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A Case of Type 1 Diabetic Ketoacidosis that Resulted in Persistent Severe Abdominal Pain

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Abstract

A 31-year-old man with severe abdominal pain and type 1 diabetic ketoacidosis (DKA) is the subject of this case report. His blood glucose level was 17.58 mmol/L, his blood pH was 7.286, his urine ketone body content was 3+, and the clinical examination revealed no signs of acute abdominal diseases like pancreatitis at the time of admission. The patient's acidosis was quickly resolved with active treatment, but their abdominal pain persisted. The urinary ketone body was finally eliminated after seven days of continuous insulin pump treatment, and the abdominal pain went away as well. This demonstrates the significance of removing the ketone body from the urine. DKA with stomach torment as the primary side effect is moderately uncommon in the centre. At the moment, it is unclear how DKA causes abdominal pain, but it may be related to how ketosis metabolites stimulate the gastrointestinal tract. It's important to note that this case has recurrent episodes and persistent severe abdominal pain.

Keywords: Ketoacidosis with diabetes; Pain in the bottom; Haemoglobin A1c

Introduction

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 develops further, the patient will suffer from a disturbance of consciousness. It is just ketosis and coma [1]. 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).2 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 [2]. 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 randomized-controlled 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. 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 [3]. 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.

Materials and Procedures

Setting and design

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. Throughout the course of the study, we examined the annual number of DKA hospital admissions [4]. 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.

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 [5]. 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.

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The administration of intravenous bolus insulin, followed by intravenous infusion or subcutaneous insulin, was recorded for the management of DKA [6]. 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) [7]. Blood glucose levels 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 Medicare-eligible 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

A two-step modified Klompas algorithm (Appendix A) was used to identify patients of all ages with T1DM in the MarketScan database [8]. 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 below-knee 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 his urinalysis revealed significant high urinary glucose and ketones, his serum glucose was relatively normal. Notably, given his normal glucose levels on the first 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 [9]. 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.

Study outcome

Outpatient or emergency encounter claims without subsequent hospitalization were excluded to reduce false positives [10]. 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, age- and 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 [11]. 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.

Due to severe abdominal pain, the patient returned to the hospital one month after being discharged. In contrast to the previous admission, this patient's blood gas analysis revealed no acidosis, but the urine ketone body test came back positive. There were no signs of acute abdominal diseases during the clinical examination. The patient experienced rapid relief from abdominal pain following stable blood glucose control and the complete disappearance of the urinary ketone body [12].

Result and Discussion

Diabetic ketoacidosis is an acute diabetes complication that mostly affects people with type 1 diabetes. DKA manifests as clinical hyperglycemia, ketosis, metabolic acidosis, and dehydration due to a severe or complete lack of insulin activity and improper elevation of the glycemic hormone. These factors disrupt the metabolism of glucose, fat, and protein. Infection, trauma, surgery, pregnancy, childbirth, a poor diet, and interrupting or reducing the dose of insulin injected for treatment are all common causes of DKA. Clinical indications of DKA contain aggravation of awareness, touchiness, polydipsia, weariness, sickness, and retching. However, DKA patients occasionally only experience abdominal pain. Acute abdominal pain only occurs in 6% of DKA patients, compared to a 22.0% foreign incidence in published studies. Abdominal distention, nausea, vomiting, dehydration, confusion, and other symptoms follow the general lack of specificity regarding the location of the pain in the abdominal region. Physical examination may not always reveal abdominal tenderness or muscle tension, so the symptoms of abdominal pain are frequently inconsistent with the light signs [13]. It is still unclear how DKA causes abdominal pain at this time. 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. 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. 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 [14]. 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.

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. 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 [15]. After ketone body stimulation, the central nervous system may be more sensitive to pain, which could explain this.

Conclusion

In conclusion, this case emphasizes the significance of prompt diagnosis and treatment, particularly the removal of ketone body stimulation in DKA patients who exhibit abdominal pain as a clinical symptom. Residual ketone bodies may continue to cause severe pain even after acidosis and hyperglycemia are corrected. It shows the significance of disposing of the ketone body in the pee. This aspect is hardly mentioned in the report on the medical record. It's also important to note that this case has recurrent episodes and prolonged severe abdominal pain. In the clinic, DKA with abdominal pain as the first symptom is uncommon and frequently misdiagnosed. As a result, clinicians should pay greater attention to distinguish diabetic ketoacidosis from other forms of abdominal pain when abdominal pain is the first symptom. The diagnosis of ketoacidosis relies heavily on the presence of ketone bodies in the blood and urine. Urinalysis has gradually moved from manual testing to automated analysis as medical science and technology continue to develop. This has increased the sensitivity and precision of experimental results and provided a foundation for rapid clinical diagnosis. As a result, determining the amount of ketones in a person's body by measuring ketones in their urine rather than their blood is an easy, quick, convenient, accurate, and trustworthy method. It is suggested that urinary ketones be examined on a regular basis for quick diagnosis and active treatment of abdominal pain.

Acknowledgement

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Conflict of Interest

None

References

1. Nair S, Yadav D, Pitchumoni CS (2000) Association of diabetic ketoacidosis and acute pancreatitis: observations in 100 consecutive episodes of DKA. Am J Gastroenterol 95: 2795-800.
2. Umpierrez G, Freire AX (2002) Abdominal pain in patients with hypergly-cemic crises. J Crit Care 17: 63-67.
3. Wang Q, Wu K, Luo X, Dong X, Liu W, et al. (2022) Dapagliflozin-Associated Euglycemic Diabetic Ketoacidosis Presenting With Severe Abdominal Pain Mimicking Acute Peritonitis.
4. Cureus 14: e22229.
5. Samsom M, Akkermans LM, Jebbink RJ, Isselt HV, Henegouwen GPVB, et al. (1997) Gastrointestinal motor mechanisms in hyperglycaemia induced delayed gastric emptying in type I diabetes mellitus. Gut 40: 641-6.
6. Horowitz M, Fraser R, Dent J (1991) Hyperglycaemia stimulates pyloric motility in normal subjects. Gut 32: 475-478.
7. Mumtaz H, Shafiq MA, Batool H, Naz T, Ambreen S, et al. (2020) Diabetic Ketoacidosis in an Euglycemic Patient. Cureus 12: e10065.
8. Pant N, Kadaria D, Murillo LC, Yataco JC, Headley AS, et al. (2012) Abdominal pathology in patients with diabetes ketoacidosis. Am J Med Sci 344: 341-344.
9. Moreno EIG, Chávez JMG, González FJL, González JGG, Caballero AE, et al. (2015) Severe Ketoacidosis (pH ≤ 6.9) in Type 2 Diabetes: More Frequent and Less Ominous Than Previously Thought. Biomed Res Int 2015: 134780.
10. Duhon B, Attridge RL, Martinez ACF, Maxwell PR, Hughes DW, et al. (2013) Intravenous sodium bicarbonate therapy in severely acidotic diabetic ketoacidosis. Ann Pharmacother 47: 970-5.
11. Naing NN (2000) Easy way to learn standardization: direct and indirect methods. Malays J Med Sci 7: 10-15.
12. Hoshina S, Andersen GS, Jorgensen ME, Ridderstrale M, Vistisen D (2018) Treatment modality-dependent risk of diabetic ketoacidosis in patients with type 1 diabetes: Danish Adult Diabetes Database Study. Diabetes Technol Ther 20: 229-234.
13. Xu G, Liu B, Sun Y, Hu FB, Bao W et al. (2018) Prevalence of diagnosed type 1 and type 2 diabetes among US adults in 2016 and 2017: population based study. BMJ 362: k1497.
14. Barski L, Boehm IH, Nevzorov R, Rabaev E, Zektser M, et al. (2011) Gender-related differences in clinical characteristics and outcomes in patients with diabetic ketoacidosis. Gend Med 8: 372-7.
15. Desai D, Mehta D, Mathias P, Menon G, Schubart UK, et al. (2018) Health care utilization and burden of diabetic ketoacidosis in the U.S. over the past decade: a nationwide analysis. Diabetes Care 41: 1631-1638.
16. Ford W, Self WH, Slovis C, McNaughton CD (2013) Diabetes in the emergency department and hospital: acute care of diabetes patients. Curr Emerg Hosp Med Rep 1: 1-9.