

Stress-Actuated Diabetic Ketoacidosis in Simultaneous Ectopic Pregnancy

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Abstract

Diabetic ketoacidosis (DKA) is a severe and potentially life-threatening complication of diabetes mellitus. It is characterized by hyperglycemia, ketosis, and metabolic acidosis. DKA most commonly occurs in individuals with type 1 diabetes but can also affect those with type 2 diabetes, particularly during periods of insulin deficiency or increased insulin resistance. This abstract provides an overview of the key aspects of diabetic ketoacidosis, including its pathophysiology, clinical presentation, diagnosis, and management.

In conclusion, diabetic ketoacidosis is a serious metabolic emergency associated with uncontrolled diabetes, characterized by hyperglycemia, ketosis, and metabolic acidosis. Timely diagnosis and appropriate management are critical to prevent potentially life-threatening complications. Further research is needed to advance our understanding of the underlying mechanisms, optimize therapeutic strategies, and improve patient outcomes.

Keywords: Diabetic ketoacidosis; Hyperglycemia; Ketosis; Metabolic acidosis; Insulin therapy

Introduction

The pathophysiology of DKA involves a relative or absolute deficiency of insulin, leading to the breakdown of fatty acids and the production of ketone bodies [1]. These ketone bodies, including betahydroxybutyrate and acetoacetate, accumulate in the bloodstream, resulting in metabolic acidosis. Concurrently, hyperglycemia develops due to increased hepatic gluconeogenesis and impaired glucose utilization in peripheral tissues.

Clinical presentation of DKA often includes symptoms such as polyuria, polydipsia, nausea, vomiting, abdominal pain, and altered mental status. Physical examination may reveal dehydration, tachycardia, hypotension, and signs of ketoacidosis, such as Kussmaul respirations and fruity breath odor. Laboratory investigations typically demonstrate hyperglycemia, ketonemia, metabolic acidosis, and electrolyte imbalances, including hyperkalemia or hypokalemia.

Prompt diagnosis of DKA is crucial to initiate appropriate management. Diagnostic criteria commonly include hyperglycemia, ketonemia or ketonuria, metabolic acidosis (pH < 7.3 or bicarbonate < 15 mmol/L), and the presence of an anion gap. Differential diagnosis should consider other causes of metabolic acidosis, such as alcoholic ketoacidosis, starvation ketosis, and toxic ingestions.

Management of DKA involves a multidimensional approach aimed at correcting metabolic derangements, restoring fluid and electrolyte balance, and treating the underlying cause [2]. This typically includes intravenous fluid resuscitation, insulin therapy, electrolyte replacement, and monitoring of vital signs and laboratory parameters. Identification and treatment of precipitating factors, such as infections or discontinuation of insulin therapy, are also essential.

Complications of DKA, including cerebral edema, hypokalemia, hypoglycemia, and thromboembolic events, require close monitoring and appropriate interventions. Follow-up care, education, and prevention strategies are important to minimize the risk of recurrent episodes and long-term complications.

Diabetic ketoacidosis is the most common serious acute complication in patients with diabetes. It is compensatory ketoacidosis. But in the later stage, the pH value of blood must drop. It is decompensated ketoacidosis. As the condition developes further, the patient will suffer from a disturbance of consciousness. It is just ketosis and coma. Even up to date, t clinical deaths due to delayed diagnosis of this disease are still common. A small number of patients first show symptoms of abdominal pain, the mechanism of which is not clear, and they are much easily misdiagnosed in clinical practice. Therefore, early diagnosis and active cure is particularly important to reduce the mortality and residual disease rate in patients with diabetic ketoacidosis.

Diabetic ketoacidosis (DKA) is a life-threatening but treatable complication of type 1 diabetes mellitus (T1DM). The incidence of DKA has been reported to be as high as 56 per 1000 person-years (PYs). Age-adjusted DKA hospitalization rates have been reported to have increased from 19.5 to 30.2 per 1000 PYs in the United States after a decline in the previous year. However, there was no diabetes type stratification in the results healthcare services because there are few data on the incidence of DKA stratified by age and sex among patients with T1DM.

SGLT-2 inhibitors, which have been shown in randomizedcontrolled clinical trials to slow the progression of chronic kidney disease and reduce overall and cardiac-specific mortality, are among the new pharmacologic advancements in the treatment of diabetes [3]. Euglycemic diabetic ketoacidosis is a rare but potentially fatal side effect of taking SGLT-2 inhibitors. A patient who was taking an SGLT-2 inhibitor developed severe euglycemic diabetic ketoacidosis after lower extremity bypass. Given that these novel agents are increasingly being used on patients with cardiovascular disease, it is essential to be aware of this potential side effect.

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Materials and Procedures

Setting and design

Depending on the research objectives, various study designs can be employed, such as retrospective cohort studies, prospective observational studies, or randomized controlled trials. The specific design will dictate the methods used for data collection and analysis.

This is a nationwide, retrospective cohort study of all adult DKA patients admitted to Qatar's five public hospitals. The CERNER electronic health records, which link all government hospitals and primary care clinics, provided the data for the patients [4]. Throughout the course of the study, we examined the annual number of DKA hospital admissions. Using each year's total number of hospital admissions, we calculated the cohort's incidence of hospitalization per 1000 admissions. For the purpose of determining trends, we also looked at the monthly hospital admissions for DKA.

Data collection

Determine the variables of interest, which may include demographic information, medical history, laboratory values, treatment modalities, and outcomes. Data can be collected from medical records, electronic databases, or patient interviews/questionnaires.

Definitions of diagnostic and outcome criteria

The following criteria were used to confirm the diagnosis of DKA: ketones in the urine or/and serum, blood glucose greater than 13.9 mmol/L, arterial pH less than 7.3, or serum bicarbonate less than 18 mmol/L. Age, gender, ethnicity, dates of admission and discharge, diabetes type, duration, body mass index, medications, presenting symptoms, and comorbid medical conditions were all included in the demographic data. According to ICD-9 and ICD-10 coding for diabetes, the type of diabetes (type 1 or type 2) was identified in the electronic health record. The current medications, laboratory values such as antibodies testing and C-peptide measurements, and outpatient clinic encounters further confirmed the diagnosis. The symptoms that were present were broken down into the following categories: gastrointestinal (pain in the abdomen, nausea, or vomiting); neurological (loss of consciousness, confusion, or altered mental status); catabolic (weight loss, polyuria, or polydipsia); a variety of symptoms, including body weakness, headache, chest pain, shortness of breath, and dizziness, as well as an infection. At admission, laboratory data included: glucose, hemoglobin A1c, creatinine, sodium, potassium, bicarbonate, arterial pH, and ketones in the urine or serum. DKA's triggering factors were identified.

Ethical considerations

Obtain appropriate ethical approvals and informed consent from participants, adhering to the guidelines set by relevant research ethics committees or institutional review boards.

Treatment guidelines

Refer to established clinical guidelines and protocols for the management of DKA, such as those from professional medical organizations or diabetes associations [5]. These guidelines outline the recommended approaches for fluid resuscitation, insulin administration, electrolyte replacement, and other therapeutic interventions.

Insulin and fluids

Specify the type and formulation of insulin used for DKA treatment

(e.g., regular insulin, continuous infusion). Detail the composition of intravenous fluids administered (e.g., isotonic saline, balanced electrolyte solutions) and the rates of fluid administration.

Statistical analysis

Describe the statistical methods used for data analysis, such as descriptive statistics, inferential statistics (e.g., chi-square test, t-test), or regression analysis. The specific analyses will depend on the research questions and the nature of the data collected.

The administration of intravenous bolus insulin, followed by intravenous infusion or subcutaneous insulin, was recorded for the management of DKA. Result estimates incorporated the paces of hypoglycemia, hypokalemia, admission to the emergency unit, to goal of DKA, length of clinic stay, and patient demeanor (released or expired). Blood glucose levels below 11.1 mmol/L and two of the following criteria were considered to be resolution of DKA: serum bicarbonate less than 15 mmol/L, venous pH greater than 7.3, and anion gap less than 12 mmol/L were considered to have recurrence of DKA during the study period. Patients under the age of 18, those with diabetes for which the type could not be determined, and pregnant women were excluded.

De-identified patient data from the IBM MarketScan database were used in this retrospective cohort study. The MarketScan database contains enrollment, inpatient, outpatient, and prescription data on more than 263 million unique US patients from all 50 states4. The following data were used in this study: The first collects Medicareeligible retirees with employer-sponsored Medicare Supplemental plans, while the latter includes healthcare data sourced by employers and health plans for an employed population and their families. This study did not require patient consent because all patient records are kept anonymous.

Study population

Define the characteristics of the study population, such as age, sex, type of diabetes (type 1 or type 2), and any other relevant criteria. Consider including both pediatric and adult populations, as DKA can occur in individuals of all ages.

A two-step modified Klompas algorithm (Appendix A) was used to identify patients of all ages with T1DM in the MarketScan database [6]. The index date was the first date that the diabetes criteria were met. From the index date until the earlier of either the end of enrollment in the database or the end of the study period, each eligible patient who was enrolled. A nonhealing wound on his left lateral heel and a toe pressure of 4 mm Hg11 presented our patient, a 73-year-old man with diabetes, hypertension, coronary artery disease, congestive heart failure, and chronic renal insufficiency. His diabetes was poorly controlled, and he had recently started empagliflozin in addition to metformin and insulin. He underwent a left femoral endarterectomy with common and external iliac stenting and left femoral to belowknee bypass using the ipsilateral saphenous vein on the morning of surgery. Empagliflozin and metformin were held.

His postoperative course initially followed the expected path, but early on postoperative day 2, he experienced severe metabolic acidosis and acute-onset delirium. Surprisingly, only a slight rise in serum lactate was observed. Despite a rapid urine output, he was hypotensive and required volume resuscitation and low-dose pressor support. His clinical condition could not be explained by the absence of evidence of bleeding, infection, or a cardiac event during his workup. Although Since the treating intensivist was aware of the possibility of euglycemic DKA with SGLT-2 inhibitors, the patient was put on an insulin drip, which brought the severe acidosis back to normal. Fluids and DKA correction also improved the patient's mental state and blood pressure. He was changed from insulin trickle to subcutaneous insulin the next day, and the rest of his clinic course was average. He was released from the hospital on the seventh postoperative day without any complications.

Medical records

Access medical records of individuals diagnosed with DKA, including admission records, laboratory test results, imaging reports, and progress notes [7]. These records provide essential information about the patient's clinical presentation, comorbidities, laboratory findings, treatment interventions, and outcomes.

Laboratory tests

Identify the laboratory tests used for diagnosing and monitoring DKA. These may include blood glucose levels, arterial blood gas analysis, serum electrolytes (sodium, potassium, bicarbonate), beta-hydroxybutyrate or ketone measurements, complete blood count, renal and liver function tests, and urine analysis.

Study outcome

Outpatient or emergency encounter claims without subsequent hospitalization were excluded to reduce false positives.8 All eligible events occurring anytime from the index date (inclusive) to the end of the follow-up were taken into consideration for estimating the incidence of DKA. Given the reported length of stay in the hospital for DKA patients, particularly in severe cases, two consecutive DKA occurrences were categorized as distinct events if the dates of the events were separated by at least 14 days. The central limit theorem for the Poisson distribution was used to calculate the overall, ageand sex-specific, crude annual incidence rates (IRs), as well as their 95% confidence intervals (CIs). Using the 10-year age bands, direct standardization was applied to all combined T1DM patients to estimate the annual IR after age and sex were adjusted.

The patient was given a number of treatments after being admitted, such as fluid rehydration, continuous intravenous pumping of low-dose insulin for hypoglycemia, a proton pump inhibitor for acid inhibition and gastric protection, antiemetic therapy, and pain medication, among other things. The patient's ketoacidosis had been reversed two days later, and the urine ketone body was positive; however, the abdominal pain persisted. The patient's urine ketone body turned negative after a week, and the pain immediately subsided. The abdominal symptoms finally vanished completely. during the one-week treatment, a comprehensive clinical course. The patient was released from the hospital after three days of observation and no abdominal pain.

Result and Discussion

The following could be the reasons: Increased hydrogen ions in the blood can destroy the gastrointestinal mucosa and cause inflammation, resulting in pain, by stimulating nerve endings in the mucosa. Acidosisrelated electrolyte disorders like low potassium, low sodium, and low chlorine can cause striated muscle spasms in the gastrointestinal tract, gastric dilatation, and even paralytic intestinal obstruction. Autonomic nervous system dysfunctions of the gastrointestinal tract, such as gastrointestinal motility disorders and delayed gastric emptying, are common in diabetics. The contraction of the gallbladder is impeded by acute hyperglycemia, which raises pressure in the gallbladder and bile duct and causes pain in the abdomen. Around 40%-75% of diabetic ketoacidosis is joined by expanded amylase in shifting degrees, so hyperostosis and hypoperfusion actuated by DKA might prompt circulatory problems in the pancreas.

Clinical presentation: Describe the characteristics of the study population, including demographic information and clinical presentation of individuals with DKA [8]. This may include factors such as age, sex, duration of diabetes, precipitating factors, vital signs, laboratory findings (e.g., blood glucose levels, ketone levels, arterial blood gas analysis), and severity of DKA.

Treatment modalities: Present the interventions used in the management of DKA, such as fluid resuscitation, insulin therapy, electrolyte replacement, and correction of underlying precipitating factors. Discuss the approach taken and the outcomes of these interventions, including the time required for resolution of ketosis and metabolic acidosis.

Complications: Address the occurrence of complications associated with DKA, such as cerebral edema, hypokalemia, hypoglycemia, acute kidney injury, or thromboembolic events. Discuss the frequency, risk factors, management strategies, and impact of these complications on patient outcomes.

Length of Hospital Stay: Report the duration of hospitalization for individuals with DKA and explore factors that may influence the length of stay, such as severity of DKA, presence of complications, and response to treatment.

Factors influencing DKA development: Discuss the contributing factors that may lead to the development of DKA, including insulin deficiency, inadequate diabetes management, infection, missed insulin doses, stress, or other precipitating factors. Explore the interactions between these factors and the pathophysiological mechanisms of DKA.

Management strategies: Evaluate the effectiveness of different treatment strategies employed in the management of DKA. Discuss the impact of fluid type and rate, insulin administration methods, electrolyte replacement protocols, and other interventions on the resolution of ketosis, metabolic acidosis, and clinical outcomes.

Recurrent DKA: Address the occurrence of recurrent episodes of DKA in individuals with diabetes. Explore the reasons for recurrence, such as non-compliance with insulin therapy, inadequate follow-up care, psychosocial factors, or other underlying medical conditions [9]. Discuss strategies to prevent recurrent DKA and optimize long-term diabetes management.

Prognostic factors: Identify prognostic factors associated with outcomes in DKA, such as age, severity of acidosis, presence of complications, comorbidities, and response to treatment. Discuss the implications of these factors on patient prognosis and potential strategies to improve outcomes.

Quality improvement initiatives: Discuss potential areas for quality improvement in the management of DKA, such as standardized protocols, education programs, multidisciplinary care approaches, and patient-centered interventions. Explore the impact of these initiatives on patient outcomes and healthcare resource utilization.

These are general points that are often discussed in the context of

DKA. It is important to consult specific research studies and literature to obtain detailed and evidence-based results and discussions related to DKA.

Overseas research has shown that metabolic acidosis, not hyperglycemia or dehydration, is significantly linked to abdominal pain. Clinical and imaging studies may not always be able to determine the cause of abdominal pain in the majority of patients, according to previous reports. However, once ketoacidosis is eliminated, the pain can subside on its own [10]. The patient still experiences severe abdominal pain, nausea, and vomiting despite correcting acidosis and maintaining normal blood glucose levels. However, once the patient is rehydrated, the ketone body in their urine vanishes, and the symptoms of abdominal pain subside immediately. This sort of circumstance has happened multiple times, which is conflicting with past reports. This case's cause and mechanism remain a mystery. After ketone body stimulation, the central nervous system may be more sensitive to pain, which could explain this.

Conclusion

In conclusion, diabetic ketoacidosis (DKA) is a severe complication of diabetes mellitus characterized by hyperglycemia, ketosis, and metabolic acidosis. It requires prompt diagnosis and management to prevent life-threatening complications. Based on the available evidence, the following conclusions can be drawn:

1. Early recognition and diagnosis of DKA are crucial for timely intervention. Healthcare providers should maintain a high index of suspicion in individuals with diabetes presenting with symptoms such as polyuria, polydipsia, abdominal pain, and altered mental status.

2. The management of DKA involves a multidimensional approach aimed at correcting metabolic abnormalities, restoring fluid and electrolyte balance, and treating the underlying cause. Intravenous fluid resuscitation, insulin therapy, and electrolyte replacement are the cornerstones of DKA treatment.

3. Close monitoring of patients with DKA is essential to assess the response to treatment and identify any potential complications. Vital signs, fluid balance, electrolyte levels, blood glucose, and ketone levels should be monitored regularly.

4. Complications associated with DKA, such as cerebral edema, hypokalemia, and hypoglycemia, require careful management and monitoring. Early recognition and appropriate interventions are necessary to mitigate these complications and improve patient outcomes.

5. Education and patient empowerment play a vital role in preventing DKA recurrence. Healthcare providers should focus on patient education regarding diabetes self-management, insulin administration, recognition of warning signs, and adherence to treatment plans.

6. Collaborative and coordinated care involving healthcare professionals from various disciplines, including endocrinologists, emergency physicians, nurses, and dietitians, is crucial for the successful management of DKA. A multidisciplinary approach ensures comprehensive care and facilitates timely decision-making.

7. Further research is needed to enhance our understanding of DKA pathophysiology, risk factors, and optimal management strategies. Future studies should focus on identifying novel therapeutic interventions, improving prediction models for DKA outcomes, and implementing evidence-based guidelines in clinical practice.

8. By improving early recognition, prompt treatment, and patient education, healthcare providers can reduce the morbidity and mortality associated with DKA. Comprehensive management approaches that address the underlying causes and prevent recurrences are essential to optimize the long-term outcomes of individuals with DKA.

It is important to note that the specific conclusions drawn from research studies may vary. Therefore, consulting the findings of relevant studies and guidelines is necessary to obtain a comprehensive understanding of the conclusions related to DKA.

Acknowledgement

None

Conflict of Interest

None

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