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The Correlation of Capillary Blood Ketone Test with Standard Parameters at the Presentation and the Resolution of Pediatric Diabetic Ketoacidosis

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Abstract: Background: The current criteria used to diagnose diabetic ketoacidosis have limitations due to their lack of specificity (bicarbonate and pH) and their qualitative nature (the presence of ketonemia/ketonuria). Currently, there are new techniques that can be used to assess capillary Beta-hydroxybutyrate at the bedside. This method is becoming more popular for diagnosing and treating diabetic ketoacidosis.

Objective: This study aimed to determine the link between the amount of capillary blood ketones and other standard parameters of diabetic ketoacidosis, including pH, venous bicarbonate, carbon dioxide, anion gap, and urine ketones, at the time of diagnosis and resolution.

Study participants and research methodology: A cross-sectional study was conducted on all patients, aged between 1 and 14 years old, who were admitted to the Department of Emergency in the Central Child Teaching Hospital, Baghdad City, Iraq, from March to July 2022. These patients met the diagnostic criteria for diabetic ketoacidosis and also fulfilled the inclusion criteria for this study. The patients were treated in accordance with the 2018 Clinical Practice Consensus Guidelines of the International Society for Pediatric and Adolescent Diabetes for diabetic ketoacidosis. They were monitored until they met the criteria for resolution of diabetic ketoacidosis and were able to tolerate oral fluids without experiencing nausea or vomiting. The capillary blood ketone levels were measured using the bedside eBketone Blood Ketone Monitoring System upon initial presentation and at the time of resolution.

Results: Out of a total of ninety-seven patients with diabetic ketoacidosis, ninety-two patients fulfilled the inclusion criteria for this study. Among them, there were 56 female cases, accounting for 60.9% of the total, and 58 patients were in the pubertal stage, representing 63% of the sample. The average ages of the patients were 9.67± standard deviation (SD) 3.53, with a range of 1-14 years. A total of twenty-five patients were recently diagnosed with type 1 diabetes, accounting for 27.3% of the cases. The acidosis resolved with a median time of 20.42 hours, with a standard deviation of 8.93 hours (range: 8-48 hours). At presentation, this study found that as capillary blood ketone

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concentrations increased, there was a simultaneous decrease in serum pH, bicarbonate, and carbon dioxide. These changes were statistically significant, with a strong correlation observed between the following parameters: pH (r = −0.493, P = 0.0001), venous bicarbonate (r = - 0.592, P = 0.0001), and carbon dioxide (r = - 0.616, P = 0.0001). At presentation This study found that as capillary blood ketone concentrations increased, there was also an increase in serum Anion gap, urine ketones, and blood glucose. The correlation between these parameters was statistically significant, with the Anion gap having a correlation coefficient of 0.372 (P = 0.0001), urine ketones having a correlation coefficient of 0.671 (P = 0.0001), and blood glucose having a correlation coefficient of 0.207 (P = 0.047).

At Time of Resolution this study found that as capillary blood ketone concentrations decreased, there was an increase in serum pH and bicarbonate levels. The correlation between these parameters was statistically significant: pH (r=-0.305, P= 0.003) bicarbonate (r = -212, P = 0.042). Additionally, during resolution, the study observed a decrease in the Anion gap as capillary blood ketone concentrations decreased, with a statistically significant correlation: Anion gap (r=0.223, P= 0.033). However, there was no significant correlation between capillary blood ketones and both carbon dioxide and urine ketones (r = 0.104, P = 0.325), (r = -0.114, P = 0.279), respectively, at the time of resolution.

Conclusions: Capillary blood ketone measurement shows a stronger correlation with standard parameters of diabetic ketoacidosis compared to urinary ketone measurement. Therefore, capillary blood ketone testing is preferable for early identification and recognition of the resolution of diabetic ketoacidosis episodes.

Introduction

Given the potential for fatal outcomes, it is imperative to define common standards for the treatment of diabetic ketoacidosis (DKA). Currently, there are only a few methods for treating DKA, and the lack of uniformity in approaches has led to inconsistencies in the management of this condition.

Previously, the Pediatric and Adolescent Diabetes Society (PADS) developed a uniform approach through its joint declaration. However, this declaration has not been universally adopted.

In general, clinical signs, such as the ability to eat or drink, and/or nonspecific biomarkers, such as blood pH, are commonly used to assess the resolution of DKA. However, these standards are limited. The criteria used for diagnosing DKA are not specific since the change in respiration degree or the existence of another acid-base disorder can impact the anion gap, HCO3, and pH [3].

The dipstick method for urinalysis measures semiquantitatively the concentration of ketones using nitroprusside base and nitroferricyanide. Its values are indicative for the presence in the urine of large, moderate, small, or trace amounts of ketone bodies. In addition, users may have disparate interpretations of the colour [4]. This test has certain limitations, as it measures acetoacetate but not BHB. BECAUSE BHOB is converted into acetoacetate, which is continuously excreted and thus can be detected even after the resolution of DKA, and causes insulin to be infused beyond the necessary length of time, it would be impossible to collect urine from ill persons suffering from oliguria or anuria that has gone beyond the limits of being ill.

Consequently, serum BHOB should be employed as a more appropriate diagnostic and therapeutic option for DKA. However, the measurement of serum BHOB levels is both time-consuming and cumbersome. In contrast, point-of-care capillary blood BHOB offers a potential advantage in terms

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of time and ease of use. It is now recognised that the estimation of BHOB in capillary blood is a method that has been validated against serum ketones.

Diabetic ketoacidosis (DKA) is a common occurrence in children who have recently developed type 1 diabetes (T1DM), with an estimated occurrence rate ranging from 15% to 70%. Individuals who are younger, do not have a first-degree relative and have a lower socioeconomic position are more susceptible to a greater risk. Diabetic ketoacidosis (DKA) is the primary reason for hospitalization in 16.5-78% of individuals who have recently been diagnosed with type 1 diabetes mellitus (T1DM). In developing countries, mortality rates are increased as a result of factors such as infection, protein-energy malnutrition, and delayed health-seeking behaviour. The majority of occurrences occur in individuals who already have diabetes, with reduced incidence rates compared to those who use insulin pumps.

Material and method

A cross-sectional study was conducted on patients aged between one and 14 years in Iraq between March and July 2022, with symptoms of diabetic ketoacidosis (DKA). The study aimed to assess the correlation between capillary blood ketone body (BHB) and standard parameters of DKA at presentation and resolution. Of the 97 patients enrolled in the study, 92 met the inclusion criteria and were eligible for participation. Five patients were excluded from the study due to complications, including discharge, viral hepatitis, and cerebral oedema. The data was collected from the case files of DKA patients who met the inclusion criteria. The exclusion criteria included patients who were unable to produce a urine specimen, those who did not complete the DKA protocol, and those with conditions that interfered with the accuracy of the eBketone Blood Monitoring System.

The study examined individuals diagnosed with diabetic ketoacidosis (DKA) who had diabetes mellitus. It assessed several factors, including age, gender, tanner stage, type 1 diabetes, and the most recent measurement of HbA1c. The categorisation of DKA severity was determined based on the presence of acidosis, with levels classified as mild, moderate, and severe. The time to resolution of diabetic ketoacidosis (DKA) was defined as the duration from the start of insulin infusion until the acidosis was resolved and the shift to subcutaneous (SC) insulin was made. Laboratory parameters included capillary glucose readings using an Accu-check Active glucometer and testing strips, as well as capillary ketone levels (β-hydroxybutyrate) using the eBketone Blood Monitoring System. The technology employs a minute electrical current to quantify blood ketone levels, providing results within 10 seconds on a digital screen. The eBketone Blood Ketone Monitoring System is calibrated utilising the RANDOX assay kit with the HITACHI 704 Automatic Analyzer.

Results

This study involved 92 DKA patients, categorized by age, pubertal stage, gender, and diagnosis. The mean age was 9.67 years, with 27.2% newly diagnosed and 72.8% known. The mean HbA1C was 11.97%. The severity of DKA varied, with 30 patients having mild, 25 having moderate, and 37 having severe. The median time to resolve acidosis was 20.42 hours.

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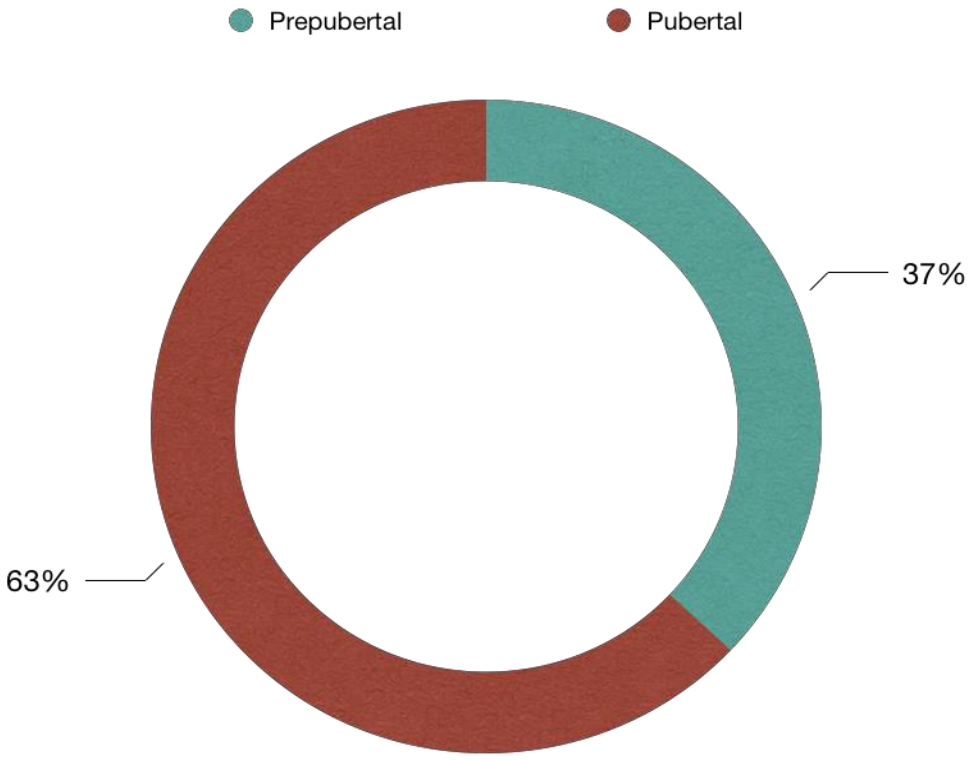

Figure 1. *The percentage of the study sample according to the pubertal stage (n 92).*

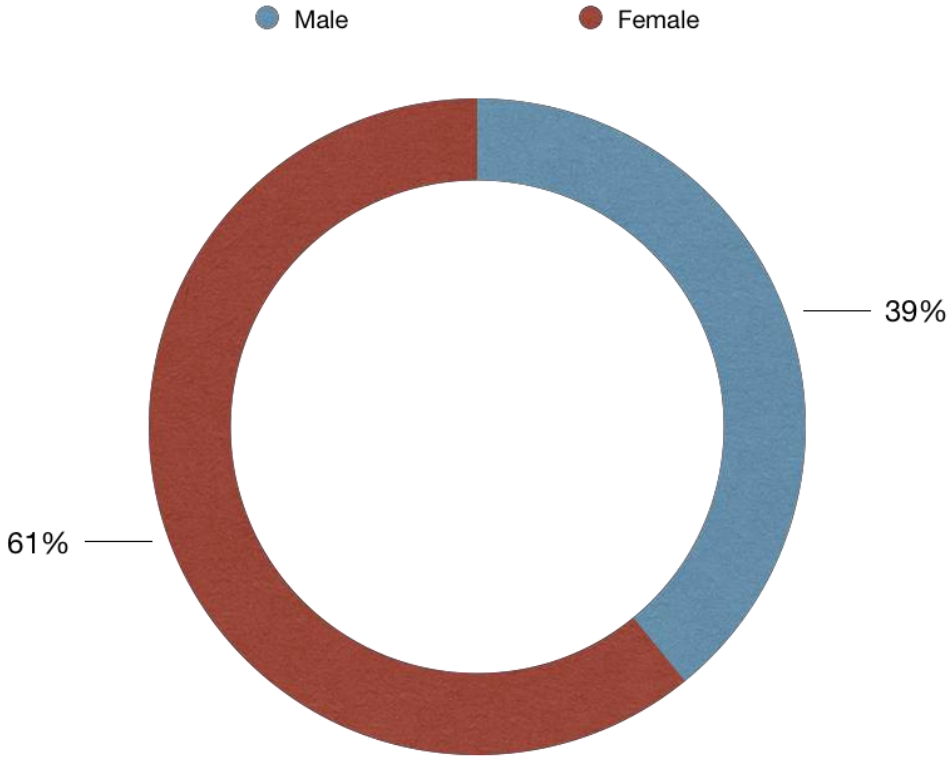

Figure 2. *The percentage of the study sample according to gender (n 92).*

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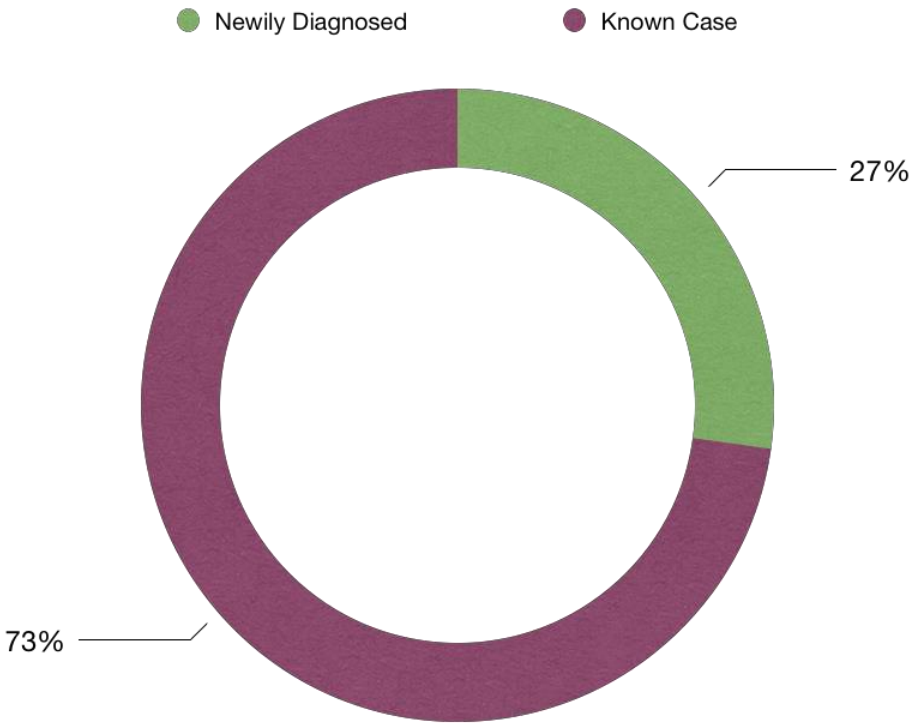

Figure 3. *The percentage of the study sample according to the chronicity of DM (n 92).*

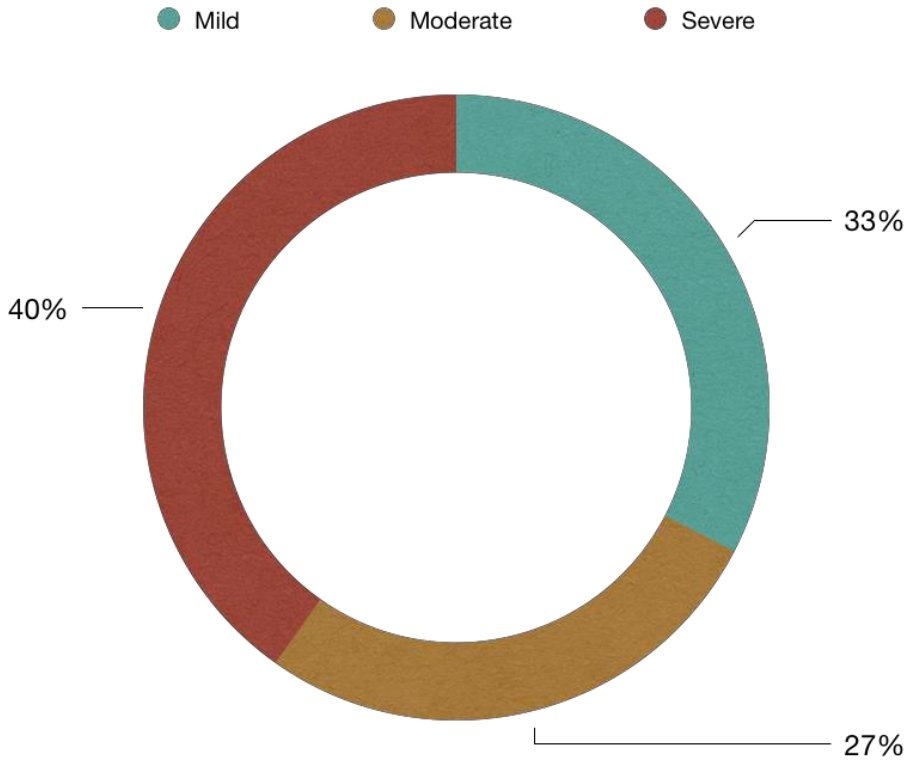

Figure 4. *The percentage of the study sample according to the severity (depending on pH value) (n92).*

Table 1. Descriptive statistics of the metabolic variables at the presentation (n 92).

Variable	Minimum	Maximum	Mean	± SD
HbA1C (%)	7.8	16	11.97	2.22
Duration of Protocol (hours)	8	48	20.42	8.93
Blood Glucose (mg/dl)	280	1085	513.28	164.21
Corrected Na (mmol/L)	129.7	150.8	138.79	3.98
Effective Osmolarity (mosm/kg)	270.9	321.4	293.16	10.64
PH	6.87	7.29	7.11	0.11
CO2 (mmHg)	6.4	43.8	21.21	8.21
HCO3 (mmol/L)	2	19.3	9.55	3.68
Cl (mmol/L)	88.2	118.9	101.86	5.51
Anion gap (mmol/L)	13.1	35.3	20.89	4.45
K (mmol/L)	2.70	6.60	4.57	0.64
Urea (mmol/L)	2.3	18.9	5.22	2.89
Creatinine (umol/L)	17.5	106.7	49.97	20.5
Capillary BHOB (mmol/L)	2.3	8	6.04	1.46

Table 2. Frequency distribution of the metabolic variables at the presentation (n 92).

Variable		*Frequency*	*%*
Blood Glucose (mg/dl)	*≥ 200*	*92*	*100*
	< 200	*0*	*0*
Corrected Na (mmol/L)	*Normal*	*68*	*19.9*
	Hyponatremia	*17*	*18.5*
	Hypernatremia	*7*	*7.6*
Effective Osmolarity (mosm/kg)	*Normal*	*61*	*66.3*
	Low	*1*	*1.1*
	High	*30*	*32.6*
PH	*7.20 – 7.29*	*30*	*32.6*
	7.10 – 7.19	*25*	*27.2*
	< 7.10	*37*	*40.2*
CO2 (mmHg)	*16 – 20*	*64*	*69.6*
	10 – 15	*23*	*25*
	< 10	*5*	*5.4*
HCO3 (mmol/L)	*10 – 15*	*38*	*41.3*
	5 – 9	*48*	*52.2*
	< 5	*6*	*6.5*
Cl (mmol/L)	*Normal*	*79*	*85.9*
	Hypochloremia	*1*	*1.1*
	Hyperchloremia	*12*	*13*
Anion Gap (mmol/L)	*Normal*	*1*	*1.1*
	High	*91*	*98.9*
K (mmol/L)	*Normal*	*77*	*83.7*
	Hypokalemia	*3*	*3.3*
	Hyperkalemia	*12*	*13*
Urea (mmol/L)	*Normal*	*79*	*85.9*
	Elevated	*13*	*14.1*
Creatinine(umol/L)	*Normal*	*86*	*93.5*
	Elevated	*6*	*6.5*

Capillary BHOB (mmol/L)	*1.6 – 2.9*	*1*	*1.1*
	≥ 3	*91*	*98.9*
Urine Ketones	*Nil*	*5*	*5.4*
	+ 2	*18*	*19.6*
	≥ + 3	*69*	*21*

Table 3. Descriptive statistics of the metabolic variables at the resolution (n 92).

Variable	Minimum	Maximum	Mean	± SD
Blood Glucose (mg/dl)	80	226	131.23	37.23
Na (mmol/L)	135	147	139.7	2.84
Effective Osmolarity (mosm/kg)	221.4	299.7	286.3	5.29
PH	7.3	7.44	7.34	0.03
CO2 (mmHg)	20.2	44.7	33	5.2
HCO3 (mmol/L)	15.1	23.7	18.56	2.17
Cl (mmol/L)	105.2	120.9	112.15	3.44
Anion Gap (mmol/L)	6	14	9	2
K (mmol/L)	2.1	5.8	3.7	0.62
Urea (mmol/L)	1.2	8.1	3.58	1.57
Creatinine (umol/L)	12.3	79	40.2	14.6
Capillary BHOB (mmol/L)	0	1.5	0.74	0.39

Table 4. Frequency distribution of the metabolic variables at the resolution (n 92).

Variable		*Frequency*	*%*
Blood Glucose (mg/dl)	*≥ 200*	*7*	*7.6*
	< 200	*85*	*92.4*
Corrected Na (mmol/L)	*Normal*	*90*	*97.8*
	Hypernatremia	*2*	*2.2*
Effective Osmolarity (mosm/kg)	*Normal*	*91*	*98.9*
	High	*1*	*1.1*
PH	*≥ 7.3*	*92*	*100*
CO2 (mmHg)	*≥ 20*	*90*	*97.8*
	< 20	*2*	*2.2*
HCO3 (mmol/L)	*≥ 15*	*92*	*100*
Cl (mmol/L)	*Normal*	*15*	*16.3*
	Hyperchloremia	*77*	*83.7*
Anion Gap (mmol/L)	*Normal*	*92*	*100*
K (mmol/L)	*Normal*	*67*	*72.8*
	Hypokalemia	*24*	*26.1*
	Hyperkalemia	*1*	*1.1*
Urea (mmol/L)	*Normal*	*92*	*100*
Creatinine (umol/L)	*Normal*	*92*	*100*
Capillary BHOB (mmol/L)	*< 1*	*70*	*22.1*
	≥ 1	*22*	*23.9*
Urine Ketones	*Nil*	*22*	*23.9*
	+ 1	*51*	*55.4*
	+ 2	*14*	*15.3*
	≥ + 3	*5*	*5.4*

At the time of presentation, the study found a significant correlation between increasing Capillary BHOB concentrations and decreased serum pH, HCO3, and CO2 levels. Similarly, increasing BHOB concentrations led to a rise in serum AG, urine ketones, and blood glucose levels, with a strong correlation between BHOB and AG and urine ketones.

At the time of Resolution, the study found that decreasing BHOB concentrations led to increased serum pH and HCO3, a significant correlation between BHOB and pH, and & HCO3 decrease in AG. However, no significant correlation was found between capillary BHOB to CO2, urine ketones, or blood glucose.

Table 5. Correlation between capillary BHOB with the metabolic characteristics at the presentation (n 92).

Variable	Variables	Mean	Correlation coefficient -r-	P-value
Capillary BHOB Mean 6.04	**Blood Glucose**	**513.28**	**0.207**	**0.047 (*)**
	PH	**7.11**	**- 0.493**	**0.0001 (**)**
	CO2	**21.21**	**- 0.616**	**0.0001 (**)**
	HCO3	**9.55**	**- 0.592**	**0.0001 (**)**
	Cl	101.86	0.183	0.023 (NS)
	Anion Gap	**20.89**	**0.372**	**0.0001 (**)**
	Urine ketone	**3.64**	**0.671**	**0.0001 (**)**
NS (Not significant)		***(significant)**	****(Highly significant)**	

Table 6. Correlation between capillary BHOB with the metabolic characteristics at the resolution (n 92).

Variable	Variables	Mean	Correlation coefficient -r-	P-value
Capillary BHOB Mean 0.74	Blood Glucose	131.23	0.022	0.836 (NS)
	PH	**7.34**	**- 0.305**	**0.003 (**)**
	CO2	33	-0.104	0.325 (NS)
	HCO3	**18.56**	**- 0.212**	**0.042 (*)**
	Cl	112.15	0.111	0.293 (NS)
	Anion Gap	**9.03**	**0.223**	**0.033 (*)**
	Urine ketone	2.78	0.114	0.279 (NS)
NS (Not significant)		***(significant)**	****(Highly significant)**	

Discussion

According to this study, there was a higher occurrence of DKA episodes in female individuals, accounting for 60.9% of all cases. This finding aligns with the observations made by Pulungan et al. [19], who also documented a higher prevalence of DKA in female patients, with a male-to-female ratio of 1:17.5. According to Wright et al. [20], recurrent DKA has a higher prevalence in females compared to males. Although Shahzad et al. [21] and Rewers et al. [22] did not observe any variation in the incidence of DKA across genders, this discrepancy may be attributed to the unequal sample sizes in this study (92 cases) compared to Shahzad et al. [21] and Rewers et al. [22] (26 and 3666 cases, respectively).

Regarding the pubertal stage, this study found that DKA episodes occurred more frequently in pubertal individuals, accounting for 63% of all cases. This finding is consistent with the studies

conducted by Rewers et al. [22] and Pulungan et al. [19]. This observation is likely attributed to the elevated insulin demand during early puberty, which can be attributed to the rising levels of sex steroid hormones and growth hormones. Both of these hormones have inhibitory effects on insulin [77]. Additionally, this can be explained by the behavioral changes that occur in teenagers, such as the development of brittle diabetes mellitus.

This study revealed that the median duration for resolving DKA was 20.42 hours, with a range of 8 to 48 hours. This finding aligns with the study conducted by Pulungan et al. [19], where the median time for DKA resolution was 21 hours, with a range of 9 to 52 hours. In the study conducted by Shahzad et al. [21], the average time it took for resolution was 17 hours (ranging from 10 to 39 hours). The slight variation in this time could be due to the difference in sample sizes between our study (92 cases) and Shahzad et al.'s study (26 cases). Additionally, the discrepancy may also be influenced by the difference in the percentage of severe cases between the two studies (40% in our study compared to 30% in Shahzad et al.'s study).

The incidence of hyperchloremia increased in this study from 13% at the presentation to 83.7% at the resolution. Tylor et al. observed a substantial increase in the occurrence of hyperchloremia, from 6% to 94%, within 20 hours of initiating treatment. Similarly, Shahzad et al. [21] found that 61.5% of the research group experienced hyperchloremia following DKA treatment.

This study found that there was no statistically significant relationship between capillary BOHB and serum chloride levels, both at the first presentation ($r = 0.183$, $P = 0.023$) and at the resolution ($r = 0.111$, $P = 0.293$). Nevertheless, this association was not examined in additional investigations. The swift progression of hyperchloremia, which can result in hyperchloremic metabolic acidosis, in these trials is mostly caused by the administration of substantial quantities of fluids containing high levels of chloride, as well as intravenous potassium chloride (which is excreted by the kidneys at a higher rate than chloride). The presence of chloride can obscure the detection of the resolution of ketoacidosis when the total base deficit is employed to assess biochemical improvement [78]. Thus, in cases of hyperchloremia, a continuous imbalance in the body's acid-base levels or a low concentration of bicarbonate might be mistakenly attributed to persistent ketosis, potentially impacting the resolution time of diabetic ketoacidosis (DKA). The user's text is "[79]".

To prevent any misinterpretation, this study determined the portion of the base deficit caused by chloride using the following equation:

The base excess caused by chloride can be calculated by subtracting the chloride level from the sodium level and then subtracting 32.

This equation can assist in identifying whether prolonged acidosis is caused by continuous ketosis, which may necessitate further medication such as adjusting insulin infusion or fluids, or by hyperchloraemia, which will naturally resolve itself and does not require specific treatment.

- connection with blood glucose: at presentation, this study found a statistically significant positive connection between capillary BHOB and blood glucose ($r = 0.207$, $P = 0.047$). This suggests that an increase in capillary BHOB was accompanied by an increase in blood glucose levels. Kinsella et al. [80] reported similar findings with a correlation coefficient of 0.29 and a p-value of 0.02. Naunheim et al. [23] also observed a correlation coefficient of 0.31 with a p-value less than 0.001. Similarly, Sheikh-Ali et al. [82] found a p-value less than 0.001.
- connection with pH: at presentation, this study found a significant negative connection between capillary BHOB and blood pH ($r = -0.493$, $P = 0.0001$). As capillary BHOB increased, blood pH decreased. This conclusion aligns with the results published by Ham et al. [30] ($r = -0.62$, $p < 0.0001$). Although the investigations conducted by Pulungan et al. [19] and Shahzad et al. [21] did not find a link between capillary BOHB level and pH, this difference may be due to the disparity in the sample sizes used in the research (92 cases in this study compared to 37 instances in Pulungan et al. [19] and 26 cases in Shahzad et al. [21]).

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At presentation, there was a strong negative correlation (r = -0.592, P = 0.0001) between capillary BHOP and HCO3 levels in this study. This means that as capillary BHOP increased, HCO3 levels decreased. This finding is consistent with previous studies by Sheikh-Ali et al. [82] and Shahzad et al. [21], who also found negative correlations between capillary BHOP and HCO3 levels (r = -0.68, p<0.001 and r = -0.37, P = 0.01, respectively). The findings of Pulungan et al. [19] contradicted the results presented here since they claimed that there was no link between capillary BOHB levels and HCO3 levels. This discrepancy may be attributed to the limited sample size of Pulungan et al.'s study (only 37 cases).

Correlation with carbon dioxide (CO2) at presentation: positive; the study found a significant and severe negative connection between capillary BHOB and CO2 levels (r = −0.616, P < 0.0001). This observation aligns with the findings reported by Naunheim et al. [23], where a negative correlation (r = −0.69, p < 0.001) was seen.

Correlation with Anion gap at presentation This study found that there was a significant positive correlation (r = 0.372, P = 0.0001) between the increase in capillary BHOB and the increase in AG. This finding is consistent with the results of previous studies by Shahzad et al. [21] (r = 0.524, P = 0.006) and Naunheim et al. (r = 0.66, p<0.001). However, the study by Pulungan et al. [19] did not show a significant correlation between BHOB and AG. The difference in findings may be due to the difference in the average AG values between this study (20.89 ± SD 4.45 mmol/L) and the study by Pulungan et al. [19] (28.7 ± SD 7.1 mmol/L).

There is a correlation between urine ketones and another factor at presentation. The study found a strong positive connection (r = 0.671, P = 0.0001) between capillary BHOB and urine ketones. This means that when BHOB levels increased, urine ketone levels also increased. This finding is consistent with Pulungan et al. [19] (r= 0.51, p= 0.001) but contradicts the results of Turan et al., who did not find any link between them during the presentation. The disagreement may be due to the fact that DKA patients in the study by Turan et al. arrived at the emergency room earlier, resulting in a higher percentage of patients without ketones in their urine compared to this study (14% vs 5%, respectively). This difference could potentially impact the correlation between capillary BHOB and urine ketones at the beginning of treatment.

The study discovered no substantial association between capillary BHOB and blood glucose at resolution or urine ketone during resolution. This finding aligns with prior research, including the slight association shown by Kinsella et al. At the time of resolution, A strong negative correlation was observed between capillary BHOB and blood pH, indicating an inverse relationship. The study also discovered a statistically significant negative relationship between capillary BHOB and HCO3 at the time of resolution, indicating that as capillary BHOB decreases, HCO3 increases. Additionally, there was a positive association between capillary BHOB and AG at resolution, further supporting the strong link. The study also discovered that there was no substantial link between capillary BOHB and urine ketones during resolution. This lack of correlation may be attributed to the varying criteria for resolving DKA in the two trials. The study's results could be attributed to the imbalanced sample size and varying criteria for resolving DKA.

Conclusion

Capillary BOHB measurement correlates more accurately with other standard parameters of diabetic ketoacidosis than urinary ketone measurement in reflecting the patient's metabolic state and early diagnosis and recognition of ketoacidosis resolution. Increasing BHOB concentrations leads to a drop in serum pH, HCO3, and CO2 and a rise in AG, blood glucose, and urine ketones at presentation.

while during resolution dropping BHOB CONCENTRATION were accompanied by a rise in serum PH and HCO3 and accompanied with a decrease in AG

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