CASE REPORT

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Mixed Hyperglycemic Crisis in a Young Obese Diabetic Triggered by Hypertriglyceridemia-Induced Pancreatitis: A Case Report and Review of Pathophysiology

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ABSTRACT

Mixed diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS), accompanied by hypertriglyceridemia-induced pancreatitis, represent a rare but life-threatening complication of type 2 diabetes mellitus (T2DM). This case report aimed to illustrate a young adult in whom these three critical conditions converged, highlighting the complexity of such presentations—a 28-year-old male presented with altered consciousness and Kussmaul respiration. The patient was diagnosed with T2DM two weeks earlier but had not yet initiated treatment. Physical examination revealed obesity (BMI: 31 kg/m^2) and acanthosis nigricans on the neck and in the axillary regions. Laboratory results showed hyperglycemia (798 mg/dL), metabolic acidosis (pH: 7.08; anion gap: 24), ketonuria, hyperosmolarity (336 mOsm/kg), severe hypertriglyceridemia (965 mg/dL), and elevated lipase (892 U/L). A diagnosis of mixed DKA-HHS secondary to hypertriglyceridemic pancreatitis was established. Treatment included aggressive intravenous (IV) fluid resuscitation of 0.9% sodium chloride (6 L in the first 12 hours) and insulin infusion (0.1 units/kg/hour). During hospitalization, the patient developed acute kidney injury, necessitating continuous renal replacement therapy (CRRT). The patient gradually recovered and was discharged after 20 days. In obese T2DM patients, insulin resistance drives severe hyperglycemia typical of HHS. However, metabolic stress caused by acute pancreatitis induces relative insulin deficiency, triggering lipolysis, ketogenesis, and hypertriglyceridemia, leading to overlapping DKA. Severe hypertriglyceridemia exacerbates systemic inflammation, insulin resistance, and ketosis, creating a vicious cycle that worsens mixed DKA-HHS. This case report highlights the importance of recognizing that T2DM can occasionally present with atypical, life-threatening metabolic complications, necessitating prompt diagnosis and multidisciplinary management.

Keywords: Young obese diabetes, diabetic ketoacidosis, hyperglycemic hyperosmolar state, hypertriglyceridemia, pancreatitis

INTRODUCTION

The global prevalence of early-onset type 2 diabetes mellitus (T2DM) typically defined as diagnosis before age 40 is rising rapidly, particularly among those with obesity and metabolic syndrome.[1,2] Between 2013 and 2021, the number of individuals aged 20–39 years living with T2DM increased from 63 million to 260 million worldwide.[1] In early-onset T2DM, insulin resistance and beta-cell dysfunction can evolve rapidly, predisposing to acute metabolic decompensations.[3,4] Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) represent two major acute hyperglycemic crises associated with diabetes, both characterized by severe hyperglycemia but differing in the degree of insulin deficiency, presence of ketosis, and plasma osmolality.[5,6] While DKA typically occurs in the context of absolute or severe relative insulin deficiency leading to ketogenesis and metabolic acidosis, HHS arises from profound insulin resistance that is sufficient to suppress lipolysis but not adequate to prevent extreme hyperglycemia and hyperosmolarity.[7,8] Although classically described as distinct entities, a mixed DKA-HHS presentation may occur, particularly in patients with underlying T2DM and superimposed metabolic stress, and is associated with higher morbidity and mortality compared to isolated presentations.[9]

One such precipitating factor is acute pancreatitis, which is itself a rare but recognized complication of uncontrolled diabetes.[10] Among the various etiologies of pancreatitis, hypertriglyceridemia-induced pancreatitis (HTG-AP) accounts for up to 10% of cases and often manifests when serum triglyceride concentrations exceed 1,000 mg/dL.[11] The pathogenesis involves the hydrolysis of excess circulating triglycerides by pancreatic lipase into toxic free fatty acids, which induce direct pancreatic injury, capillary leakage, and systemic inflammation.[12] In patients with poorly controlled diabetes, insulin resistance promotes increased hepatic very-low-density lipoprotein (VLDL) production and reduced lipoprotein lipase activity, further accelerating triglyceride accumulation.[13,14] Although the patient denied a family history of diabetes or dyslipidemia, genetic predisposition cannot be excluded. Early-onset T2DM is often associated with genetic variants that impair β-cell function and insulin signaling, such as mutations in *TCF7L2*, *KCNJ11*, or *HNF1A*. Similarly, familial combined hyperlipidemia and lipoprotein lipase (LPL) gene polymorphisms may predispose individuals to severe hypertriglyceridemia and pancreatitis under metabolic stress. These factors could partly explain the severity of metabolic derangements in this young patient despite the short history of diagnosed diabetes.[15] The rare coexistence of HTG-AP triggering a DKA and HHS in a young adult with newly diagnosed type 2 diabetes mellitus highlights a rare and clinically significant metabolic presentation. The inflammatory response triggered by HTG-AP not only worsens glycemic control through elevated counter-regulatory hormones—such as cortisol, glucagon, and catecholamines—but also further impairs insulin secretion.[16–20] As a result, a vicious cycle is initiated in which hyperglycemia, ketosis, acidosis, and hyperosmolarity co-exist, resulting in a mixed DKA-HHS state. This triad is rarely reported and poses a high risk of multiorgan dysfunction, underscoring the importance of early recognition and prompt, multidisciplinary intervention. This case report aimed to describe a 28-year-old male with recently diagnosed but untreated T2DM who presented with overlapping DKA and HHS precipitated by hypertriglyceridemia-induced acute pancreatitis.

CASE DESCRIPTION

A 28-year-old male presented to the emergency department with decreased consciousness and deep, labored breathing

consistent with Kussmaul respiration. According to information provided by family members upon arrival, the patient had been experiencing progressive fatigue, decreased oral intake, excessive thirst, and frequent urination over the past several days. The patient had been diagnosed with type 2 diabetes mellitus two weeks before presentation but had not initiated pharmacological therapy. The patient denied any prior history of chronic illness and, when conscious, reported no use of alcohol, recent trauma, or intake of medications or herbal products. There was no known family history of pancreatitis, hyperlipidemia, or early-onset diabetes.

On initial physical examination, the patient appeared somnolent but arousable, with vital signs revealing tachycardia (heart rate: 122 bpm) and hypotension (blood pressure: 88/56 mmHg). The respiratory rate was 30 breaths/min with signs of respiratory compensation. Central obesity was evident, with a body mass index of 31 kg/m² and a waist circumference of 112 cm. Dermatological examination revealed extensive acanthosis nigricans involving the posterior neck and bilateral axillary regions.

Initial laboratory investigations demonstrated increased hyperglycemia (798 mg/dL), severe metabolic acidosis (arterial pH: 7.08; bicarbonate: 9 mmol/L; anion gap: 24), and hyperosmolarity (serum osmolality: 336 mOsm/kg). Serum triglyceride level was markedly elevated at 965 mg/dL, and serum lipase increased to 892 U/L, supporting the diagnosis of acute pancreatitis. Urinalysis showed ketonuria and glucosuria. Based on these findings, the patient was diagnosed with a mixed presentation of DKA and HHS complicated by HTG-AP.

Management was initiated with aggressive intravenous (IV) fluid resuscitation, starting with 0.9% sodium chloride at 1 L/hour for the first few hours, followed by volume titration according to hemodynamic status, with a total of 10 L administered within the first 24 hours. A continuous intravenous infusion of regular insulin was initiated at a rate of 0.1 units/kg/hour. Subsequently, subcutaneous basal insulin was added at a dose of 0.3 units/kg body weight. The total amount of insulin administered until resolution was 224 units. Empiric antibiotic therapy with IV ceftriaxone 2 g once daily was administered due to clinical suspicion of possible pancreatic necrosis.

On the third day of hospitalization, the patient developed acute kidney injury characterized by persistent anuria, reduced estimated glomerular filtration rate (eGFR), and progressive elevation in serum creatinine levels. Subsequently, continuous renal replacement therapy (CRRT) was initiated. Renal function gradually recovered with supportive management. The patient showed progressive metabolic stabilization, resolution of acidosis, and improvement in pancreatic enzyme levels. After 20 days of hospitalization, the patient was discharged in stable condition with instructions for outpatient diabetes management and follow-up with endocrinology.

DISCUSSION

The coexistence of DKA, HHS, and HTG-AP represents a complex and rare metabolic interplay often referred to as an “enigmatic triangle.” Although each condition may occur independently, their convergence—particularly as the initial manifestation of T2DM in a young adult—is exceptionally rare and associated with significant clinical implications. The convergence of DKA, HHS, and HTG-AP represents a pathophysiological continuum driven by profound insulin deficiency, dysregulated lipid metabolism, and systemic inflammation.[15–21] Recognizing the bidirectional associations among these conditions is critical for timely diagnosis and management, particularly in young patients with previously undiagnosed or untreated diabetes.

In young individuals with undiagnosed T2DM, this triad can occur when prolonged insulin resistance—often asymptomatic—is compounded by sudden metabolic stress.[22] In youth and young adults, insulin resistance often appears first, but a swift decline in beta-cell function is a key factor in the rapid worsening of glucose control.[16,17,22] Central obesity, as observed in this patient, enhances insulin resistance, increasing the risk of severe hyperglycemia and dyslipidemia even at an early age.[23] Once beta-cell function declines, the system becomes acutely vulnerable to tipping into ketosis and metabolic crisis, particularly when challenged by triggers such as pancreatitis or infection.[24] The additive effect of obesity, delayed diagnosis, and lack of prior treatment likely contributed to the severity of the presentation.

In the present case, a young patient with T2DM presented with features indicative of a mixed DKA-HHS state. Like previous observations in type 1 diabetes, the overlap between these two metabolic emergencies may be exacerbated by the administration of glucose-containing fluids during early resuscitation, which can worsen hyperglycemia and serum osmolality.[25] Such management strategies, although intended to address dehydration, may inadvertently shift the clinical picture toward that of HHS.[26]

The diagnosis of type 2 diabetes in this case was based on the patient's obesity, presence of acanthosis nigricans, and absence of autoimmune history. However, given acute presentation with ketosis, differential diagnoses such as ketosis-prone type 2 diabetes or latent autoimmune diabetes in adults should be considered. In such cases, measurement of pancreatic autoantibodies and C-peptide levels during follow-up is essential for confirming diabetes classification and optimizing long-term management.[27]

Furthermore, in patients with prolonged and severe dehydration—as observed in this case—significant metabolic acidosis may also occur due to lactic acidosis, secondary to sustained tissue hypoperfusion.[26] This lactic acidosis can contribute to the overall acid-base disturbance, compounding the ketone-driven acidosis characteristic of DKA.[26] However, in the present case, serum lactate levels were low, suggesting that lactic acidosis was not the primary driver of metabolic acidosis. This finding supports the hypothesis that other mechanisms, such as alterations in glucose metabolism and impaired cellular oxygen utilization, may contribute to the acid-base imbalance in mixed DKA-HHS presentations.[26]

In the present case, the patient had high-anion gap metabolic acidosis, as indicated by low serum bicarbonate and elevated anion gap—findings consistent with DKA. However, the history of reduced oral intake suggests a potential contribution from starvation ketosis. This condition typically arises during prolonged fasting and is associated with mild to moderate hyperglycemia and bicarbonate levels below 18 mEq/L.[26] In contrast, DKA is characterized by more severe hyperglycemia, often exceeding 250 mg/dL.[26] Although the patient's serum glucose met the DKA threshold, the acid-base disturbance may represent a mixed picture. Alcoholic ketoacidosis, another differential diagnosis, is less likely given the absence of alcohol use and typically higher serum bicarbonate levels.

The interrelation begins with insulin deficiency, a hallmark of DKA, which promotes unrestrained lipolysis in adipose tissue.[20] This process results in a surge of free fatty acids (FFAs) transported to the liver, stimulating hepatic production of VLDL.[20] Concurrently, insulin deficiency suppresses LPL activity in peripheral tissues, impairing the clearance of circulating triglycerides.[20] The net effect is the

development of severe hypertriglyceridemia, which may surpass 1,000 mg/dL and is sufficient to precipitate acute pancreatitis in susceptible individuals.[11]

The pathophysiology of HTG-AP is multifaceted. Pancreatic lipases hydrolyze excess circulating triglycerides into FFAs.[28,29] When FFA concentrations exceed the buffering capacity of plasma albumin, they self-aggregate into toxic micellar complexes that damage pancreatic acinar cells, vascular endothelium, and platelets.[29] This damage results in pancreatic ischemia, acidosis, and local inflammation, all of which accelerate trypsinogen activation and pancreatic autodigestion.[29] Additionally, hypertriglyceridemia increases serum viscosity, further impairing pancreatic perfusion and amplifying local ischemia.[29]

Conversely, acute pancreatitis can also initiate or worsen DKA and HHS. Pancreatic inflammation induces acute beta-cell dysfunction, causing transient insulin deficiency, which may be sufficient to trigger DKA in patients with underlying glucose intolerance.[9] Furthermore, pancreatitis generates a robust systemic inflammatory response characterized by the release of stress hormones—cortisol, catecholamines, glucagon, and growth hormone—that exacerbate insulin resistance.[9] These hormonal changes not only impair glucose utilization and worsen hyperglycemia but also promote hepatic ketogenesis and dehydration, culminating in the overlapping features of DKA and HHS.

The temporal sequence between these entities is often challenging to establish. Some studies suggest DKA may precede pancreatitis through the pathway of DKA → HTG → AP, while others support the reverse sequence of HTG → AP → DKA, especially when pancreatitis is the initial event in a previously undiagnosed diabetic patient.[30–33] In the present case, the elevated HbA1c supports longstanding, unrecognized hyperglycemia, suggesting that insulin deficiency and hyperglycemia had progressed before the acute event. Thus, it is plausible that the patient developed DKA first, which in turn led to HTG and subsequent pancreatitis. However, acute pancreatitis may have also acted as a precipitating stressor that aggravated insulin deficiency and unmasked the underlying diabetes.

The management of hypertriglyceridemia-induced pancreatitis follows standard acute pancreatitis guidelines, emphasizing aggressive fluid resuscitation, early enteral nutrition, and pain control. According to the 2024 American College of Gastroenterology (ACG) and International Association of Pancreatology (IAP) guidelines, insulin infusion remains a practical approach to rapidly reduce triglyceride levels in diabetic patients, while plasmapheresis is reserved for refractory cases or triglycerides >2000 mg/dL with organ dysfunction.[34]

As a single case report, this study is inherently limited in its ability to establish causality or generalize findings to broader populations. The temporal relationship between diabetic ketoacidosis, hypertriglyceridemia, and acute pancreatitis could not be definitively determined, as serial measurements of insulin, C-peptide, and inflammatory markers were not performed. Moreover, serum β-hydroxybutyrate—the predominant ketone body in diabetic ketoacidosis—was not assessed, limiting the ability to characterize the degree of ketosis accurately. Genetic testing for familial dyslipidemia was also not performed, which may have provided insight into the underlying predisposition to severe hypertriglyceridemia in this young adult.

Another diagnostic limitation was absence of autoantibody testing (e.g., GAD65, IA-2, ZnT8) and C-peptide follow-up to differentiate between ketosis-prone type 2 diabetes (Flatbush diabetes) and atypical type 1 diabetes. This distinction is fundamental in young adults presenting with mixed DKA-HHS, as overlapping clinical and

biochemical features may obscure classification. Furthermore, serum osmolality and anion gap monitoring were limited to the acute phase, restricting the ability to characterize the dynamic transition between DKA and HHS states fully.[34] Future research should focus on prospective cohort studies to better define the pathophysiological sequence linking DKA, HHS, and HTG-AP, particularly in young adults with new-onset type 2 diabetes mellitus. Investigating early biomarkers of beta-cell dysfunction and inflammatory mediators in such presentations may help identify individuals at risk for severe metabolic complications. Furthermore, clinical trials evaluating the optimal timing and role of insulin therapy, lipid-lowering agents, and renal support modalities in the management of this triad are warranted to inform evidence-based treatment strategies.

CONCLUSION

This case illustrates a rare yet life-threatening presentation of mixed DKA and HHS, precipitated by HTG-AP in a young, obese adult with newly diagnosed and untreated type 2 diabetes mellitus. The patient's age and obesity reflect a phenotype characterized by marked insulin resistance, β-cell dysfunction, and accelerated lipolysis, contributing to profound hyperglycemia, ketogenesis, and extreme hypertriglyceridemia. These metabolic derangements, compounded by the pro-inflammatory response from pancreatitis, created a vicious cycle of lipotoxicity and systemic inflammation. This case underscores the importance of recognizing the evolving epidemiology and atypical presentations of type 2 diabetes in younger populations. Clinicians should maintain a high index of suspicion for mixed metabolic emergencies in obese individuals presenting with severe hyperglycemia and abdominal symptoms to enable timely diagnosis and coordinated multidisciplinary intervention.

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