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Introduction

e pathophysiology of DKA involves a relative or absolute de ciency of insulin, leading to the breakdown of fatty acids and the production of ketone bodies [1]. ese ketone bodies, including beta-hydroxybutyrate 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 o en 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. Di erential 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 uid and electrolyte balance, and treating the underlying cause [2]. is typically includes intravenous uid resuscitation, insulin therapy, electrolyte replacement, and monitoring of vital signs and laboratory parameters. Identi cation 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 su er 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 rst show symptoms of abdominal pain, the mechanism of which is not clear, and they are much easily misdiagnosed in clinical practice. erefore, 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). e 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 a er a decline in the previous year. However, there was no diabetes type strati cation in the results healthcare services because there are few data on the incidence of DKA strati ed by age and sex among patients with T1DM.

SGLT-2 inhibitors, which have been shown in randomized-controlled clinical trials to slow the progression of chronic kidney disease and reduce overall and cardiac-speci c mortality, are among the new pharmacologic advancements in the treatment of diabetes [3]. Euglycemic diabetic ketoacidosis is a rare but potentially fatal side e ect of taking SGLT-2 inhibitors. A patient who was taking an SGLT-2 inhibitor developed severe euglycemic diabetic ketoacidosis a er 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 e ect.

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his urinalysis revealed signi cant high urinary glucose and ketones, his serum glucose was relatively normal. Notably, given his normal glucose levels on the rst postoperative day, he had not received any insulin.

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]. ese records provide essential information about the patient's clinical presentation, comorbidities, laboratory ndings, treatment interventions, and outcomes.

Laboratory tests

Identify the laboratory tests used for diagnosing and monitoring DKA. ese 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. e central limit theorem for the Poisson distribution was used to calculate the overall, age- and sex-speci c, crude annual incidence rates (IRs), as well as their 95% con dence intervals (CIs). Using the 10-year age bands, direct standardization was applied to all combined T1DM patients to estimate the annual IR a er age and sex were adjusted.

e patient was given a number of treatments a er being admitted, such as uid 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. e patient's ketoacidosis had been reversed two days later, and the urine ketone body was positive; however, the abdominal pain persisted. e patient's urine ketone body turned negative a er a week, and the pain immediately subsided. e abdominal symptoms nally vanished completely. during the one-week treatment, a comprehensive clinical course. e patient was released from the hospital a er three days of observation and no abdominal pain.

Result and Discussion

e following could be the reasons: Increased hydrogen ions in the blood can destroy the gastrointestinal mucosa and cause in ammation, resulting in pain, by stimulating nerve endings in the mucosa. Acidosis-related 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. e 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 shi ing 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]. is may include factors such as age, sex, duration of diabetes, precipitating factors, vital signs, laboratory ndings (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 uid 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 in uence the length of stay, such as severity of DKA, presence of complications, and response to treatment.

Factors in uencing DKA development: Discuss the contributing factors that may lead to the development of DKA, including insulin de ciency, 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 e ectiveness of di erent treatment strategies employed in the management of DKA. Discuss the impact of uid 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.

ese are general points that are o en discussed in the context of

DKA. It is important to consult speci c 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 signi cantly 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]. e 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. is sort of circumstance has happened multiple times, which is con icting with past reports. is case's cause and mechanism remain a mystery. A er 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:

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.

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

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

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.

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.

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.

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.

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.