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Are intensive care settings essential in the management of diabetic ketoacidosis in children?

Running title: Pediatric DKA outside the PICU

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Abstract

Purpose: Diabetic ketoacidosis (DKA) is a potentially life-threatening complication of diabetes in children and is frequently managed in pediatric intensive care units (PICUs). This study aimed to evaluate the feasibility and safety of managing DKA in the pediatric emergency department (PED).

Methods: A retrospective analysis was conducted of pediatric patients presenting with DKA between 2017 and 2024 at a tertiary care hospital. Patients were categorized according to DKA severity and evaluated for demographic characteristics, risk factors, clinical findings, laboratory parameters, complications, and outcomes. All patients were managed using a standardized institutional treatment protocol. Clinical characteristics of patients admitted to the PICU were compared with those managed in the PED.

Results: A total of 169 patients were included (mean age, 10.3 ± 4.4 years). Most patients were managed in the PED until resolution of DKA (95.3%). Moderate and severe DKA accounted for 71% of cases. Complications occurred in 27 patients (15.9%). Although tachycardia, respiratory abnormalities, decreased Glasgow Coma Scale scores (≤14), and electrolyte abnormalities were more frequent among patients admitted to the PICU, most patients with these findings were successfully managed in the PED. Patients requiring PICU admission had significantly lower pH levels, lower bicarbonate concentrations, more frequent neurologic impairment, and a higher rate of electrolyte abnormalities.

Conclusion: Most pediatric patients with DKA can be safely managed outside the PICU when standardized protocols and close clinical monitoring are implemented. A selective PICU admission strategy based on clinical indicators of deterioration may therefore help optimize the use of limited intensive care resources while maintaining patient safety.

Keywords: Diabetic ketoacidosis, Child, Emergency treatment, Diabetes mellitus

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Introduction

Diabetic ketoacidosis (DKA) is a potentially life-threatening metabolic complication of diabetes mellitus (DM) in children. The global incidence of type 1 diabetes (T1D) is reported to be 15 per 100,000 individuals. Nearly half of the children with newly diagnosed diabetes present with DKA [1, 2]. The prevalence of T1D and the proportion of DKA at diagnosis have increased over time, with a shift toward more severe presentations [3].

The treatment of DKA is often provided in pediatric intensive care units (PICUs) for close monitoring in many countries, particularly in cases of severe ketoacidosis. However, several studies have demonstrated that, in centers with experienced staff and standardized treatment protocols, selected patients with severe DKA can be safely managed outside the PICU with close clinical observation, frequent neurologic assessment, and serial laboratory monitoring. Admission to the PICU is generally recommended for children with DKA who are at high risk of clinical deterioration or require advanced monitoring. According to the International Society for Pediatric and Adolescent Diabetes (ISPAD) Clinical Practice Consensus Guidelines, PICU admission should be considered in patients with severe DKA accompanied by significant neurologic impairment, suspected or confirmed cerebral edema, hemodynamic instability, or the need for advanced cardiorespiratory monitoring and support [4]. Additional indications include Glasgow Coma Scale (GCS) score ≤12–13, recurrent vomiting with inability to protect the airway, severe electrolyte disturbances, or the presence of comorbid conditions requiring intensive monitoring. Young children, particularly those under 5 years of age, and patients presenting with severe acidosis, elevated blood urea nitrogen (BUN) levels, or prolonged symptom duration may also be considered at higher risk for cerebral edema and may benefit from management in a PICU or in units with equivalent monitoring capabilities [4]. Management of appropriate patients in non-intensive care settings both optimizes resource utilization and minimizes the psychological stress and potential complications associated with PICU admissions. Managing patients in inpatient wards also facilitates earlier initiation of diabetes education for families and rapid discharge of patients after resolution of DKA [5]. This approach can also decrease the cost per patient [6]. Estimates from the United States of America (USA) indicate that the average cost of hospital admission for DKA is 27,000

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USD [7]. In the USA, one in five children with DKA required intensive care, and median costs per child with diabetes were twice as high for those with DKA compared with those without [8]. Therefore, the decision for PICU admission should be individualized based on the severity of illness, risk of complications, and institutional resources.

Effective management outside the PICU is especially crucial during times of heightened global demand for PICU resources, such as the COVID-19 pandemic, which resulted in an unprecedented worldwide need for PICU beds [9]. During this period, reserving PICU beds for critically ill patients and managing appropriate cases outside the intensive care setting became increasingly necessary [10, 11]. In parallel, the incidence of diabetic ketoacidosis also increased during the COVID-19 pandemic [12]. These challenges underscore the need for efficient and safe management strategies that can be implemented outside the PICU when appropriate.

For several years, the majority of children with DKA in our hospital have been managed in the PED until metabolic acidosis is resolved and the IV insulin infusion can be discontinued. The management is based on a written protocol developed by the Turkish Society of Pediatric Emergency and Intensive Care Medicine, adapted from the ISPAD Guidelines [13]. The aim of the present study was to evaluate whether pediatric DKA can be safely managed outside the PICU under close clinical monitoring and standardized treatment protocols and to identify clinical factors associated with PICU admission.

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Materials and methods

Patients aged 0 to 18 years who presented to the PED of a tertiary care children's hospital with DKA between January 2017 and December 2024 were enrolled retrospectively. Patients were identified via an electronic medical record query using ICD-10 diagnostic codes corresponding to DKA and DM. Multiple admissions of the same patient at different times were included if they met the inclusion criteria. Diagnosis of DKA was based on ISPAD criteria [4]. Patients with comorbid conditions were excluded. For each patient, demographic features, type of diabetes (newly diagnosed or previously known), risk factors for DKA (young age, low socioeconomic status, lack of health insurance, refugee status, delayed diagnosis, infection, peripubertal and pubertal adolescent girls, psychiatric disorders, intentional

omission of insulin) were recorded. Clinical findings, vital signs, symptoms and GCS scores at admission were documented. Capillary refill times were categorized as <2 seconds, 2-5 seconds, or >5 seconds. Hemodynamic instability was defined as capillary refill time >2 seconds, weak pulses, cool extremities, tachycardia, and hypotension. Dehydration was estimated clinically: 5% for mild, 7% for moderate, and 10% for severe DKA [14-16]. Laboratory parameters (biochemical parameters and blood gas analysis) at admission were recorded.

To determine the severity of the disease, patients were divided into three subgroups according to the ISPAD Clinical Practice Consensus Guidelines; mild (venous pH <7.3 or bicarbonate <18 mmol/L), moderate (pH <7.2 or bicarbonate <10 mmol/L), and severe (pH <7.1 or bicarbonate <5 mmol/L) [4]. These three groups were compared in terms of demographic characteristics, clinical and laboratory findings, and outcomes. Pediatric intensive care unit admission, length of stay in the PED and pediatric endocrinology ward, complications associated with the treatment and DKA itself (cerebral edema, electrolyte and metabolic abnormalities, such as hypoglycemia, hypokalemia, hypomagnesemia, hypocalcemia, hyperchloremic metabolic acidosis, thrombosis, acute kidney injury (AKI), and pulmonary complications), transition time to subcutaneous insulin, and survival rates were recorded. Acute kidney injury was defined by using the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines [17]. Cerebral edema was diagnosed clinically by using the ISPAD criteria and confirmed with cranial imaging [4]. Pediatric intensive care admission was considered for the patients with suspected or confirmed cerebral edema, severe neurologic impairment (GCS ≤ 12–13), hemodynamic instability, severe electrolyte disturbances and need for advanced monitoring or organ support.

All patients were managed using a standardized institutional protocol derived from the ISPAD guidelines [13]. According to this protocol, all patients without hypovolemic shock received 10-20 mL/kg of 0.9% sodium chloride over the first 60 minutes of their treatment. The two-bag system is implemented as the standard of care in patients with DKA after bolus fluid treatment. The calculated fluid deficit was given over 48 hours, along with maintenance fluids. Forty millimoles of potassium were added per liter of fluid (20 mmol/L KCl + 20 mmol/L KPO_4). Subsequent potassium replacement therapy was based on serum potassium measurements. Maintenance fluids were calculated using the

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Holliday-Segar formula or 1500 mL/m^2. Continuous infusion of regular insulin was administered (0.1 unit/kg/h). Venous pH ≥7.3 and tolerating oral intake was used as the criterion for transitioning from IV to subcutaneous (SC) insulin. Intravenous insulin infusion was discontinued 15 to 30 minutes after the first injection of rapid-acting insulin. Blood glucose concentrations were monitored hourly while the patient was receiving IV insulin infusion. Venous pH, pCO_2, and bicarbonate were measured every 2-4 hours. Electrolytes, BUN and creatinine were measured less frequently (every 4-6 hours) unless significant derangements were present. Clinical parameters, including heart rate, respiratory rate, blood pressure, and oxygen saturation, were monitored continuously. Neurologic examinations, including GCS and pupil assessments, were evaluated hourly to detect possible cerebral edema.

In addition, a subgroup analysis including only patients with severe DKA was performed to compare clinical and laboratory characteristics between patients managed in the PED and those admitted to the PICU.

1. Ethical approval

The study protocol was designed in compliance with the Declaration of Helsinki. Ethics approval was obtained from the local clinical research ethics committee (2021/13-11).

2. Statistical analysis

Descriptive statistics were used to analyze all data. The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range (IQR)) depending on data distribution. Continuous variables were compared using the Student's t-test or Mann–Whitney U test, as appropriate. Comparisons among more than two groups were performed using the Kruskal–Wallis test. Categorical data are summarized as percentages and compared using the Chi-square test and Fisher's exact test as needed. Univariate analyses were performed to evaluate factors associated with PICU admission. Due to the small number of PICU admissions, multivariable logistic regression analysis was not performed. Statistical

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significance was defined as $p<0.05$. Statistical analysis was performed using IBM SPSS version 22.0 (released 2012, IBM SPSS Statistics for Windows; IBM Corp, Armonk, NY, USA).

Results

A total of 169 patients with a mean age of 10.3 ± 4.4 years and a female-to-male ratio of 1.6:1 were evaluated. Eighty-nine (52.7%) patients had newly diagnosed diabetes. Risk factors for the development of DKA were present in 104 patients (61.5%). The median duration from the onset of patients' symptoms was 48 hours (IQR: 24-168). Gastrointestinal symptoms were the most common presenting complaints, with abdominal pain reported in 64.5% of patients and nausea and vomiting in 58.6%. Respiratory abnormalities were present in 51 patients (30.2%). Polyuria and polydipsia were present in 78 patients (46.1%). The GCS score was below 14 in 10 patients at admission (5.9%). There were 49 children with mild DKA (29%), 49 with moderate DKA (29%), and 71 with severe DKA (42%). The demographic characteristics, and clinical and laboratory findings of the patients are presented in Table 1.

Complications related to DKA were observed in 27 patients (15.9%). Cerebral edema developed in one patient with severe DKA, and AKI occurred in four patients. Acute kidney injury was more common in patients with severe DKA. At presentation, patients who developed AKI had significantly higher levels of BUN (27.1 ± 5.9 vs. 12.6 ± 5.2; $p<0.001$), anion gap (26.7±4.8 vs. 19.9 ± 4.2; $p<0.001$), and lactate (4.3 ± 2.6 mmol/L vs. 2.2 ± 1.5 mmol/L; $p=0.017$) compared with those who did not develop AKI. However, blood glucose and corrected sodium levels did not differ significantly between the groups. None of the patients required dialysis. Electrolyte disturbances were detected in 24 patients (14.2%) during follow-up. Hypophosphatemia occurred in 12 patients (7.1%), hypokalemia in 13 (7.7%), hypomagnesemia in five (3.0%), and hypocalcemia in one patient (0.6%). No other complications, such as thrombosis or cardiac compromise, were observed, and no deaths occurred in the study cohort.

Eight (4.7%) patients with severe DKA were admitted to the PICU. Although tachycardia, neurologic impairment, respiratory or electrolyte abnormalities were more common among patients admitted to the PICU, the majority of patients with abnormal respiratory patterns (83.3%) and tachycardia (78.5%), as

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well as nearly half of those with a GCS score ≤14 were treated in the PED until resolution of DKA (Table 2). Five patients (62.5%) admitted to the PICU had complications including cerebral edema, AKI, or severe electrolyte disturbances (Table 2). A subgroup analysis including only patients with severe DKA (n=71) was performed to compare those managed in the PED with those admitted to the PICU. Patients requiring PICU admission had significantly lower pH levels, lower bicarbonate concentrations, more frequent neurologic impairment (GCS ≤14), and a higher rate of electrolyte abnormalities (Table 3). One-quarter of these patients were aged under 5 years, but there was no statistical difference in terms of age among patients who were or were not admitted to the PICU. Most patients with severe DKA who were younger than 5 years were managed in the PED (80%) (Table 3). Sodium bicarbonate therapy was administered only in the prehospital setting to six patients with severe DKA (pH range: 6.83–6.92). None of these patients had evidence of cardiac compromise. Bicarbonate treatment was not continued in the PED or during hospitalization. Length of stay in the PED did not differ significantly between patients who received bicarbonate and those who did not (p=0.69). However, patients treated with bicarbonate had a significantly longer hospital length of stay and prolonged duration of IV insulin therapy (p=0.007 and p=0.005, respectively). Furthermore, in a subgroup analysis restricted to patients with severe DKA (n=71), the duration of IV insulin infusion was significantly longer in patients who received bicarbonate compared with those who did not (34.2 ± 13.4 vs. 15.4 ± 6.4 hours; p=0.006).

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Discussion

Severe DKA is one of the most common indications for PICU admission worldwide, primarily due to the risk of rapid clinical deterioration and the development of life-threatening complications. However, managing DKA in the intensive care setting is associated with increased healthcare costs and substantial use of limited critical care resources. During periods of increased demand for intensive care services—such as the COVID-19 pandemic—routine PICU admission of all patients with severe DKA may further strain healthcare systems and limit bed availability for children with more critical illnesses requiring advanced organ support. Therefore, in settings where ICU resources are limited, patients with severe

DKA may be managed in the PED with close monitoring and treatment in accordance with established protocols.

The major complications that physicians must consider during the management of DKA include cerebral edema, thrombosis, acute kidney injury (AKI), and electrolyte disturbances. Among these, cerebral edema remains the most feared complication, as it represents the leading cause of DKA-related mortality in children. Cerebral edema occurs in approximately 0.5–1% of pediatric DKA cases and is associated with reported mortality rates of 20–25%, as well as a substantial risk of long-term neurological morbidity among survivors [5]. Clinically, cerebral edema should be suspected in patients with decreased levels of consciousness, abnormal motor or verbal responses to pain, decorticate or decerebrate posturing, altered mental status, cranial nerve palsy, bradycardia, age-inappropriate incontinence, or abnormal respiratory patterns [18]. Because neuroimaging findings may lag behind clinical deterioration, the diagnosis is primarily clinical, and treatment should not be delayed while awaiting radiologic confirmation. Therefore, careful neurological monitoring is essential during the management of DKA, and patients with suspected cerebral edema should be managed in the PICU [18]. In the present study, a decreased GCS score was observed in 5.9% of patients; however, only one patient developed clinically and radiologically confirmed cerebral edema and required PICU admission. Forty percent of patients with a GCS score ≤14 were managed in the PED because cerebral edema was not clinically suspected. After resolution of DKA, these patients were transferred to the pediatric endocrinology service with normal consciousness.

Particular caution is required when monitoring patients at increased risk for cerebral edema, including those younger than five years of age, with prolonged symptom duration, severe acidosis, elevated BUN levels, or low partial carbon dioxide pressure. The ISPAD guidelines recommend that such high-risk patients be managed in the PICU or in units with equivalent monitoring capacity and experienced supervision, such as the PED or a specialized pediatric endocrinology ward [19]. In the present study, 92% of patients younger than five years and 88.7% of those with severe acidosis were successfully managed in the PED under close monitoring.

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Adequate hydration is a cornerstone of DKA management. Although fluid administration has not been consistently associated with the development of cerebral edema, fluid composition and infusion rates have been linked to other complications, particularly AKI, as defined by the KDIGO criteria [20,21]. A recent systematic review reported that AKI of any stage occurred in approximately 47% of pediatric DKA episodes, with severe AKI observed in 28% of cases; however, only 7.9% of affected children required renal replacement therapy [22]. Several factors have been proposed to contribute to the development of AKI during DKA, including volume depletion during ketoacidosis, large-volume administration of 0.9% sodium chloride leading to hyperchloremia, severe hyperglycemia, acidosis, elevated uric acid levels, lower serum bicarbonate concentrations, lower corrected sodium levels, higher BUN at presentation, increased heart rate, and pre-existing chronic kidney disease [20–22]. In the present study, AKI occurred in four patients (2.4%). At presentation, patients who developed AKI had higher serum lactate levels, anion gap values, and BUN levels compared with those who did not develop AKI; however, no individual variable was independently associated with AKI development. Most patients with AKI were successfully managed in the pediatric emergency department (75%), and none required dialysis.

The accumulation of ketoacids in the extracellular fluid leads to a loss of bicarbonate anions and an increase in ketoacid anions [23]. Sodium bicarbonate administration is not part of routine pediatric DKA management and is not recommended by current international guidelines, except in rare cases of severe acidosis accompanied by hemodynamic instability or significant cardiac compromise [4]. Although bicarbonate therapy may transiently increase extracellular pH, it has been associated with several adverse effects, including paradoxical intracellular acidosis, reduced tissue oxygen delivery due to a leftward shift of the oxygen dissociation curve, hypokalemia, an increased risk of cerebral edema, enhanced ketogenesis, and delayed resolution of DKA [24]. Nevertheless, withholding bicarbonate therapy remains challenging for many clinicians, particularly in cases of severe ketoacidosis. In the present study, bicarbonate therapy was administered only in the prehospital setting to a small number of patients with severe DKA (pH 6.83–6.92) and was not continued after hospital admission. The length of stay in the PED did not differ significantly between patients who received bicarbonate therapy and

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those who did not. However, even among patients with severe ketoacidosis, those who received bicarbonate therapy had a significantly longer overall hospital stay and required a longer duration of intravenous insulin therapy. These findings further support current guideline recommendations discouraging routine bicarbonate use in pediatric DKA.

Lactic acidosis is commonly observed in pediatric DKA; however, its clinical significance remains unclear. Cully et al. reported no significant association between lactic acidosis and length of stay in the PICU or hospital, nor with morbidity or mortality [25]. Consistent with these findings, serum lactate levels in the present study were significantly higher in patients with moderate and severe DKA but were not associated with hospital length of stay, duration of intravenous insulin therapy, or the need for PICU admission. Notably, lactate levels were higher in patients who developed AKI, suggesting a potential association with greater disease severity.

The main strength of the present study is that all patients were managed using the same standardized treatment protocol [13]. As a tertiary referral center, our institution treats a wide spectrum of DKA severity, including a substantial proportion of severe cases. However, this study has several limitations. The first limitation of this study is its retrospective design, which relied on data obtained from medical records. Second, blood ketone measurements were not available at our institution during the study period. In our center, patients were monitored in the PED until resolution of DKA because pediatric emergency physicians and nurses have extensive experience in managing critically ill children. Consequently, the length of stay in the PED was relatively longer. In settings where inpatient wards have adequately trained medical and nursing staff experienced in pediatric DKA management, these patients may also be safely managed in such units by following established clinical practice guidelines, particularly in pediatric wards specializing in diabetes care. This approach may help reduce the length of stay in the PED.

In conclusion, our findings suggest that most pediatric patients with DKA, including selected cases of severe DKA, can be safely managed outside the PICU with appropriate monitoring and standardized treatment protocols. Although severe metabolic derangements were common in our cohort, only a small proportion of patients required PICU admission. Neurologic impairment, deeper metabolic acidosis,

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and significant electrolyte abnormalities were the main factors associated with intensive care requirement. These findings suggest that PICU admission in pediatric DKA should be based on clinical indicators of deterioration rather than biochemical severity alone. A selective approach to intensive care utilization may allow safe management of many pediatric DKA cases while preserving critical care resources for patients requiring advanced support. Future prospective multicenter studies are needed to further validate clinical criteria for safe management of pediatric DKA outside the PICU.

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Table footnotes:

Table 1: Values are presented as number (%) or mean ± standard deviation. $p < 0.05$ indicates statistical significance.

Abbreviations: DKA, diabetic ketoacidosis; GCS, Glasgow Coma Scale; BUN, blood urea nitrogen; HbA1c, hemoglobin A1c; IV, intravenous; LOS, length of stay; PED, pediatric emergency department.

Table 2: Values are presented as number (%), mean ± standard deviation or median (interquartile range). $p < 0.05$ indicates statistical significance.

Abbreviations: ED, emergency department; PICU, pediatric intensive care unit; DKA, diabetic ketoacidosis; GCS, Glasgow Coma Scale; HbA1c, hemoglobin A1c; BUN, blood urea nitrogen; IV, intravenous; LOS, length of stay.

Table 3: Values are presented as number (%) or mean ± standard deviation. $p < 0.05$ indicates statistical significance.

Abbreviations: ED, emergency department; PICU, pediatric intensive care unit; GCS, Glasgow Coma Scale; BUN, blood urea nitrogen; IV, intravenous; LOS, length of stay.

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Table 1: Clinical and laboratory characteristics of patients according to diabetic ketoacidosis severity

	Mild DKA (n=49)	Moderate DKA (n=49)	Severe DKA (n=71)	*p*
Age (years)	8.9 ± 4.4	11.2 ± 4.0	10.8 ± 4.5	**0.026**
Female sex	30 (61.2%)	31 (63.3%)	43 (60.6%)	0.955
New-onset diabetes	28 (57.1%)	23 (46.9%)	37 (51.4%)	0.591
Presence of a risk factor	24 (48.9%)	27 (55.1%)	53 (74.6%)	0.108
Signs and Symptoms				
Heart rate (beats/min)	109.5 ± 13.4	112.7 ± 21.5	125.4 ± 19.8	**0.001**
Polyuria/polydipsia	26 (53.0%)	21 (42.8%)	31 (43.7%)	0.562
GCS≤14 at admission	-	1 (2.3%)	9 (12.7%)	**0.003**
Tachypnea	4 (8.1%)	5 (10.2%)	42 (59.2%)	**<0.001**
Abdominal pain	26 (53.0%)	33 (67.3%)	50 (70.4%)	0.101
Vomiting/nausea	20 (40.8%)	29 (59.1%)	50 (70.4%)	**0.007**
Laboratory				
Glucose (mg/dL)	403.6 ± 172.1	393.0 ± 152.4	477.3 ± 164.9	**0.008**
Venous pH	7.25 ± 0.03	7.15 ± 0.04	6.98 ± 0.09	**<0.001**
pCO_2 (mm Hg)	25.5 ± 6.2	23.8 ± 5.6	20.4 ± 5.7	**<0.001**
Bicarbonate (mmol/L)	11.4 ± 2.4	8.8 ± 2.0	5.4 ± 2.0	**<0.001**
Lactate (mmol/L)	1.8 ± 1.2	2.4 ± 1.8	2.6 ± 1.3	**<0.001**
Anion gap	17.6 ± 3.5	20.6 ± 8.3	23.3 ± 4.0	**<0.001**
Corrected sodium (mmol/L)	138.2 ± 3.4	138.9 ± 4.1	139.4 ± 3.9	0.346
Potassium (mmol/L)	4.5 ± 0.8	4.4 ± 0.8	4.7 ± 0.9	**<0.001**
Phosphorus (mg/dL)	3.6 ± 1.3	3.6 ± 1.3	4.5 ± 2.9	0.076
Magnesium (mg/dL)	1.9 ± 0.2	1.9 ± 0.2	2.1 ± 0.3	**<0.001**
Creatinine (mg/dL)	0.9 ± 0.2	1.04 ± 0.29	1.17 ± 0.32	**<0.001**
BUN (mg/dL)	11.9 ± 5.4	13.4 ± 5.5	14.9 ± 7.2	0.098
HbA1c (%)	11.8 ± 1.6	11.6 ± 1.3	11.9 ± 1.9	0.620
Complications				
Cerebral edema	-	-	1 (1.4%)	-
Acute kidney injury	-	2 (4.5%)	2 (2.8%)	0.510
Electrolyte imbalance	4 (8.1%)	3 (6.1%)	17 (23.9%)	**0.010**
Outcomes				
LOS in PED (hours)	16.8 ± 12.0	20.8 ± 13.8	23.7 ± 16.7	**0.018**

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LOS in hospital (hours)	193.8 ± 99.5	162.9 ± 104.6	182.8 ± 118.2	0.247
IV insulin duration (hours)	7.8 ± 5.5	8.5 ± 4.7	16.3 ± 9.9	**<0.001**

Values are presented as number (%) or mean ± standard deviation. $p < 0.05$ indicates statistical significance.

Abbreviations: DKA, diabetic ketoacidosis; GCS, Glasgow Coma Scale; BUN, blood urea nitrogen; HbA1c, hemoglobin A1c; IV, intravenous; LOS, length of stay; PED, pediatric emergency department.

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Table 2: Clinical characteristics of patients managed in the pediatric emergency department and those admitted to the pediatric intensive care unit

	Pediatric ED (n = 161)	**PICU (n=8)**	***p***
Age (years)	10.4 ± 4.4	9.9 ± 5.7	0.982
Age <5 years	23 (14.3%)	2 (25%)	0.406
Severe DKA	63 (39.1%)	8 (100%)	**0.002**
Clinical findings			
Abnormal respiratory pattern	43 (26.7%)	8 (100%)	**<0.001**
Heart rate (beats/min)	115.4 ± 18.6	133.9 ± 24.0	**0.017**
GCS ≤14 at admission	4 (2.5%)	5 (62.5%)	**<0.001**
Duration of symptoms (hours), median (IQR)	48 (24;168)	48 (24;66)	0.069
Laboratory findings			
Venous pH	7.11 ± 0.12	6.92 ± 0.07	**<0.001**
Bicarbonate (mmol/L)	8.3 ± 3.2	4.2 ± 1.2	**<0.001**
Lactate (mmol/L)	2.3 ± 1.5	2.6 ± 1.0	0.164
Anion gap	20.7 ± 6.0	22.4 ± 4.7	0.293
HbA1c (%)	11.6 ± 1.6	11.5 ± 0.9	0.619
Creatinine (mg/dL)	1.05 ± 0.32	1.09 ± 0.22	0.475
BUN (mg/dL)	13.6 ± 6.4	14.1 ± 4.6	0.544
Complication			
Cerebral edema	-	1 (12.5%)	-
Acute kidney injury	3 (1.8%)	1 (12.5%)	0.691
Electrolyte imbalance	19 (11.8%)	5 (62.5%)	**0.002**
Outcomes			
Duration of IV insulin (hours)	10.4 ± 6.3	27.3 ± 17.0	**0.005**
Hospital LOS (hours)	176.3 ± 108.4	258.0 ± 97.5	**0.027**

Values are presented as number (%), mean ± standard deviation or median (interquartile range). $p < 0.05$ indicates statistical significance.

Abbreviations: ED, emergency department; PICU, pediatric intensive care unit; DKA, diabetic ketoacidosis; GCS, Glasgow Coma Scale; HbA1c, hemoglobin A1c; BUN, blood urea nitrogen; IV, intravenous; LOS, length of stay.

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Table 3. Clinical characteristics of severe diabetic ketoacidosis cases managed in the pediatric emergency department compared with those requiring pediatric intensive care

	Pediatric ED (n = 63)	**PICU (n=8)**	***p***
Age (years)	10.9 ± 4.4	9.9 ± 5.7	0.627
Age <5 years	8 (12.7%)	2 (25.0%)	0.117
Female sex	37 (58.7%)	6 (75.0%)	0.466
Newly diagnosed diabetes	33 (52.4%)	5 (62.5%)	0.716
Clinical findings			
Abnormal respiratory pattern	34 (54.0%)	8 (100%)	**0.044**
Heart rate (beats/min)	123.5 ± 18.6	133.9 ± 24.0	0.282
GCS ≤14 at admission	4 (6.3%)	5 (62.5%)	**<0.001**
Laboratory findings			
Venous pH	6.99 ± 0.09	6.92 ± 0.07	**0.042**
Bicarbonate (mmol/L)	5.5 ± 2.0	4.2 ± 1.2	**0.023**
Lactate (mmol/L)	2.6 ± 1.4	2.6 ± 1.0	0.987
Creatinine (mg/dL)	1.18 ± 0.33	1.09 ± 0.22	0.321
BUN (mg/dL)	14.9 ± 7.5	14.1 ± 4.6	0.640
Complications			
Cerebral edema	-	1 (12.5%)	-
Acute kidney injury	2 (3.2%)	1 (12.5%)	0.280
Electrolyte imbalance	12 (19.0%)	5 (62.5%)	**0.030**
Outcomes			
Duration of IV insulin (hours)	14.1 ± 6.3	27.3 ± 17.1	**<0.001**
Hospital LOS (hours)	173.1 ± 117.8	258.0 ± 97.5	**0.048**

Values are presented as number (%) or mean ± standard deviation. $p < 0.05$ indicates statistical significance.
Abbreviations: ED, emergency department; PICU, pediatric intensive care unit; GCS, Glasgow Coma Scale; BUN, blood urea nitrogen; IV, intravenous; LOS, length of stay.

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