ORIGINAL RESEARCH

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Evaluation of the Relationship Between the Change in Base Excess and Clinical Conditions in Patients with Diabetic Ketoacidosis

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Abstract

Objective: The aim of this study was to evaluate the post-treatment change in base excess (ΔBE) in diabetic ketoacidosis (DKA) and to assess how both the magnitude of correction and the resulting acid–base status relate to presenting severity and routine laboratory parameters.

Materials and Methods: We retrospectively analyzed 202 adults presenting with DKA to a tertiary emergency department between 2020 and 2025 who underwent blood gas analysis at presentation and after treatment. ΔBE was defined as post-treatment minus baseline base excess. Comparisons were performed using the Mann–Whitney U and Kruskal–Wallis tests, and associations were assessed using Spearman's rho.

Results: The median age was 39 years, and 59.9% were male. At presentation, the median pH was 7.08 and HCO_3 was 7.7 mmol/L, indicating marked acidemia. Following treatment, the median ΔBE was 11.9 mmol/L. The magnitude of correction increased with the depth of presenting acidosis (Spearman's rho=−0.75) and was greater in patients with higher baseline white blood cell counts, glucose, urea, creatinine, and C-reactive protein levels and in those admitted to the intensive care unit versus the ward (16.1 vs 7.8 mmol/L, p<0.001). Although ΔBE differed according to presenting severity (p=0.026), post-treatment base excess was statistically similar across severity strata (p=0.374), indicating that treatment brought patients to a comparable acid–base endpoint regardless of the severity at presentation.

Conclusion: In DKA, the change in base excess following treatment (ΔBE) is a parameter that can be easily derived from laboratory data and is associated with patients' clinical status.

Keywords: Acidosis, base excess, blood gas analysis, critical care, diabetic ketoacidosis

Introduction

Diabetic ketoacidosis (DKA) is more than a simple complication; it is a life-threatening diabetic emergency. Developing against a background of insulin deficiency and increased levels of counter-regulatory hormones, it is characterized by hyperglycemia, ketosis, and anion-gap metabolic acidosis and requires rapid diagnosis and treatment [1,2]. Although classically associated with type 1 diabetes, DKA can also occur in the context of type 2 diabetes [2]. Most patients with DKA first present to the emergency department, where diagnosis, severity assessment, and risk stratification are largely performed. Despite advances in diagnostic and treatment options, DKA remains a significant cause of morbidity, mortality, and healthcare expenditure among patients with diabetes, and its diagnosis and management continue to be actively debated [3]. This highlights the importance of both early diagnosis and objective monitoring of treatment response.

The latest international consensus report on the diagnosis and treatment of DKA, published in 2024, has redefined the criteria for diagnosis and resolution. In this report, the anion gap has been removed from the diagnostic criteria; beta-hydroxybutyrate measurement has been placed at the center of diagnosis, severity assessment, and monitoring of resolution; the treatment regimen has been simplified to focus on the fluid–insulin–potassium axis; and, in the assessment of acid–base status, arterial blood gas

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sampling has been deemed unnecessary, with venous sampling considered sufficient [1,3]. These changes have increased interest in integrated acid–base parameters, which can be readily obtained from venous blood gas analysis, during the process of suppressing ketone production and correcting metabolic acidosis, the primary treatment objectives [1,4].

Various blood gas parameters are used to assess the severity and course of acidosis. Base excess is a measure that reflects the degree of metabolic acid–base disturbance as a single numerical value and is automatically reported in every blood gas analysis [5]. In DKA, baseline base excess has been shown to be more useful than the anion gap in predicting disease severity and the duration of acidosis resolution and to be unaffected by short-term changes in carbon dioxide pressure [6]. Furthermore, although base excess is not included in current severity classifications, it may contribute to risk stratification [7].

The extent to which acidosis resolves in a critically ill patient may be as informative as the initial severity. In patients with sepsis and those in intensive care, the rate at which lactate is cleared over a specific period (lactate clearance) is a widely used indicator [8]; similarly, base excess has been reported to be superior to pH in assessing the resolution of acidosis following traumatic shock [9] and to have prognostic value in intensive care [10,11]. In DKA, high lactate levels have also been associated with adverse outcomes [12]. However, while lactate is not routinely measured in every center or by every blood gas analyzer, base excess is universally available.

It has been reported that the median resolution time for DKA is approximately 18 hours, although this may vary among patients from 2 to 32 hours [6]; such interpatient variation points to determinants of treatment response that remain poorly understood. Nevertheless, existing studies have primarily focused on the predictive value of base excess at presentation; the magnitude of the change in base excess following treatment and the clinical determinants of this change have not been specifically investigated. In this study, we aimed to characterize the change in base excess (ΔBE) following treatment in patients with DKA and to evaluate its relationship with patients' clinical and laboratory status.

Materials and Methods

Study Design and Patient Selection

This single-center, retrospective, observational cohort study included adult patients (aged ≥18 years) who presented to the emergency department of a tertiary teaching and research hospital with diabetic ketoacidosis between March 1, 2020, and March 1, 2025. The diagnosis of DKA was made in accordance with current guidelines [1], based on the combination of hyperglycemia (blood glucose >250 mg/dL), metabolic acidosis (pH <7.30, HCO_3 <18 mmol/L), and ketosis (documented by urine or blood ketone positivity). Because the study period spanned the 2024 revision of the diagnostic criteria, the diagnosis rested primarily on the combination of metabolic acidosis and ketosis; the blood glucose value of 250 mg/dL was applied as a supporting threshold rather than a strict cutoff, and lower-glucose or euglycemic ketoacidosis presentations that met the acidosis and ketosis criteria were not excluded. A total of 245 adults presenting with DKA during the study period were evaluated. Forty-three patients were excluded: those under 18 years of age, those who died within the first 24 hours, and those with incomplete data. To enable calculation of the change in base excess, each patient was required to have at least two blood gas measurements, one at presentation and one following treatment. The 202 cases meeting these criteria were analyzed (Fig. 1). The study was reported in accordance with the STROBE guideline for observational studies.

Data Collection and Definitions

Demographic data, type of diabetes, admission laboratory values, serial blood gas measurements, admission unit (intensive care unit/ward), length of stay, and in-hospital mortality were obtained from the hospital's electronic records system. The severity of presentation was classified as mild, moderate, or severe according to the American Diabetes Association criteria, based on the pH value at presentation [1]. Diabetes type was recorded as type 1, type 2, or other/unspecified from the diagnostic records in the

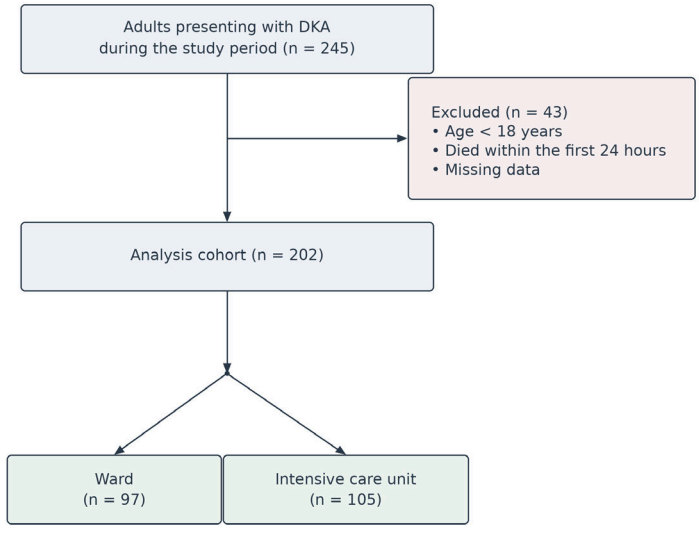

Figure 1. Study flow chart. Of the 245 adults who presented with diabetic ketoacidosis during the study period, 43 were excluded (those under 18 years of age, those who died within the first 24 hours, and those with incomplete data), leaving 202 cases for analysis; of these, 97 were managed on the ward and 105 in the intensive care unit.

hospital's electronic records system. Base excess is a parameter calculated from blood gas analysis that reflects metabolic acid–base status [13,14]; in this study, base excess and other blood gas values were obtained from venous or arterial blood gas analysis. Although venous and arterial blood gas measurements have been reported to agree closely for bicarbonate and overall acid–base parameters [15,16], this agreement has not been demonstrated specifically for base excess. For each patient, blood gas base excess values at presentation (presentation BE) and after treatment (post-treatment BE) were recorded; post-treatment base excess was defined as the last blood gas measurement obtained during the treatment course following admission. The change in base excess, ΔBE, was defined as post-treatment BE−presentation BE; percentage improvement was calculated as ΔBE/|presentation BE|×100. In cases where post-treatment base excess reached positive values, this percentage could exceed 100%.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA). The normality of distributions was assessed using the Shapiro–Wilk test. Continuous variables that did not follow a normal distribution were summarized as medians with 25th and 75th percentiles, and two groups were compared using the Mann–Whitney U test. The Kruskal–Wallis test was used to compare three independent groups, such as the presentation severity strata. Categorical variables were presented as counts (percentages), and groups were compared using the chi-square or Fisher's exact test. Relationships between continuous variables were examined using Spearman's rank correlation coefficient (rho). A p value of <0.05 was considered statistically significant.

Ethics

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the local ethics committee (approval no. E-62190176-663.05-269193889, dated August 7, 2024). Owing to the retrospective design, the requirement for informed consent was waived.

Results

Cohort Characteristics

Following the exclusion of 43 of the 245 patients who presented during the study period, 202 cases of DKA were analyzed (Fig. 1); of these, 97 (48.0%) were managed on the ward and 105 (52.0%) in the intensive care unit. The median age of the patients was 39 (26–52) years, and 59.9% were male. Regarding diabetes type, 26.2% of cases were recorded as type 1 and 7.4% as type 2; the remaining 66.3% were coded as other or unspecified. On admission, the median pH was 7.08 (6.90–7.19), and HCO_3 was 7.7 (4.0–11.0) mmol/L. Baseline characteristics by admission unit are presented in Table 1. Intensive care unit patients were older than ward patients and, at admission, had more severe acidosis and higher white blood cell counts and glucose, urea, creatinine, and C-reactive protein (CRP) levels.

Change in Base Excess from Presentation to Post-Treatment

While the median base excess (BE) at presentation was −15.9 (−21.9, −11.9) mmol/L, it increased to −3.4 (−7.3, 0.6) mmol/L after treatment. The corresponding median change in base excess (ΔBE) was 11.9 (7.0–17.7) mmol/L, indicating a median improvement of 77% in baseline base excess.

Variables Associated with the Change in Base Excess

Spearman's rho values between ΔBE and admission and clinical variables are summarized in Table 2. ΔBE showed the strongest association with the severity of acidosis at presentation: there was a strong negative correlation between baseline base excess and ΔBE (rho=−0.75, p<0.001); the more severe the acidosis at presentation, the greater the improvement (Fig. 2). Similarly, ΔBE was negatively correlated with baseline HCO_3 (rho=−0.47, p<0.001) and baseline pH (rho=−0.19, p=0.007). ΔBE was also positively correlated with admission white blood cell count (rho=+0.38), glucose (rho=+0.30), urea (rho=+0.20), creatinine (rho=+0.18), and CRP (rho=+0.15) (all p<0.05). No significant relationship was found with age, sodium, potassium, or length of stay.

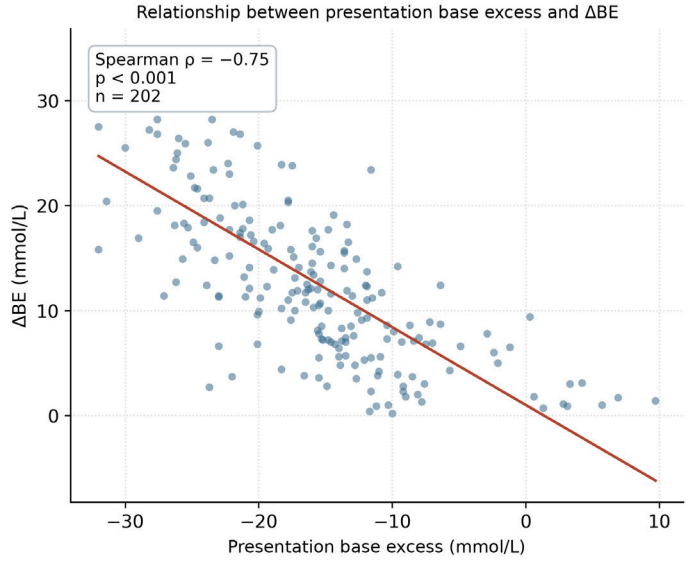

Figure 2. Relationship between presentation base excess and the change in base excess (ΔBE). Each point represents a patient. As acidosis at presentation deepens, ΔBE increases (Spearman's rho = −0.75, p < 0.001; n = 202). The solid line indicates the linear trend.

Table 1. Characteristics of the overall cohort and subgroups by admission unit

Variable	Overall (n=202)	Ward (n=97)	ICU (n=105)	p
Age, years	39 (26–52)	33 (25–48)	45 (28–57)	0.004
Male, n (%)	121 (59.9)	64 (66.0)	57 (54.3)	0.121
DM type, n (%)				0.008
Type 1	53 (26.2)	34 (35.1)	19 (18.1)	
Type 2	15 (7.4)	9 (9.3)	6 (5.7)	
Other/unspecified	134 (66.3)	54 (55.7)	80 (76.2)	
Severity at presentation				
Presentation pH	7.08 (6.90–7.19)	7.10 (7.00–7.20)	7.00 (6.90–7.18)	0.022
Presentation HCO_3, mmol/L	7.7 (4.0–11.0)	9.7 (5.3–12.6)	7.1 (3.9–9.7)	<0.001
Severe / Moderate / Mild, n	68 / 117 / 17	22 / 66 / 9	46 / 51 / 8	
Base excess				
Presentation BE, mmol/L	−15.9 (−21.9, −11.9)	−13.4 (−16.0, −9.3)	−20.4 (−24.1, −15.5)	<0.001
Post-treatment BE, mmol/L	−3.4 (−7.3, 0.6)	−4.2 (−7.6, −0.1)	−2.9 (−6.8, 1.7)	0.125
ΔBE, mmol/L	11.9 (7.0–17.7)	7.8 (4.8–11.9)	16.1 (11.3–23.0)	<0.001
Improvement, %	77 (51–100)	68 (42–92)	83 (64–103)	0.005
Admission laboratory				
White blood cells, 10^3/μL	13.3 (9.7–18.0)	10.4 (7.2–14.4)	16.0 (12.2–21.4)	<0.001
Glucose, mg/dL	255 (187–319)	208 (156–273)	287 (210–367)	<0.001
Urea, mg/dL	28 (18–50)	21 (15–30)	42 (26–60)	<0.001
Creatinine, mg/dL	0.85 (0.67–1.09)	0.75 (0.62–0.96)	1.00 (0.77–1.32)	<0.001
Sodium, mmol/L	135 (133–138)	135 (133–137)	135 (132–141)	0.533
Potassium, mmol/L	4.08 (3.61–4.58)	4.08 (3.64–4.43)	4.14 (3.60–4.92)	0.359
CRP, mg/L	14.2 (4.9–73.8)	10.9 (3.3–24.0)	25.0 (6.9–124.7)	<0.001
Clinical course				
Length of stay, days	3.0 (2.0–4.0)	3.0 (2.0–5.0)	2.0 (2.0–4.0)	0.263
In-hospital mortality, n (%)	8 (4.0)	1 (1.0)	7 (6.7)	0.067

BE: base excess; ΔBE: post-treatment BE − presentation BE; CRP: C-reactive protein; ICU: intensive care unit. Continuous variables are presented as median (25th–75th percentile) and categorical variables as count (%). p-values were obtained from the Mann-Whitney U or chi-square/Fisher test.

Change in Base Excess According to Severity at Presentation

The base excess variables according to severity at presentation are presented in Table 3. Base excess at presentation differed significantly between the severity groups (Kruskal–Wallis p=0.011), with the most severe acidosis observed in the severe group. In parallel, ΔBE also differed between the groups (p=0.026), with the greatest improvement observed in the severe group. In contrast, post-treatment base excess (p=0.374) and percentage improvement (p=0.429) were similar across the severity groups.

Change in Base Excess by Admission Unit

The change in base excess was significantly higher in intensive care cases than in ward cases: the median ΔBE was 7.8 (4.8–11.9) mmol/L on the ward, whereas it was 16.1 (11.3–23.0) mmol/L in the intensive care unit (p<0.001) (Fig. 3). Similarly, the percentage improvement was higher in the intensive care unit (83% vs. 68%; p=0.005). This difference was consistent with intensive care patients presenting with more severe acidosis (a more negative baseline BE) on admission (Table 1).

Discussion

In this retrospective cohort, we demonstrated that treatment produced a median improvement of 77% in baseline base excess in patients admitted with DKA. The magnitude of the change in base excess (ΔBE) was largely determined by the severity of acidosis at presentation, was associated with laboratory markers at presentation, and showed marked differences according to the admission unit. These findings suggest that ΔBE is an easily obtainable indicator in DKA that reflects patients' clinical condition.

The relationship between ΔBE and severity at presentation was as expected: the lower the baseline base excess, bicarbonate, and pH at presentation, the greater the improvement achieved with treatment. A substantial part of this relationship is conceptual because, by definition, the amount of base excess to be corrected is greater in patients presenting with more severe acidosis. The positive correlation between ΔBE and admission markers such as white blood cell count, CRP, glucose, urea, and creatinine is also consistent with this framework: in patients with more severe illness and more pronounced metabolic disturbances, these markers are elevated and the amount of base excess corrected is greater. ΔBE therefore tracks the overall disease burden and is not an independent marker in its own right.

Table 2. Spearman's rho values between ΔBE and the admission and clinical variables (n=202)

Variable	rho	p
Presentation HCO_3	−0.47	<0.001
White blood cells	+0.38	<0.001
Glucose	+0.30	<0.001
Urea	+0.20	0.010
Presentation pH	−0.19	0.007
Creatinine	+0.18	0.018
CRP	+0.15	0.036
Potassium	+0.13	0.074
Length of stay	+0.09	0.187
Age	+0.06	0.421
Sodium	−0.05	0.539

rho: Spearman's rank correlation coefficient. Positive values indicate an association with a greater ΔBE. ΔBE: post-treatment BE − presentation BE. The baseline base excess is not included in the table, as it is associated with ΔBE by definition.

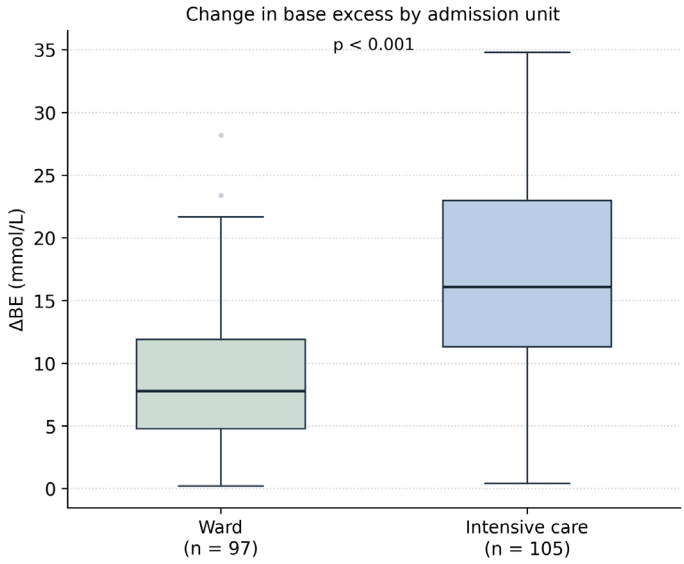

Figure 3. Change in base excess (ΔBE) by admission unit. Boxes show the median and interquartile range, and whiskers show the extremes of the distribution. ΔBE is higher in intensive care cases than in ward cases (Mann-Whitney U, p < 0.001).

Our findings are complementary to previous studies examining base excess in DKA. Jasso-Avila et al. [6] reported that, in a cohort of 79 adult patients, baseline pH, bicarbonate, and base excess predicted the duration of DKA resolution; that a lower baseline base excess was associated with a longer resolution time; and that base excess was superior to the anion gap for this purpose. Güzel et al. [7] correlated base excess with the American Diabetes Association severity strata using serial venous blood gas measurements and demonstrated that base excess contributes to risk stratification. While these studies focused on the predictive value of baseline base excess, we addressed a complementary question: we quantified the change in base excess (ΔBE) in response to treatment, as well as its clinical determinants, in a

Table 3. Base excess variables according to severity strata at presentation.

Variable	Severe (n=68)	Moderate (n=117)	Mild (n=17)	p
Presentation BE, mmol/L	−19.3 (−24.1, −13.6)	−15.5 (−21.0, −11.5)	−14.6 (−15.9, −12.0)	0.011
Post-treatment BE, mmol/L	−3.2 (−7.4, 2.1)	−3.2 (−7.2, 0.0)	−4.6 (−7.6, −2.1)	0.374
ΔBE, mmol/L	15.2 (8.6, 20.5)	11.4 (6.5, 16.6)	8.5 (7.1, 12.5)	0.026
Improvement, %	80 (53, 106)	78 (49, 99)	65 (51, 85)	0.429

BE: base excess; ΔBE: post-treatment BE − presentation BE. Severity was classified according to American Diabetes Association criteria based on presentation pH (severe: pH < 7.0; moderate: 7.0–7.24; mild: 7.25–7.30). Values are median (25th–75th percentile). p-values were obtained from the Kruskal-Wallis test.

relatively large cohort. The fact that the severity of acidosis on admission determines ΔBE (rho=−0.75 for baseline base excess) links these two observations: the lower the baseline base excess on admission, the longer resolution may take and the greater the correction required.

The mechanism underlying the improvement in base excess with treatment can be explained by the pathophysiology of DKA. In DKA, the fundamental problem is lipolysis triggered by insulin deficiency and an increase in counter-regulatory hormones; beta-oxidation of free fatty acids in the liver produces acetoacetate and beta-hydroxybutyrate, both strong acids, and the hydrogen ions released during their breakdown deplete the bicarbonate buffer, leading to anion-gap metabolic acidosis [1–4]. Insulin therapy halts ketogenesis; bicarbonate is regenerated through the metabolism of accumulated keto-anions, and the improvement in tissue perfusion following fluid therapy reduces the lactate load. The combined effect of these processes is observed as a substantial improvement in base excess, shifting from the deeply negative values seen on admission to near-normal levels following treatment.

However, in most cases, base excess did not return to normal following treatment (median −3.4 mmol/L). One reason for this is that chloride-rich fluids, commonly used in the treatment of DKA, can cause iatrogenic hyperchloremic metabolic acidosis, thereby partially masking the expected resolution of acidosis [17]. Indeed, this is one of the reasons why the 2024 consensus removed the anion gap from the diagnostic criteria: hyperchloremic acidosis and mixed acid–base disorders developing during treatment with isotonic fluids can confound the anion gap and, indirectly, base excess, thereby complicating the assessment of resolution [1]. For this reason, post-treatment base excess should be interpreted as a composite measure reflecting not only residual ketoacidosis but also the effect of the administered fluid therapy on acid–base balance.

The severity-stratified analysis is consistent with this: while the absolute improvement (ΔBE) was greatest in patients with the most severe acidosis at presentation, the level of base excess achieved post-treatment and the percentage improvement were similar across the strata (Table 3). This pattern can be interpreted as treatment steering patients of varying severity toward a common acid–base target; indeed, current guidelines define the resolution of DKA as the suppression of ketonemia and the normalization of acid–base parameters [1]. Consequently, while the difference in severity at presentation is reflected in the magnitude of the absolute improvement, the level achieved post-treatment more closely reflects the common treatment target.

The fact that ΔBE is approximately twice as high in intensive care cases as in ward cases can be explained by two factors. First, patients admitted to intensive care presented with more severe acidosis; consequently, the correction required was greater. Second, the more intensive treatment administered in intensive care (insulin infusion and strict fluid and electrolyte monitoring) may have contributed to faster and near-complete correction of the acidosis. It is known that the use of intensive care in DKA varies between centers and is generally required in more severe cases [18–20]. For this reason, ΔBE should be interpreted here not as a prognostic tool but as an indicator of disease severity and intensity of care; concluding that ΔBE predicts the need for intensive care would be misleading, as patients are admitted to intensive care precisely because they are already in a severe condition.

Assessing the rate of acidosis resolution is an approach that has long been used in sepsis and trauma through lactate clearance [8] and base excess [9–11]; our study applies this concept of resolution to base excess in DKA. Although high lactate levels in DKA have been shown to be associated with adverse outcomes [12], lactate is not always measured; the availability of base excess in every blood gas analysis makes ΔBE an accessible monitoring parameter, particularly in settings where lactate is not routinely measured.

Approximately one-quarter of our cases were registered as having type 1 diabetes. As adult diabetic ketoacidosis can occur in the context of both type 1 and type 2 diabetes [21,22], whether the change in base excess differs according to diabetes type is a question that requires further investigation.

From a clinical perspective, ΔBE is a measure that can be obtained from every blood gas analysis already performed, without requiring any additional cost or intervention, and can support monitoring as a practical indicator summarizing the severity of the patient's presentation and the intensity of care. The fact that the 2024 consensus renders arterial sampling unnecessary for acid–base assessment makes base excess derived from venous blood gas analysis even more useful in clinical monitoring [1]. However, the management of DKA, including fluid selection and insulin administration, remains an area of active research [23], and the treatment protocol employed may directly influence acid–base correction. For now, then, ΔBE is best regarded as a supporting marker of severity and treatment response; it has not been validated as an independent prognostic tool.

Study Limitations

This study has several limitations. Its single-center, retrospective design limits its generalizability. As diabetes type was not

specified in the records for approximately two-thirds of the cases, a type-specific analysis could not be performed. Because time data were recorded at the daily level, the hourly rate of improvement could not be calculated, and the timing of the post-treatment measurement could not be standardized; consequently, ΔBE reflects the course of care rather than a single time point. Furthermore, the more frequent collection of blood gas samples in intensive care cases may have introduced differences in measurement frequency between the groups. In addition, because base excess values were obtained from either venous or arterial blood gas samples, the mixture of sample types, which are not strictly equivalent and were not standardized in terms of type or timing, may be a further source of variability. As patients who died within the first 24 hours were excluded, the earliest and most severe fatal cases fell outside the ΔBE cohort, introducing selection bias whereby the analyzed sample underrepresents the most fulminant presentations. As the number of events was small, the association with mortality was not analyzed, and mortality is presented descriptively only. Finally, as ΔBE is mathematically dependent on baseline base excess at presentation, a model including baseline base excess as a predictor would be tautological; therefore, ΔBE was not modeled as a prognostic outcome but was addressed at a descriptive and correlational level.

Conclusion

In diabetic ketoacidosis, the change in base excess (ΔBE) following treatment is a parameter that can be easily obtained from laboratory data and is associated with patients' clinical status. Whether ΔBE has independent prognostic value should be investigated in future studies involving serial measurements with precisely recorded measurement times and outcome data.

Ethics

Ethics Committee Approval: This study was approved by The Ethics committe of University of Health Sciences, Taksim Training and Research Hospital (approval no. E-62190176-663.05-269193889, dated 7 August 2024).

Informed Consent: Owing to the retrospective design of the study, the requirement for written informed consent was waived by the ethics committee.

Conflict of Interest: The authors declare that there is no conflict of interest.

Use of AI for Writing Assistance: The authors declared that artificial intelligence was not used in the study.

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Authorship Contributions: Concept: S.Y.; Design: S.Y., M.T.; Supervision: S.Y.; Resource: S.Y.; Materials: S.Y.; Data Collection and/or Processing: S.Y.; Analysis and/or Interpretation: S.Y., M.T.; Literature Review: S.Y.; Writing: S.Y.; Critical Review: S.Y., M.T.

Peer-review: Externally peer-reviewed.

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