent recurrence and enhance patient outcomes.

Ethical Approval

Ethical approval for the publication of this case report was obtained in accordance with the hospital's guidelines for ethical conduct in case reporting.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of Supporting Data

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Authors' Contributions

All authors were involved in the conception and design of the article. The primary author drafted the initial manuscript, while the co-authors provided critical revisions for significant intellectual content. All authors reviewed and approved the final version to be published and accepted responsibility for all aspects of the work.

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Conflicts of Interest

The authors declare that they have no competing interests.

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