

DIABETIC KETOACIDOSIS IN PEDIATRICS: A REVIEW OF PATHOPHYSIOLOGY, DIAGNOSIS, AND CLINICAL MANAGEMENT

CETOACIDOSE DIABÉTICA EM PEDIATRIA: REVISÃO SOBRE
FISIOPATOLOGIA, DIAGNÓSTICO E MANEJO CLÍNICO

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ABSTRACT

Diabetic ketoacidosis (DKA) is among the most severe metabolic emergencies related to diabetes mellitus in children and adolescents, especially type 1 diabetes mellitus, and remains a relevant preventable cause of morbidity and mortality. This study aimed to synthesize recent evidence on the pathophysiology, diagnostic criteria, severity stratification, and clinical management of pediatric DKA, emphasizing implications for healthcare practice and prevention of complications. A literature review was conducted in PubMed/MEDLINE, SciELO, and LILACS, including full-text publications from 2020 to 2026, as well as guidelines and institutional documents considered essential for clinical contextualization. The evidence shows that DKA is defined by the simultaneous presence of hyperglycemia, metabolic acidosis, and ketonemia or ketonuria; however, clinical manifestations in children may be nonspecific, contributing to delayed recognition and increased risk of adverse outcomes. Management requires early identification, assessment of severity, cautious fluid replacement, continuous insulin therapy, correction of electrolyte disturbances, investigation of precipitating factors, and ongoing clinical, laboratory, and neurological monitoring. The findings also reinforce the importance of standardized protocols, trained multidisciplinary teams, family education, and structured follow-up after hospital discharge to reduce recurrence and improve safety. Because the included publications were heterogeneous and no meta-analysis was performed, the results should be interpreted as a qualitative synthesis of the available literature. Overall, pediatric DKA demands timely, protocol-based care to minimize complications and improve clinical outcomes in this vulnerable population.

Keywords: Diabetic ketoacidosis; Type 1 diabetes mellitus; Pediatrics; Diagnosis; Clinical management.

RESUMO

A cetoacidose diabética (CAD) está entre as emergências metabólicas mais graves relacionadas ao diabetes mellitus em crianças e adolescentes, sobretudo ao diabetes mellitus tipo 1, e permanece como causa evitável de morbidade e mortalidade. Este estudo teve como objetivo sintetizar evidências recentes sobre fisiopatologia, critérios diagnósticos, classificação de gravidade e manejo clínico da CAD pediátrica, enfatizando implicações para a prática assistencial e para a prevenção de complicações. Realizou-se revisão da literatura nas bases PubMed/MEDLINE, SciELO e LILACS, com inclusão de publicações disponíveis na íntegra entre 2020 e 2026, além de diretrizes e documentos institucionais essenciais para contextualização clínica. As evidências demonstram que a CAD é definida pela presença simultânea de hiperglicemia, acidose metabólica e cetonemia ou cetonúria; entretanto, suas manifestações em crianças podem ser inespecíficas, favorecendo atraso diagnóstico e maior risco de desfechos adversos. O manejo exige identificação precoce, avaliação da gravidade, reposição hídrica criteriosa, insulinoaterapia contínua, correção de distúrbios eletrolíticos, investigação dos fatores precipitantes e monitorização clínica, laboratorial e neurológica contínua. Os achados reforçam ainda a importância de protocolos padronizados, equipes multiprofissionais capacitadas, educação familiar e seguimento estruturado após a alta hospitalar para reduzir recorrências e ampliar a segurança do cuidado. Devido à heterogeneidade das publicações incluídas e à ausência de metanálise, os resultados devem ser interpretados como síntese qualitativa da literatura disponível. Em síntese, a CAD pediátrica demanda cuidado oportuno e protocolizado, capaz de minimizar complicações e melhorar desfechos clínicos nessa população vulnerável.

Palavras-chave: Cetoacidose diabética; Diabetes mellitus tipo 1; Pediatria; Diagnóstico; Manejo clínico.

1. INTRODUCTION

Diabetic ketoacidosis (DKA) is one of the most severe metabolic emergencies associated with diabetes mellitus, especially type 1 diabetes mellitus (T1DM), and represents an important preventable cause of morbidity and mortality in childhood and adolescence. Its rapid clinical progression, combined with the risk of severe dehydration, hydroelectrolytic disorders, and cerebral edema, makes early recognition and the implementation of systematic therapeutic management essential (Liu et al., 2025; Sociedade Brasileira de Diabetes, 2019).

According to the International Society for Pediatric and Adolescent Diabetes (ISPAD), DKA is defined by the simultaneous presence of hyperglycemia, metabolic acidosis, and ketonemia or ketonuria. Although its diagnostic criteria are well established, the clinical presentation in children may be nonspecific, contributing to diagnostic delays, especially in cases of previously unrecognized T1DM (International Society for Pediatric and Adolescent Diabetes, 2022; Alfayez et al., 2025).

From an epidemiological perspective, it is estimated that 25% to 40% of children newly diagnosed with T1DM present with DKA as the initial manifestation, a proportion that may exceed 60% in low- and middle-income countries. In Brazil, where there is a high number of T1DM cases among individuals under 20 years of age, studies indicate the occurrence of DKA in approximately 30% to 50% of new pediatric diagnoses, with greater vulnerability among young

children and populations with limited access to healthcare services (International Diabetes Federation, 2021; Ramos et al., 2022; International Diabetes Federation, 2025; Dei-Tutu et al., 2025).

Despite advances in clinical guidelines, pediatric DKA still poses care-related challenges involving timely recognition, severity stratification, clinical and laboratory monitoring, and prevention of complications. Management involves careful fluid replacement, insulin therapy, correction of electrolyte disturbances, and investigation of the precipitating factor, requiring integrated and qualified action by the healthcare team (International Society for Pediatric and Adolescent Diabetes, 2022; Pott et al., 2024; Korula; Kozgar; Bwanaisa, 2025).

Therefore, this article aims to synthesize recent evidence on the pathophysiology, diagnostic criteria, severity stratification, and clinical management of diabetic ketoacidosis in children and adolescents, highlighting implications for pediatric care practice and the prevention of complications.

2. THEORETICAL FRAMEWORK

2.1. Pathophysiology

The development of DKA, understood as a highly severe clinical emergency, occurs in patients with decompensated diabetes, especially those with type 1 diabetes mellitus. The condition results from absolute or relative insulin deficiency associated with an increased glucagon-insulin ratio, which reduces peripheral glucose uptake, intensifies lipolysis, increases ketone body production, and culminates in metabolic acidosis. These mechanisms explain the morbidity and mortality associated with the condition, particularly in

the pediatric population (Tilinca; Gliga; Varga, 2022; Ahadiat; Hosseinian, 2023).

The elevation of circulating glucose causes marked hyperglycemia and exceeds renal reabsorption capacity, favoring urinary glucose excretion. This process triggers osmotic diuresis, loss of water and electrolytes, and consequent dehydration, factors that worsen the patient's metabolic instability (Lima et al., 2023).

Insulin deficiency also promotes the mobilization of adipose tissue and the release of free fatty acids into the bloodstream, which are metabolized in the liver and converted into ketone bodies. When the production of these compounds exceeds the body's capacity for utilization and elimination, metabolic acidosis develops. Clinically, DKA may manifest as polyuria, polydipsia, polyphagia, weight loss, blurred vision, ketotic breath, abdominal pain, nausea, and vomiting. In more severe cases, progression of acidosis may impair the central nervous system, causing lethargy, mental confusion, and decreased level of consciousness (Lizzo; Goyal; Gupta, 2023).

In the pediatric population, ketoacidosis is closely related to the imbalance between the production and utilization of ketone bodies, triggered by insulin deficiency and increased glucagon. This process may be intensified in situations of prolonged fasting, infections, and low carbohydrate intake, in which greater mobilization of free fatty acids from adipose tissue occurs. These substrates are directed to the liver and converted into ketone bodies in hepatic mitochondria through ketogenesis (Meoli et al., 2024).

In infants and children under seven years of age, this process may occur more rapidly due to lower glycogen stores and higher

metabolic demand, favoring a rapid increase in beta-hydroxybutyrate levels, a direct marker of the ketotic state. When ketone body production exceeds its utilization and renal excretion, systemic accumulation occurs, leading to metabolic acidosis with an increased anion gap, resulting from the dissociation of keto acids and consumption of plasma bicarbonate. Dehydration worsens this condition by reducing renal ketone clearance (Holman et al., 2023).

2.2. Diagnosis

In addition to clinical signs and symptoms, the main international guidelines use three biochemical criteria for the pediatric diagnosis of DKA: blood glucose above 200 mg/dL, venous pH equal to or below 7.3 or reduced serum bicarbonate, usually below 18 mmol/L, and the presence of ketonemia, with beta-hydroxybutyrate equal to or above 3 mmol/L, or significant ketonuria. These parameters indicate, respectively, hyperglycemia, metabolic acidosis, and ketosis, forming the diagnostic basis of the condition (Umpierrez et al., 2024; Glaser et al., 2022).

The severity of the condition is defined mainly by the intensity of metabolic acidosis and is classified as mild when venous pH is between 7.3 and 7.2, moderate between 7.2 and 7.1, and severe when below 7.1. Other clinical indicators, such as altered level of consciousness, hypotension, tachycardia, peripheral hypoperfusion, and signs of significant dehydration, should also be considered when assessing severity and determining therapeutic management (Santomauro et al., 2023).

2.3. Clinical Management

The clinical management of pediatric DKA is complex and requires early recognition of signs and symptoms, severity stratification, and the implementation of systematic treatment. The approach should include initial stabilization, progressive correction of metabolic abnormalities, and prevention of complications, especially cerebral edema. In addition, it is essential to identify and treat the triggering factor, individualizing the therapeutic plan according to each child's clinical and laboratory findings (Fundação Oswaldo Cruz, 2021; Alnaim et al., 2024).

Therapeutic goals include maintaining airway patency, correcting dehydration, gradually restoring electrolyte and acid-base balance, controlled reduction of hyperglycemia and osmolarity, and investigation and treatment of the precipitating factor, according to recommendations from the Brazilian Diabetes Society (Sociedade Brasileira de Diabetes, 2025).

The initial assessment should follow principles similar to those used in other pediatric emergencies, including the ABCDE protocol, with evaluation of airway, breathing, circulation, neurological status, and patient exposure. Monitoring should be continuous, and the use of two-bag fluid replacement systems may support better glycemic control during treatment (Fundação Oswaldo Cruz, 2021; Younis et al., 2025).

During the maintenance phase, daily fluid replacement should be calculated and the deficit estimated, generally between 5% and 10% of body weight, with gradual correction over 24 to 48 hours. The choice of solution should consider clinical status, osmolarity, and serum sodium levels, avoiding abrupt reductions in plasma osmolarity (Ikeda, 2024).

Insulin therapy should preferably be initiated as a continuous intravenous infusion of low-dose regular insulin, usually 0.1 U/kg/h, after fluid replacement has begun and serum potassium has been assessed. The goal is to promote a gradual decrease in blood glucose, avoiding rapid reductions that may increase the risk of hypoglycemia and cerebral edema. When the glycemic reduction is excessive, dose adjustment to 0.05 U/kg/h is recommended according to clinical and laboratory evolution (Ranzini; Abramovici, 2025).

Transition to subcutaneous insulin should occur only when there is clinical improvement and progressive resolution of acidosis, with normalization of mental status, tolerance of oral feeding, pH equal to or above 7.3, serum bicarbonate equal to or above 15 mmol/L, and blood glucose at safe levels. The subcutaneous dose should be administered before discontinuing the continuous infusion in order to prevent recurrence of hyperglycemia and ketosis (Conti et al., 2024).

The use of insulin bolus is not recommended in pediatric patients, as there is no evidence of benefit and it is associated with a higher risk of complications, including cerebral edema. After resolution of the acute episode, a basal insulin regimen should be established, with individualized selection among NPH, glargine, or degludec, considering age, family routine, therapeutic availability, and specialized follow-up (Gripp et al., 2023; Fundação Oswaldo Cruz, 2021).

Electrolyte replacement should be performed carefully. Serum potassium must be assessed before starting insulin therapy, as insulin administration promotes intracellular shifting of this ion and

may precipitate hypokalemia. It is recommended to maintain potassium within a safe range, generally between 4 and 5 mEq/L; when below 3.3 mEq/L, insulin therapy should be postponed until adequate correction has been achieved (Fonseca Filho; De Lima, 2024; Sociedade Brasileira de Diabetes, 2025).

When significant hypokalemia is present, potassium replacement should be administered intravenously, preferably after restoration of diuresis and with clinical and laboratory monitoring. At intermediate levels, replacement should accompany fluid therapy; in cases of hyperkalemia, administration should be temporarily suspended, with serial reassessment and, when indicated, electrocardiographic monitoring (Costa et al., 2022).

Hypophosphatemia is frequent during DKA treatment but usually follows an asymptomatic or mild course. Routine phosphate replacement is not indicated due to the risk of hypocalcemia and the lack of consistent evidence of clinical benefit; it should be reserved for specific situations, such as marked muscle weakness, cardiac dysfunction, or respiratory failure (Liu et al., 2025).

Cerebral edema is one of the most severe complications of pediatric DKA, although it occurs in a small proportion of cases. It is associated with rapid changes in osmolarity, inadequate fluid replacement, and vasogenic mechanisms. Its management requires immediate recognition, reduction of fluid infusion when indicated, and administration of mannitol or hypertonic saline according to clinical assessment. Hyperventilation should be used cautiously and only in specific situations due to the risk of reduced cerebral perfusion (Ranzini; Abramovici, 2025).

Continuous monitoring is indispensable in DKA management. Vital signs, neurological status, fluid balance, and capillary blood glucose should be assessed at regular intervals, while electrolytes, blood gas analysis, and other laboratory tests should be repeated according to severity and therapeutic response, generally every two to four hours (Costa et al., 2022).

Ketonuria is more useful for diagnosis than for treatment monitoring because, during insulin therapy, part of beta-hydroxybutyrate may be converted into acetoacetate, producing an apparent persistence of urinary ketosis. Thus, resolution of DKA should preferably be assessed by clinical and laboratory parameters, including improvement in general condition, blood glucose below 200 mg/dL, pH above 7.30 or serum bicarbonate above 18 mEq/L, anion gap below 12 mEq/L, and resolution of ketonemia (Sociedade Brasileira de Diabetes, 2025).

3. METHODOLOGY

This is a qualitative literature review, with a descriptive and comparative approach, developed with the aim of gathering and analyzing recent evidence on the pathophysiology, diagnosis, severity stratification, prognosis, and clinical management of diabetic ketoacidosis (DKA) in children and adolescents. The guiding question was: what is the main available evidence on the diagnosis and clinical management of DKA in the pediatric population?

The bibliographic search was conducted in the PubMed/MEDLINE, SciELO, and LILACS databases. In PubMed/MEDLINE, the following MeSH terms and Boolean operators were used: “Diabetic Ketoacidosis” AND (“Child” OR “Adolescent” OR “Pediatrics”) AND

("Diagnosis" OR "Therapeutics" OR "Disease Management" OR "Clinical Protocols"). In the SciELO and LILACS databases, equivalent DeCS descriptors in Portuguese and Spanish were used: "cetoacidose diabética" AND ("criança" OR "adolescente" OR "pediatria") AND ("diagnóstico" OR "terapêutica" OR "manejo clínico").

Full-text publications published between 2020 and 2026, in Portuguese, English, or Spanish, that addressed DKA in pediatric patients and presented information related to pathophysiology, diagnosis, severity, treatment, complications, prognosis, or prevention of recurrence were included. Relevant clinical guidelines, consensus statements, and institutional documents published outside this time frame were also considered when indispensable for the epidemiological, diagnostic, or therapeutic contextualization of the topic.

Editorials, letters to the editor, conference abstracts, isolated case reports, duplicates, studies without full text, publications focused exclusively on the adult population, and articles with no direct relationship to the objectives of the review were excluded. Study selection occurred in two stages: first, titles and abstracts were read; subsequently, potentially eligible full texts were assessed to confirm thematic relevance.

The following information was extracted from the selected studies: author, year of publication, type of publication, population addressed, objective, main findings, and clinical implications. These data were organized narratively, allowing comparison among studies and identification of the main points of convergence and divergence in the literature.

The analysis was performed through qualitative synthesis, without meta-analysis, due to the heterogeneity of the study designs, populations, care contexts, and outcomes assessed in the included publications. The results were grouped into thematic axes: precipitating factors and clinical vulnerabilities; diagnosis and severity stratification; fluid replacement, insulin therapy, and electrolyte correction; complications and monitoring; and prevention of recurrence. Table 1 presents the core studies used in the synthesis of the findings.

4. RESULTS AND DISCUSSION

The analysis of the selected studies made it possible to organize the evidence into five central axes: precipitating factors and clinical vulnerabilities; diagnostic criteria and severity stratification; fluid replacement, insulin therapy, and electrolyte correction; complications and monitoring; and health education for the prevention of recurrences. The synthesis of the main studies used as the basis for the discussion is presented in Table 1.

Table 1. Core studies included in the literature review on pediatric diabetic ketoacidosis

AUTHOR / YEAR	ARTICLE TITLE	HIGHLIGHTED POINTS
Nunes <i>et al.</i> , 2021	Incidence, characteristics and long-term outcomes of patients with diabetic ketoacidosis: a prospective prognosis cohort study in an	The study identified non-adherence to treatment and the presence of infections as the main factors associated with the occurrence of diabetic ketoacidosis in the emergency care setting. The results highlight the importance of early recognition of precipitating factors and continuous clinical follow-up to

	emergency department	reduce episodes of metabolic decompensation.
Isik e Aydin, 2024	The effect of serum biochemical parameters on clinical prognosis in children presenting with diabetic ketoacidosis	The findings demonstrated that biochemical parameters, especially blood glucose, blood pH, and serum bicarbonate, showed a relevant association with the clinical course and prognosis of children with diabetic ketoacidosis. These indicators proved useful for guiding severity assessment, therapeutic monitoring, and defining the appropriate time for transition from insulin therapy.
Souza et al., 2020	Diabetic ketoacidosis as the initial presentation of type 1 diabetes in children and adolescents: epidemiological study in southern brazil	The results indicated that diabetic ketoacidosis may constitute the initial manifestation of type 1 diabetes mellitus in children and adolescents. The presence of DKA at diagnosis was associated with relevant clinical repercussions, including greater initial severity and the need for strategies aimed at early recognition of signs of glycemic decompensation.
Espinola et al., 2024	Cetoacidosis diabética en niños y adolescentes. Actualización en diagnóstico y tratamiento	The analyzed update highlighted changes in diagnostic and therapeutic recommendations for pediatric diabetic ketoacidosis, emphasizing the standardization of clinical and laboratory criteria, careful fluid replacement, correction of electrolyte disturbances, and prevention of complications during treatment.

Gripp <i>et al.</i> , 2023	Current recommendations for management of paediatric diabetic ketoacidosis	The results emphasized that pediatric DKA presents clinical manifestations resulting from metabolic decompensation, including dehydration, Kussmaul breathing, ketotic breath, abdominal pain, nausea, vomiting, and altered level of consciousness. These findings are generally preceded by polydipsia, polyuria, and weight loss, reinforcing the need for early identification of clinical signs.
Kostopoulou <i>et al.</i> , 2023	Diabetic Ketoacidosis in Children and Adolescents; Diagnostic and Therapeutic Pitfalls	The study highlighted that pediatric diabetic ketoacidosis is a severe metabolic emergency characterized by hyperglycemia, ketosis, and metabolic acidosis. The findings showed diagnostic and therapeutic difficulties related to clinical overlap with other acute conditions and the need to differentiate DKA from other hyperglycemic conditions.
Rodrigues <i>et al.</i> , 2021	Reconhecendo os principais sinais e sintomas da cetoacidose diabética: uma revisão integrativa	The review identified polydipsia, polyuria, and weight loss as the most frequent symptoms among patients with diabetic ketoacidosis. The results also indicated that insufficient recognition of these signs, associated with the low specificity of the clinical presentation, may hinder timely diagnosis, control, and adequate monitoring of the condition.

Source: Authors' own work (2026).

In the axis of precipitating factors, the studies converge in indicating that infections, failures in therapeutic adherence, delayed diagnosis

of T1DM, and difficulties in accessing specialized care are among the main determinants of metabolic decompensation. Nunes et al. (2021) emphasize that effective management of DKA requires not only correction of hypovolemia, acidosis, hyperglycemia, and electrolyte disturbances, but also active identification of the triggering factor. This perspective broadens the focus of treatment, shifting it from an exclusively acute intervention to a preventive and longitudinal approach.

Regarding diagnosis and severity, blood glucose, venous pH, serum bicarbonate, and the presence of ketonemia or ketonuria are the main clinical and laboratory parameters used to confirm DKA and guide management. Isik and Aydin (2024) emphasize that blood glucose, blood pH, and bicarbonate have a relevant association with clinical evolution and with the timing of transition to subcutaneous insulin therapy, reinforcing that serial laboratory assessment should be interpreted together with the child's clinical status.

The literature also demonstrates that pediatric DKA has a rapid and potentially severe course, marked by hyperglycemia, ketosis, metabolic acidosis, dehydration, and hydroelectrolytic changes. Although consolidated guidelines for diagnosis and management exist, gaps persist regarding the uniform implementation of protocols, team training, and early recognition in first-contact healthcare services (Wei et al., 2020).

In the epidemiological axis, Souza et al. (2020) highlight that DKA may represent the first manifestation of T1DM in children and adolescents, a situation associated with greater initial severity and risk of poorer subsequent metabolic control. This finding reinforces the need for educational campaigns directed at families, schools,

and primary care professionals, with emphasis on polydipsia, polyuria, weight loss, and signs of dehydration.

Regarding treatment, Espinola et al. (2024) reinforce that evidence-based protocols favor the standardization of fluid replacement, insulin therapy, electrolyte correction, and neurological monitoring. The main clinical implication is that DKA management should avoid abrupt corrections of glycemia and osmolarity, as such fluctuations may increase the risk of complications, especially cerebral edema.

The clinical presentation described by Gripp et al. (2023), including dehydration, Kussmaul breathing, ketotic breath, abdominal pain, nausea, vomiting, and altered level of consciousness, shows that signs may vary according to age, duration of evolution, and severity of acidosis. In younger children, nonspecific manifestations may make diagnosis difficult, making clinical suspicion indispensable in the presence of persistent or progressive symptoms.

In the monitoring axis, Druken et al. (2025) describe the importance of serial monitoring of vital signs, neurological status, blood glucose, and laboratory parameters. This surveillance allows assessment of therapeutic response, adjustment of fluid replacement and insulin therapy, and early identification of signs of neurological deterioration or electrolyte disturbances.

Kostopoulou et al. (2023) emphasize that DKA is a severe metabolic emergency and that its main challenges include differential diagnosis with other acute conditions, recognition of atypical presentations, and prevention of therapeutic errors. Thus, the use of protocols should be accompanied by individualized clinical judgment.

Severity classification based on venous pH and serum bicarbonate, described by Santomauro et al. (2023), contributes to guiding the place of care, intensity of monitoring, and need for specialized support. Additional studies indicate that the occurrence of DKA at the diagnosis of T1DM is associated with poorer future metabolic control, also characterizing it as a marker of clinical vulnerability (Alves; Braz; Linhares, 2023; Dovic et al., 2025).

Rodrigues et al. (2021) identified polydipsia, polyuria, and weight loss as frequent symptoms, but highlighted their low specificity. This limitation reinforces the importance of health education and timely screening strategies, especially in young children, in whom diagnostic delay may result in severe dehydration, marked acidosis, and the need for admission to an intensive care unit.

Finally, prevention of recurrences requires continuous and interdisciplinary care. De Wit et al. (2022), Dabas, Sarin, and Madhu (2023), and Silva et al. (2024) highlight the relevance of psychosocial support, adherence to insulin therapy, health education, and family follow-up to improve glycemic control and quality of life. Thus, DKA management should not be restricted to the hospital environment, but should include discharge planning, family guidance, and structured outpatient follow-up.

5. CONCLUSION

It is concluded that pediatric diabetic ketoacidosis remains a highly severe metabolic emergency, requiring early recognition, diagnostic confirmation through clinical and laboratory criteria, and systematic therapeutic management. The evidence analyzed reinforces that careful fluid replacement, low-dose insulin therapy, individualized

correction of electrolyte disturbances, and continuous clinical, laboratory, and neurological monitoring are essential to reduce complications, especially cerebral edema, hypoglycemia, and hydroelectrolytic changes. In addition to treating the acute episode, preventing recurrences depends on health education, therapeutic adherence, family support, interdisciplinary follow-up, and continuity of care after hospital discharge. However, the heterogeneity of the included studies, the presence of different methodological designs, and the absence of meta-analysis limit the strength of the inferences, indicating that the findings should be interpreted as a qualitative synthesis of the available literature. Thus, the need for standardized care protocols, training of healthcare teams, strengthening of primary care for timely diagnosis, and the development of new pediatric studies with greater methodological rigor is emphasized, so that clinical outcomes, therapeutic safety, and effective strategies for preventing DKA in children and adolescents can be assessed.

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