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Case Report

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# Diabetic ketoacidosis in a pediatric patient with type 1 diabetes mellitus and the impact of insulin therapy discontinuation: a case report

*Cetoacidose diabética em paciente pediátrico com diabetes mellitus tipo 1 e o impacto da interrupção da insulinoterapia: um relato de caso*

Cetoacidosis diabética en un paciente pediátrico con diabetes mellitus tipo 1 y el impacto de la interrupción de la terapia con insulina: reporte de un caso

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**ABSTRACT**

**Introduction:** Diabetic ketoacidosis is a serious acute complication of diabetes mellitus, characterized by hyperglycemia (blood glucose > 200 mg/dL), metabolic acidosis (venous pH < 7.3 and/or bicarbonate < 15 mEq/L), ketonemia, and ketonuria. It is triggered by infections, insulin administration failures, or progressive pancreatic beta-cell failure. **Objective**: To present a case of diabetic ketoacidosis in a ten-year-old patient diagnosed with type 1 diabetes mellitus three years ago, with poor treatment adherence. **Case description:** A ten-year-old school-aged child, diagnosed with type 1 diabetes mellitus at age seven, had not used NPH insulin for one week and regular insulin for one day. The patient developed vomiting associated with nausea, dizziness, and pain in both upper and lower limbs. The diagnosis was moderate diabetic ketoacidosis, the third episode since the diabetes diagnosis. **Discussion**: The diagnosis of diabetic ketoacidosis relies not only on blood glucose but also on pH, ketonemia, and ketonuria. Classification depends on pH and clinical presentation. The pathophysiology includes insulinopenia, hyperglycemia, dehydration, ketone body production, and depletion of the bicarbonate buffer system. Clinical manifestations include polyphagia, polydipsia, polyuria, vomiting, abdominal pain, and dehydration. Treatment involves insulin therapy and hydroelectrolytic replacement, with a focus on fluid resuscitation and potassium supplementation. In this case, the patient presented vomiting, dizziness, and paresthesia in the lower limbs, with a blood glucose level of 864 mg/dL and arterial pH of 7.16.
**Keywords:** Diabetes mellitus type 1; Case report; Hyperglycemia; Diabetic ketoacidosis; Insulin therapy.

**RESUMO**

**Introdução:** A cetoacidose diabética é uma complicação aguda grave do diabetes mellitus, caracterizada por hiperglicemia (glicemia maior que 200 mg/dL), acidose metabólica (pH venoso menor que 7,3 e/ou bicarbonato menor que 15 mEq/L), cetonemia e cetonúria, desencadeada por infecções, falhas na administração da insulina e falência progressiva das células beta pancreáticas. **Objetivo:** Apresentar um caso de cetoacidose diabética em paciente de dez anos, diagnosticado com diabetes mellitus tipo 1 há três anos e com má adesão ao tratamento. **Descrição de caso:** Escolar de dez anos, com diagnóstico de diabetes mellitus tipo 1 aos sete anos, sem o uso de insulina NPH por uma semana e da insulina regular por um dia, iniciou quadro de vômito associado a náusea, tontura e dor em ambos os membros superiores e inferiores, sendo diagnosticado com cetoacidose diabética moderada, terceiro episódio desde o diagnóstico de diabetes. **Discussão:** O diagnóstico da cetoacidose diabética baseia-se não só na glicemia, mas também nos valores de pH, cetonemia e cetonúria. Já a classificação depende do pH e do quadro clínico apresentado. A fisiopatologia envolve insulinopenia, hiperglicemia, desidratação, produção de corpos cetônicos e esgotamento do sistema tampão de bases. A clínica é composta por polifagia, polidipsia, poliúria, vômitos, dor abdominal e desidratação. O tratamento consiste em insulinoterapia associada à reposição hidroeletrolítica, com ênfase em reposição volêmica e de potássio. No presente caso, o paciente apresentou vômitos, tontura e parestesia em membros inferiores, com glicemia de 864 mg/dL e pH arterial de 7,16.
**Palavras-Chave:** Diabetes mellitus tipo 1; Relato de caso; Hiperglicemia; Cetoacidose diabética; Insulinoterapia.

**RESUMEN**

**Introducción:** La cetoacidosis diabética es una complicación aguda grave de la diabetes mellitus, caracterizada por hiperglucemia (glucosa en sangre mayor de 200 mg/dL), acidosis metabólica (pH venoso menor de 7.3 y/o bicarbonato menor de 15 mEq/L), cetonemia y cetonuria, desencadenada por infecciones, fallos en la administración de insulina e insuficiencia progresiva de las células beta pancreáticas. **Objetivo**: Presentar un caso de cetoacidosis diabética en un paciente de diez años, diagnosticado con diabetes mellitus tipo 1 tres años antes y con mala adherencia al tratamiento. **Descripción del caso**: Un escolar de diez años, diagnosticado con diabetes mellitus tipo 1 a los siete años, que no había usado insulina NPH durante una semana e insulina regular durante un día, comenzó a experimentar vómitos asociados con náuseas, mareos y dolor en miembros superiores e inferiores, y fue diagnosticado con cetoacidosis diabética moderada, el tercer episodio desde el diagnóstico de diabetes. **Discusión**: El diagnóstico de cetoacidosis diabética se basa no solo en los niveles de glucosa en sangre, sino también en el pH, la cetonemia y la cetonuria. La clasificación depende del pH y la presentación clínica. La fisiopatología implica insulinopenia, hiperglucemia, deshidratación, producción de cuerpos cetónicos y depleción del sistema tampón de base. La presentación clínica consiste en polifagia, polidipsia, poliuria, vómitos, dolor abdominal y deshidratación. El tratamiento consiste en terapia con insulina combinada con reposición de líquidos y electrolitos, con énfasis en la reposición de volumen y potasio. En este caso, el paciente presentó vómitos, mareos y parestesias en las extremidades inferiores, con un nivel de glucosa en sangre de 864 mg/dL y un pH arterial de 7,16.
**Palabras Clave:** Diabetes mellitus tipo 1; Reporte de caso; Hiperglucemia; Cetoacidosis diabética; Terapia con insulina.

## INTRODUCTION

Diabetic ketoacidosis (DKA) is a serious acute complication of diabetes mellitus (DM), characterized by hyperglycemia (blood glucose greater than 200 mg/dL), metabolic acidosis (venous pH less than 7.3 and/or bicarbonate less than 15 mEq/L), ketonemia, and ketonuria. It is more frequent in patients with type 1 DM and, in many cases, may be the initial presentation of the disease. The main triggering factors are infections, failures in insulin administration, especially in adolescence, and the progressive failure of pancreatic beta cell function. Clinically, DKA manifests with characteristic signs and symptoms, including intense polydipsia, polyuria, nausea, vomiting, abdominal pain, Kussmaul breathing, ketotic breath, and altered state of consciousness, which can range from lethargy to coma.[1,2]

The management of DKA initially involves detailed medical history and clinical examination, promptly followed by complementary tests. According to the clinical picture presented, initial hydroelectrolytic replacement should be performed with 0.9% saline solution and subsequently, according to blood glucose levels, with glucose solution. In addition, potassium replacement should be performed according to serum levels and intravenous insulin therapy should be initiated.[1]

The authors report the case of a ten-year-old schoolchild with a previous diagnosis of type 1 diabetes mellitus (DM1) and clinical manifestations of diabetic ketoacidosis, diagnosed as moderate DKA. He was hospitalized and treated appropriately, evolving with clinical improvement and hospital discharge. The importance of this case report lies in the recurrence of diabetic ketoacidosis in a patient with poor adherence to treatment, which resulted in three episodes of decompensation. The persistent difficulty in following the appropriate therapeutic regimen highlights the importance of effective education and follow-up strategies, reinforcing adherence to treatment from the first months of diagnosis, avoiding serious complications such as DKA.

## OBJECTIVE

To report the case of a ten-year-old patient diagnosed with type 1 diabetes mellitus (DM1) at age seven, hospitalized with diathetic kala-acetaminophen (DKA) and with a history of two previous hospitalizations for DKA, emphasizing the current clinical evolution and the therapeutic approach adopted by the medical team, as well as the importance of health education in adherence with the aim of minimizing serious complications of DM1 such as DKA.

## CASE REPORT

A ten-year-old male patient, a student residing in the city of Petrópolis-RJ, with a previous diagnosis of type 1 diabetes for three years. The diagnosis was established after an episode of diabetic ketoacidosis that led to hospital admission. At the time, the patient presented classic symptoms of the disease, such as polyuria, polydipsia, significant weight loss, and vomiting, evolving favorably after the start of insulin therapy.

Over the following year, he showed good clinical response to treatment. However, about a year after the diagnosis, he presented a new episode of DKA, again beginning with intense vomiting and paresthesia in the lower limbs, and was promptly attended to and treated.

Approximately eight months after the second episode, the patient discontinued the use of NPH and regular insulins, according to his guardian, due to the lack of regular supply of medications by the public health system. The day after the complete discontinuation of insulin, he began experiencing symptoms like those of the previous episodes, including nausea, dizziness, vomiting, and paresthesia in the lower limbs. He was then taken to the hospital emergency service.

On admission, the patient was conscious, oriented, Glasgow Coma Scale score 15, hydrated, acyanotic, anicteric, with good peripheral capillary pressure, breathing

normally in room air, with no ketotic breath odor, and with stable vital signs. Cardiac and respiratory auscultation were normal, the abdomen was soft and non-tender. Laboratory tests revealed a blood glucose level of 864 mg/dL, arterial pH of 7.16, $pCO_2$ of 22.5 mmHg, $pO_2$ of 103.3 mmHg, bicarbonate of 9.4 mEq/L, and lactate of 49.7 mmol/L (Chart 1), confirming the diagnosis of severe DKA, although without apparent neurological impairment. In the emergency room, treatment was initiated with insulin therapy and fluid and electrolyte replacement, as recommended by the protocols for managing diabetic ketoacidosis.[3,4]

Treatment continued, and the patient was admitted to the intensive care unit, where he initially remained fasting and underwent intravenous hydration with 1:1 glucose-saline solution, potassium replacement, and continuous infusion of regular insulin at a dose of 0.1 IU/kg/h and hourly capillary blood glucose monitoring. Clinical and laboratory monitoring was continuous, with progressive evolution towards normalization of acid-base and glycemic parameters, as shown in Charts 2 and 3. After approximately nine hours of treatment, stabilization of the clinical picture was observed, with blood glucose of 99 mg/dL, pH of 7.38 (Charts 2 and 3), and vital signs within normal parameters. Complete correction of the acidosis was achieved after approximately ten hours of hospitalization, at which point the patient was transferred to the pediatric ward for clinical follow-up.

**Chart 1 -** Laboratory parameters at the time of hospital admission

| Parameters | Result |
|---|---|
| Na+ | 133 mEq/L |
| K+ | 4.1 mEq/L |
| Lactate | 49.7 mmol/L |
| Arterial pH | 7.16 |
| pCO2 | 30 mmHg |
| pO2 | 147 mmHg |
| HCO3- | 10 mEq/L |
| BE | -18 mEq/L |

**Caption:** Na+: Sodium; K+: Potassium; pCO2: Partial pressure of carbon dioxide; pO2: Partial pressure of oxygen; HCO3-: Bicarbonate; BE: Base excess.

**Chart 2 -** Laboratory parameters obtained during hospitalization in an intensive care unit

| Time | pH | pCO2 | pO2 | HCO3- | BE | Lactate |
|---|---|---|---|---|---|---|
| 20:30 | 7,22 | 22,5 mmHg | 103,3 mmHg | 9,4 mEq/L | -15,7 mEq/L | 49,7 mmol/L |
| 22:30 | 7,33 | 21,6 mmHg | 147,6 mmHg | 11,5 mEq/L | -11,6 mEq/L | 5,1 mmol/L |
| 00:30 | 7,38 | 31,1 mmHg | 113,1 mmHg | 18,5 mEq/L | -4,9 mEq/L | 2 mmol/L |
| 06:00 | 7,39 | 31,4 mmHg | 109,2 mmHg | 19,1 mEq/L | -4,2 mEq/L | 0,9 mmol/L |

**Caption: pCO2:** Partial pressure of carbon dioxide; pO2: Partial pressure of oxygen; HCO3-: Bicarbonate; BE: Base excess.

**Chart 3 –** Blood glucose levels obtained from arrival at the emergency department up to 4 hours after hospital admission

| Measurement time | In the emergency room | Upon hospital admission | 1 hour after admission | 2 hours after admission | 3 hours after admission | 4 hours after admission |
|---|---|---|---|---|---|---|
| Blood glucose | 846 mg/dL | 207 mg/dL | 99 mg/dL | 75 mg/dL | 83 mg/dL | 136 mg/dL |

During hospitalization, the patient maintained clinical stability and did not present new episodes of vomiting. The therapeutic plan included rigorous monitoring of capillary blood glucose, dietary adjustments, and reintroduction of subcutaneous insulin therapy. However, difficulty in glycemic control was observed, with marked hyperglycemia during the early morning hours and blood glucose readings of 310, 493, 435, and 465 mg/dL. These alterations were attributed to factors common in a hospital setting, such as metabolic stress caused by changes in dietary routine, poor acceptance of the offered diet, and ingestion of foods incompatible with the nutritional plan, such as the consumption of orange juice, rich in rapidly absorbed carbohydrates, during dinner.

After joint intervention between the medical team, nutrition team, and family members, it was possible to adjust the diet, re-educate the mother regarding insulin use, and re-establish glycemic control. Considering the good clinical progress and the mother's guidance regarding adherence to treatment, the patient was discharged from the hospital on the fourth day of hospitalization. Due to the recurrence of DKA episodes, the unsupervised interruption of insulin therapy, and the potential risk of serious complications such as cerebral edema, the case was reported to the Child Protective Services to ensure social support and adequate follow-up for the family.

## DISCUSSION

This case illustrates the vulnerability of children with type 1 diabetes mellitus (DM1) to treatment discontinuation, especially when dependent on insulin provided by public services. Diabetic kala-acetaminophen (DKA) results from severe insulinopenia, leading to hyperglycemia, lipolysis, and ketone body production, culminating in metabolic acidosis.[5] Treatment consists of fluid replacement, electrolyte correction, and continuous intravenous insulin therapy.[6] It was identified that the recent decompensation was precipitated by a failure in insulin use, reflecting a lack of adherence to the therapeutic regimen. The family's inadequate understanding of the silent nature of hyperglycemia and the importance of rigorous diabetes management contributed significantly to the clinical deterioration.

It is important to highlight that many patients with type 1 diabetes initially go through the "honeymoon phase", in which, due to the functional preservation of pancreatic beta cells, they may not need exogenous insulin temporarily, even with 80% to 90% of these cells already compromised.[7] The lack of understanding about this transitional phase, especially by children and their families, can contribute to poor adherence to treatment. This can lead to episodes of decompensation, since such patients may underestimate the severity of the condition and the importance of regular blood glucose monitoring and adequate insulin administration. Consequently, complications such as diabetic ketoacidosis (DKA) may arise, which requires hospitalization and can have serious and even fatal outcomes, such as cerebral edema, seizures, acute renal failure, cardiac arrhythmias, cardiorespiratory arrest, and death.[8]

The reported case is of a ten-year-old patient, diagnosed with type 1 diabetes at age seven and with a history of two previous hospitalizations for DKA. Furthermore, persistent hyperglycemia during hospitalization is common, even with adequate treatment, due to factors such as stress, hospital routine, and a diet unsuitable for the pediatric profile.[1] Family education and personalization of the dietary plan were fundamental to therapeutic success.

## CONCLUSION

DKA is a metabolic emergency associated with type 1 diabetes mellitus (DM1), requiring early diagnosis and immediate therapeutic intervention. Treatment is based on three fundamental pillars: adequate fluid replacement, correction of hydroelectrolytic disturbances, especially potassium, and continuous insulin therapy.[1]

In addition to clinical management, the identification and treatment of the precipitating

factor are essential, as they contribute significantly to the prevention of relapses. The proper management of the reported case reinforces the importance of rapid recognition of the condition and implementation of a well-structured therapeutic protocol to reduce morbidity and mortality associated with DKA.[2]
Diabetic ketoacidosis remains a challenge in pediatric clinical practice, especially in socially vulnerable settings.[8] The case highlights the importance of effective public policies to ensure continuous access to insulin therapy, as well as the need for multidisciplinary follow-up and health education for patients and families.

## REFERENCES

1. Wolfsdorf JI, Glaser N, Agus M, Fritsch M, Hanas R, Rewers A, et al. ISPAD Clinical Practice Consensus Guidelines: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Pediatr Diabetes*. 2018;19(Suppl 27):155-77.
2. Sherry NA, Tsai EB, Herold KC. Natural history of beta-cell function in type 1 diabetes. *Diabetes*. 2005;54(Suppl 2):S32-9.
3. Sociedade Brasileira de Diabetes. Diretriz oficial da Sociedade Brasileira de Diabetes: diagnóstico e tratamento da cetoacidose diabética [Internet]. São Paulo: SBD; 2022 [citado 2025 jun 23]. Disponível em: https://diretriz.diabetes.org.br/diagnostico-e-tratamento-da-cetoacidose-diabetica/
4. Müller HE. Protocolo de tratamento da cetoacidose diabética. In: XXVII Congresso Brasileiro de Medicina Intensiva – CBMI 2022; 2022 nov 10-12; Brasília, Brasil [Internet]. São Paulo: AMIB; 2022 [citado 2025 jun 23]. Disponível em: https://portal.afya.com.br/terapia-intensiva/cbmi-2022-protocolo-de-tratamento-da-cetoacidose-diabetica-cad
5. Van Ness-Otunnu R, Hack JB. A review of diabetic ketoacidosis in children and adolescents. *Curr Opin Pediatr*. 2013;25(4):448–55.
6. Dhatariya KK, Vellanki P. Treatment of diabetic ketoacidosis (DKA)/hyperglycemic hyperosmolar state (HHS): novel advances in the management of hyperglycemic crises (UK versus USA). *Curr Diab Rep*. 2017;17(5):33.
7. Bonifacio E, Ziegler AG. Advances in the prediction and natural history of type 1 diabetes. *Endocrinol Metab Clin North Am*. 2010;39(3):513-25.
8. Haller MJ, Schatz DA, Skyler JS. Type 1 diabetes mellitus: etiology, presentation, and management. *Pediatr Clin North Am*. 2005;52(6):1553-78.

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