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Mixed Presentation of Hyperglycemic Hyperosmolar State and Diabetic Ketoacidosis in a Pediatric Patient: a Case Report and Literature Review

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ABSTRACT

The coexistence of diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS), known as mixed hyperglycemic crisis, is a rare but potentially fatal metabolic emergency in pediatrics, and is frequently underrecognized because it is misclassified as isolated DKA. It carries a higher risk of complications than either entity alone, particularly in patients with complex comorbidities. We describe the case of a 16-year-old female with myelomeningocele, neurogenic bladder, obesity, and insulin resistance, who had self-discontinued treatment with metformin, atorvastatin, and allopurinol one year prior and presented with sudden neurological deterioration. Initial workup revealed extreme hyperglycemia (1,777 mg/dL), severe hyperosmolarity (431 mOsm/kg), decompensated metabolic acidosis (pH 7.13), and positive urine ketones (+++), consistent with a mixed DKA-HHS presentation. She subsequently developed septic shock of urinary origin, acute kidney injury, and acute pancreatitis (lipase 3,911 U/L). Despite intensive management guided by *International Society for Pediatric and Adolescent Diabetes-ISPAD* recommendations, she progressed to refractory shock and multiorgan failure, dying within 48 hours of admission. To the best of our knowledge, this represents one of the most extreme metabolic derangements reported in pediatric mixed hyperglycemic crisis. The convergence of extreme hyperosmolarity, concurrent sepsis, and structural comorbidities created a lethal risk phenotype in which sepsis, rather than the degree of metabolic derangement alone, appeared to determine the fatal outcome. Early recognition and an individualized therapeutic approach are essential to improve outcomes in vulnerable pediatric patients.

Keywords: Diabetic ketoacidosis, hyperglycemic hyperosmolar non-ketotic coma, pediatric emergency medicine, pediatric endocrinology, septic shock

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What is already known on this topic?

The coexistence of diabetic ketoacidosis and hyperglycemic hyperosmolar state, termed mixed hyperglycemic crisis, is uncommon in pediatrics but is associated with a higher risk of complications than either entity alone and is frequently underdiagnosed due to erroneous coding as pure diabetic ketoacidosis (DKA).

What this study adds?

This case documents one of the most extreme metabolic presentations of mixed DKA-hyperglycemic hyperosmolar state in a pediatric patient. The present case shows that concurrent septic shock, rather than the degree of metabolic derangement, may be the primary determinant of fatal outcome.

Introduction

Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) are two of the most severe acute metabolic complications of diabetes. They have traditionally been considered distinct clinical entities with well-defined pathophysiological mechanisms and diagnostic criteria. However, in recent years, mixed presentations have been recognized in which features of both metabolic derangements coexist (1). This phenomenon has been described more frequently in adults with type 2 diabetes. Although its presentation in the pediatric population is uncommon, it tends to be associated with greater clinical severity, more complex metabolic disturbances, and a high risk of complications and mortality (2).

In children, DKA is one of the most common initial presentations of type 1 diabetes, reported in approximately 15-70% of cases in Europe and North America (3). In contrast, HHS is rare in this age group, with an estimated incidence of approximately 2% in young patients with type 2 diabetes (4). The overlap between DKA and HHS accounts for approximately 13.8% of described hyperglycemic emergencies, although its true frequency in the pediatric population is likely underestimated (5).

From a pathophysiological perspective, these hyperglycemic crises can be understood as a metabolic continuum. This spectrum is illustrated in Figure 1, where DKA and HHS occupy distinct pathophysiological extremes, while the mixed state represents a transitional zone in which biochemical features of both entities coexist (6).

Clinically, the mixed state combines the dehydration, tachypnea, gastrointestinal symptoms, and reduced consciousness of DKA with the profound hyperosmolar dehydration of HHS, usually preceded by polyuria, polydipsia, and weight loss (7). In pediatric type 2 diabetes, hyperglycemic emergencies with a hyperosmolar component carry substantially greater severity and mortality than isolated DKA, with obesity, insulin resistance, treatment non-adherence, male sex, and non-White race as recognized risk factors (8).

The differential diagnosis must distinguish the overlap from isolated DKA, isolated HHS, and other conditions with similar presentations, such as urinary tract infection, pneumonia, gastroenteritis, acute abdomen, and central nervous system infection (7). Management rests on careful fluid resuscitation, timely intravenous insulin, electrolyte correction, and treatment of the precipitating factors, adapted to the hyperosmolar component of the overlap (6).

The objective of this report is to describe a case of mixed hyperglycemic crisis in an adolescent with multiple comorbidities, characterized by extremely elevated blood glucose and osmolality, and the development of severe systemic complications including acute pancreatitis and septic shock with fatal outcome.

Case Report

We present the case of a 16-year-old female adolescent with a history of myelomeningocele repaired in the neonatal period, tethered spinal cord with surgical untethering, hydrocephalus treated with ventriculoperitoneal shunt and ventriculostomy, neurogenic bladder, obesity, insulin resistance, and metabolic syndrome. Approximately one year before admission, the patient had self-discontinued her prescribed treatment, defined as cessation of medication by the patient and family without medical indication. Her regimen included metformin for insulin resistance, atorvastatin for dyslipidemia, and allopurinol for hyperuricemia. She had been lost to medical follow-up during that year. Her last documented evaluation before loss to follow-up had shown an HbA1c of 6.1% and a Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) of 14.5 [calculated from fasting serum insulin and fasting glucose using the formula: fasting insulin (μ U/mL) $\times$ fasting glucose (mg/dL)/405], consistent with marked insulin resistance.

She presented to the emergency department with neurological deterioration following a sudden-onset episode of nausea and dizziness. On arrival, she was found to have tachycardia (120

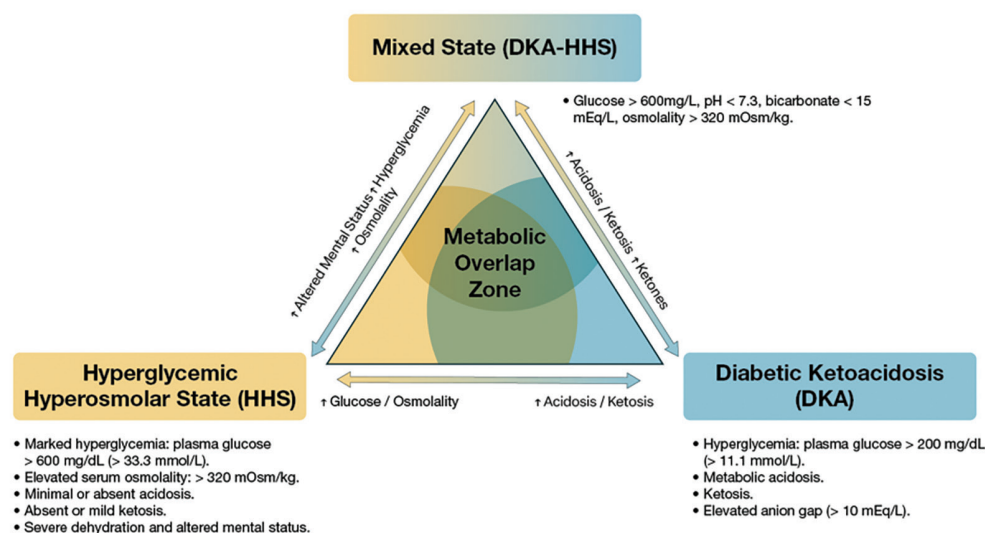


Figure 1. Metabolic triangle of hyperglycemic crises. The clinical spectrum between diabetic ketoacidosis, characterized by metabolic acidosis and ketosis, and hyperglycemic hyperosmolar state, characterized by severe hyperglycemia and dehydration. The upper vertex represents a transitional zone, the Mixed State, where criteria from both entities converge. Figure created by the authors; clinical content and diagnostic criteria adapted from Zahran and Jadidi (9)

beats/min), tachypnea (32 breaths/min), and clinical signs of dehydration. Initial assessment revealed a Glasgow Coma Scale score of 8, prompting advanced airway management with orotracheal intubation. On physical examination, she weighed 76 kg [+1.51 standard deviation score (SDS)], with a height of 148 cm (-2.30 SDS) and a body mass index of 34.7 kg/m² (+2.01 SDS), corresponding to class 1 obesity (117% of the 95th percentile), with a waist-to-height ratio of 0.74. She had extensive grade IV acanthosis nigricans on the neck and axillae, acne vulgaris on the cheeks, and whitish abdominal striae. Pubertal development was Tanner stage IV.

Based on the patient's phenotype of obesity, insulin resistance, metabolic syndrome, prior response to metformin, first-degree family history of type 2 diabetes, and clinical characteristics, her underlying condition was consistent with type 2 diabetes mellitus. Due to the acute presentation of the complications, a new HbA1c measurement was ordered, with a result of 6.5%, however we were unable to analyze C-peptide level and pancreatic antibody positivity due to lack of availability.

Laboratory studies demonstrated severe hyperglycemia (glucose 1,777 mg/dL), an elevated corrected serum sodium (151 mmol/L), a total serum osmolality of 431 mOsm/kg with an elevated effective serum osmolality of 331 mOsm/L (>320 mOsm/kg), and decompensated hyperlactemic metabolic acidosis with elevated anion gap (pH 7.13, pCO₂ 36.7 mmHg, pO₂ 42 mmHg, bicarbonate 11.9 mEq/L, base excess -17.2, anion gap 45.5 mmol/L and lactate 9.7 mmol/L), with positive urine ketones (++++), findings consistent with a mixed DKA-HHS presentation.

Blood β -hydroxybutyrate could not be measured, as the assay was not available at our institution.

During her clinical course, she developed septic shock of urinary origin in the context of neurogenic bladder and acute kidney injury. Urinalysis revealed positive nitrites, positive leukocyte esterase, pyuria, and bacteriuria, and a catheter urine culture grew *Escherichia coli* (800,000 CFU/mL), confirming the urinary source; paired blood cultures (peripheral and central) remained negative after 5 days of incubation. Due to clinical signs of cerebral edema that included an altered response to painful stimuli and a decreased level of consciousness, an infusion of 3% hypertonic saline at 2.5 mL/kg over 15 minutes was administered. Cross-sectional neuroimaging (computed tomography/magnetic resonance imaging) could not be performed due to hemodynamic instability; bedside optic nerve sheath diameter ultrasound showed a diameter of 5.5 mm with an estimated intracranial pressure of 23 mmHg, consistent with intracranial hypertension. She also exhibited marked elevation of pancreatic enzymes (lipase 3,911 U/L and amylase 475 U/L). In addition, abdominal ultrasound showed increased density of the peripancreatic fat, supporting the diagnosis of acute pancreatitis.

Intensive management was initiated, according to current International Society for Pediatric and Adolescent Diabetes (ISPAD) guidelines, including fluid resuscitation, beginning with two 0.9% saline boluses for initial fluid replacement, up to a maximum volume of 1,000 mL. This was followed by fluid therapy based on a 12% fluid deficit plus daily maintenance requirements, administered as 0.45% saline through a two-bag

system, for a total calculated volume of 16 liters over 48 hours (from which the initial boluses were subtracted). An insulin infusion at 0.05 U/kg/h started approximately 1 hour after the onset of fluid administration. Electrolyte correction included 20 mEq/m² of potassium and 20 mEq/m² of phosphate, both later suspended and the fluid administration rate adjusted upon identification of acute kidney injury. Broad-spectrum antimicrobial therapy and vasopressor support were also provided. Figure 2 shows the temporal evolution of glucose and corrected serum sodium.

Despite the therapeutic measures implemented, hemodynamic deterioration progressed, multiple organ failure developed, and vasopressor-refractory shock was present. Cardiorespiratory arrest occurred 48 hours after admission. In accordance with a previously expressed maternal decision, no resuscitation was performed, and death was declared.

Discussion

The DKA-HHS overlap, or mixed state, represents a particularly severe form within the spectrum of pediatric hyperglycemic crises. Pathophysiologically, the mixed state reflects the coexistence of two mechanisms that are ordinarily distinct: the ketogenesis characteristic of DKA, driven by relative insulin deficiency and elevation of counter-regulatory hormones; and the profound osmotic dehydration characteristic of HHS,

resulting from sustained osmotic diuresis with net free water loss (1,6).

A clinically relevant finding is that patients with mixed presentation have a 3.7-fold higher risk of complications compared to those with isolated DKA, and 81.5% of these cases are erroneously coded as pure DKA, delaying recognition of hyperosmolarity and the necessary therapeutic adjustments (5).

The initial glucose decline rate (~150 mg/dL/h during the first 3 hours) exceeded the ISPAD-recommended threshold of 100 mg/dL/h (6). This likely reflected aggressive fluid resuscitation combined with the patient's extreme baseline hyperglycemia. The rate subsequently moderated to a controlled range for the remainder of treatment, and corrected serum sodium declined gradually throughout the admission, without an abrupt fall.

In the presented case, the extreme values recorded, with blood glucose of 1,777 mg/dL and osmolarity of 431 mOsm/kg, markedly exceed the diagnostic thresholds for the mixed state (glucose >600 mg/dL, effective osmolality >320 mOsm/kg) and those reported in other pediatric overlap cases (10). This places our patient at the most severe end of the spectrum, where even aggressive interventions may be insufficient to reverse the cascade of multiorgan failure.

Table 1 summarizes the pediatric cases of DKA-HHS overlap reported to date with available data on metabolic values, complications, and outcomes. Comparative analysis allows identification of clinically relevant patterns: the majority of cases with osmolarity below 400 mOsm/kg and without concurrent systemic infection had favorable outcomes, even in the presence of severe acidosis. In contrast, cases with extreme osmolarity values combined with a second hemodynamic insult, such as sepsis, showed a higher risk of multiorgan failure and mortality. This observation is consistent with epidemiological data reporting a mortality rate of 8% in mixed DKA-HHS presentation, significantly higher than isolated DKA (3%) or HHS alone (5%) (1).

In the pediatric population, most reported cases of DKA-HHS overlap occur in adolescents with obesity or insulin resistance, characteristics that were also present in the presented patient (5). The combination of insulin resistance and acute metabolic stress may promote a marked increase in hepatic glucose production and severe dehydration secondary to osmotic diuresis, contributing to the development of severe hyperosmolarity.

In the case reported by Lu et al. (11), an adolescent with type 1 diabetes presented with deterioration of consciousness and criteria for mixed presentation with moderate hyperosmolarity (334 mOsm/kg) and severe acidosis (pH 6.988; HCO₃⁻ 2.9 mmol/L), triggered by a respiratory infectious focus; she received

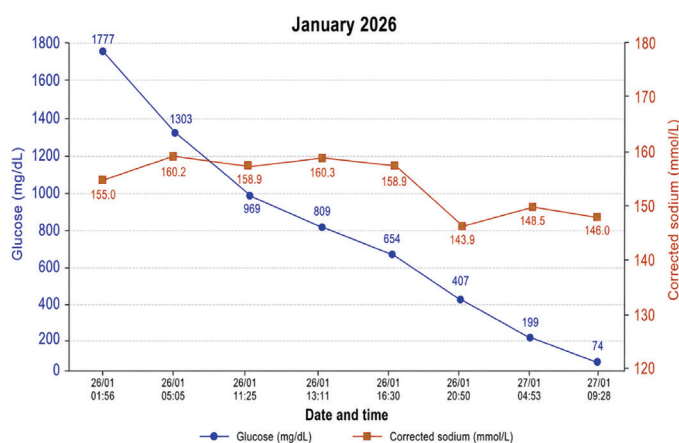


Figure 2. Temporal evolution of serum glucose (blue, left axis) and corrected serum sodium (gold, right axis) during the first 32 hours of management. Serum glucose decreased progressively from a peak of 1,777 mg/dL at admission to 74 mg/dL, with an initial rate of decline of approximately 150 mg/dL/hour during the first 3 hours, moderating to 26-90 mg/dL/hour thereafter. Corrected serum sodium remained within a high-normal range and declined gradually, without an abrupt fall, consistent with a controlled reduction in effective osmolality. Corrected sodium was calculated as: measured sodium + 1.6 × [(glucose - 100)/100], for glucose > 100 mg/dL

Table 1. Pediatric cases of DKA-HHS overlap reported in the literature

Author (year)	Country	Age/sex/ comorbidities	Diabetes type	Glucose (mg/dL)	Osmolality (mOsm/kg)	pH	Precipitating factor	Complications	Outcome
Filippatos et al. 10 (2026)	Greece	5 years/male/ previously healthy	T1DM debut	826	344	7.29	Post-vaccine influenza	Severe hypertriglyceridemia	Survived
Lu et al. (11) (2025)	Indonesia	15 years/female/ known T1DM	T1DM	>600	334	6.99	Respiratory infection	Deterioration of consciousness	Survived
Noh et al. (12) (2023)	South Korea	16 years/ female/obesity, nephronophthisis	Mixed DM	>600	>320	<7.3	Not specified	AKI, rhabdomyolysis, pancreatitis	Survived
Saeed and Ziyae (15) (2023)	Iran	13 years/female/ known T1DM	T1DM	685	>320	6.67	Septic shock	Septic shock	Survived
Lee et al. (14) (2023)	South Korea	11-15 years/ male and female/ obesity	T2DM	>600	>320	6.81	Non-adherence to treatment	Arrhythmia, AKI, rhabdomyolysis, pancreatitis	All survived
Parra Villasmil et al. (4) (2022)	USA	14 years/female/ intellectual disability/VPS	T1DM debut	1,188	428	7.09	Not specified	Decompensated shock/ hypernatremia	Survived
Li et al. (13) (2014)	China	11 and 14 years/ male/obesity	T2DM	>600	>320	<7.3	Non-adherence to treatment	Rhabdomyolysis/AKI	Both survived
Current case (2026)	Mexico	16 years/female/ MMC, neurogenic bladder, IR	T2DM	1,777	431	7.13	UTI/sepsis + treatment abandonment	Refractory septic shock/AKI/Acute pancreatitis (lipase 3,911 U/L)/cerebral edema	Death (48 h)

DKA: diabetic ketoacidosis; HHS: hyperglycemic hyperosmolar state; T1DM: type 1 diabetes mellitus; T2DM: type 2 diabetes mellitus; VPS: ventriculoperitoneal shunt; UTI: urinary tract infection; AKI: acute kidney injury; MMC: myelomeningocele; IR: insulin resistance

fluid therapy, insulin, antibiotics, and intensive care unit (ICU) care, with recovery and discharge on day 7. This contrast suggests that, although acidosis may be extreme, the degree of hyperosmolality, the magnitude of effective dehydration, and the presence of refractory shock may tip the clinical course toward multiorgan failure.

A critical differentiating element in the presented case is the concurrent septic shock of urinary origin, the precipitant of which was the neurogenic bladder associated with myelomeningocele. Recurrent urinary tract infection is one of the leading causes of morbidity in patients with neurogenic bladder. Sepsis can induce peripheral insulin resistance and increase the release of catecholamines, cortisol, and glucagon, promoting both hyperglycemia and lipolysis and ketogenesis. Furthermore, the tissue hypoperfusion inherent to septic shock exacerbates lactic acidosis and end-organ injury, generating a vicious cycle that is therapeutically difficult to break (12).

Comparatively, the case reported by Parra Villasmil et al. (4) shared two features with our patient: underlying neurological vulnerability (ventriculoperitoneal shunt) and severe hyperosmolality (428 mOsm/kg). However, in that case, no second hemodynamic insult of infectious nature was documented, which likely explains the favorable outcome. Similarly, the

case of Filippatos et al. (10) of a 5-year-old child with newly diagnosed mixed presentation achieved favorable resolution without the concurrent presence of a systemic infectious focus. These contrasts reinforce the hypothesis that concomitant sepsis, rather than the magnitude of the metabolic derangement *per se*, may be the primary determinant of outcome in these patients.

Contrasted with reported cases, our patient combined the most extreme metabolic derangement with the only fatal outcome. Glucose (1,777 mg/dL) and osmolality (431 mOsm/kg) exceeded those of Parra Villasmil et al. (4) (1,188 mg/dL; 428 mOsm/kg) and surpassed the remaining reports. Unlike cases triggered by new-onset diabetes or infection, including two with type 2 diabetes and treatment non-adherence (13,14), the presented case combined treatment discontinuation with a urinary septic focus. Although septic shock was also reported with survival (15), the fatal course here reflected the convergence of extreme hyperosmolality, sepsis, pancreatitis, and cerebral edema rather than any single factor. Acute pancreatitis in the context of mixed hyperglycemic crisis warrants a thorough pathophysiological interpretation. In our patient, with lipase levels of 3,911 U/L and amylase of 475 U/L, pancreatic injury likely resulted from the convergence of three mechanisms: 1) profound insulinopenia with secondary lipotoxicity; 2) tissue hypoperfusion from septic

shock; and 3) amplified systemic inflammation. Noh et al. (12) reported a case of DKA-HHS with pancreatitis, rhabdomyolysis, and renal failure in an obese adolescent who, unlike our patient, survived after a prolonged clinical course. The difference in outcome may be attributed to the absence of concurrent septic shock in that case.

Acute kidney injury, also documented in our case, is the most frequent organ complication in pediatric DKA series, occurring in 12 of 24 cases reviewed by Brar et al. (16). This complication, together with rhabdomyolysis, has similarly been reported in pediatric type 2 diabetes presenting with mixed hyperglycemic crisis (13). Furthermore, recent studies demonstrated that measured serum osmolality is an independent marker of complications in pediatric DKA, as patients with osmolality >320 mOsm/kg had an incidence of acute kidney injury of 58%, compared to 12.1% in normo-osmolar patients, and an ICU admission rate of 31.9% versus 12.1% (17). These data support the incorporation of measured serum osmolality as a risk stratification tool in the initial assessment of every pediatric patient presenting with a hyperglycemic crisis.

The markedly elevated anion gap (45.5 mmol/L) was multifactorial rather than attributable to ketoacidosis alone. Hyperlactatemia is common in pediatric DKA and is associated with markers of hemodynamic compromise and greater severity (18). In the presented patient, the elevated lactate (9.7 mmol/L) from septic shock and tissue hypoperfusion contributed substantially, alongside ketoacid accumulation and retained anions from acute kidney injury.

Management of the mixed DKA-HHS state imposes a therapeutic paradox that distinguishes it from the treatment of either entity in isolation. On the other hand, correction of hyperosmolality requires more aggressive fluid resuscitation than that recommended for classic DKA; ISPAD guidelines suggest assuming a fluid deficit of 12-15% of body weight and using initial isotonic fluid boluses of up to 20 mL/kg (6).

This is particularly relevant when contrasted with the historically proposed threshold of ≤ 4.0 L/m²/day for pediatric DKA, aimed at reducing cerebral edema risk (19); this limit was derived from DKA cohorts and is not directly applicable to HHS or mixed presentations. In the presented patient, the total calculated fluid volume of 16 L over 48 hours corresponded to approximately 4.3 L/m²/day (body surface area 1.87 m²), which exceeded the DKA-specific threshold but was consistent with the higher fluid requirements recommended for mixed DKA-HHS.

Weight-based calculations in obese patients should also be capped (75 kg per National Institute for Health and Care Excellence-80 kg per ISPAD) rather than using actual weight; both

guidelines recommend fluid boluses of 10-20 mL/kg in shock, with reassessment of perfusion between doses, whereas non-shock boluses are capped at 20 mL/kg or 1,000 mL, as applied in our patient, and are subtracted from the total deficit, unlike shock boluses (20).

However, excessively rapid correction of osmolality increases the risk of cerebral edema, particularly in a patient with a history of treated hydrocephalus and prior intracranial hypertension (21). In the context of concurrent septic shock, this therapeutic tension deepens, as peripheral hypoperfusion mandates volume administration that could aggravate the risk of edema. The absence of specific protocols for the management of the mixed state with concurrent septic shock underscores the need for a multidisciplinary and individualized approach. Structured diagnostic and management frameworks for pediatric hyperosmolar crises may support this process (9).

The history of self-discontinuation of pharmacological treatment approximately one year prior to admission constitutes a determining precipitating factor that distinguishes this patient from those presenting at diabetes onset. Pre-existing insulin resistance and chronic metabolic decompensation prior to the crisis partly explain the magnitude of glucose and osmolality values at admission, as uncontrolled progressive hyperglycemia generates sustained osmotic diuresis that progressively depletes free water reserves. In a patient with neurogenic bladder, even monitoring of urinary output as an early warning sign is functionally compromised.

In comparison, Lu et al. (11) described a case of new-onset diabetes with mixed presentation triggered by respiratory infection, with resolution on day 7. The contrast is illustrative: in new-onset diabetes, hyperosmolality tends to be moderate because the deterioration is acute; in patients with pre-existing insulin resistance and treatment abandonment, chronic decompensation allows glucose and osmolality to reach extreme values before the clinical presentation becomes apparent.

This report has limitations inherent to the emergency scenario. At admission, serum insulin, C-peptide, HbA1c, pancreatic autoantibodies, and serum β -hydroxybutyrate were not measured, and cross-sectional neuroimaging could not be performed, owing to the patient's rapid deterioration and hemodynamic instability. The diagnosis of type 2 diabetes was therefore based on the clinical phenotype and previously documented metabolic data, and the unavailability of C-peptide and pancreatic autoantibodies precludes the definitive exclusion of other diabetes subtypes. The previously documented HOMA-IR was interpreted only as a marker of insulin resistance and not as a diagnostic criterion for diabetes.

Conclusion

This case illustrates that pediatric hyperglycemic crises do not always present as clearly distinct metabolic entities. The mixed DKA-HHS state represents a pathophysiological transitional zone in which biochemical features of both metabolic derangements may coexist. Early identification of this phenotype is essential, as these patients may follow a more complex clinical course and require intensive management strategies adapted to hyperosmolarity and the associated systemic complications.

Ethics

Informed Consent: Informed consent for publication was obtained from the patient's parents.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Mónica Lizeth Pineda Macal, Concept: Irvin Ordoñez-González, Design: Irvin Ordoñez-González, Data Collection or Processing: Rosalía García-Couturier, Ana Paula Gómez Pérez, Analysis or Interpretation: María Paola Saucedo Coronado, Mónica Lizeth Pineda Macal, Literature Search: Ana Paula Gómez Pérez, Writing: Irvin Ordoñez-González, Rosalía García-Couturier.

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