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Correlation between Serum Albumin Level and Diabetic Ketoacidosis among Children with Type I Diabetes

Dina ElKhedr Ali Hassan Abouelhoda *; Mohamed Ibrahim Abd El-Aal; Anas Mohamed Elshreif

Department of Pediatrics, Damietta Faculty of Medicine, Al-Azhar University, Damietta, Egypt.

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*Corresponding author

Email: drdinakhedr89@gmail.com

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Abstract

Background: Diabetic ketoacidosis [DKA] is a state of catabolism, where protein breakdown prompts decline in serum albumin levels. As a result, hypoalbuminemia could serve as a valuable indicator of the risk of ketosis, which in turn implies an underlying deficiency in insulin.

Objective: Evaluation of serum albumin level and its correlation with DKA severity among Type 1 diabetes mellitus children [T1DM].

Patients and Methods: This cross-sectional study included 100 children with DKA admitted to pediatric intensive care unit [PICU], Al-Azhar University Hospital, New Damietta from July 2023 to May 2024. Patients were divided into three groups according to severity of DKA. Demographic, anthropometric data, medical history, arterial blood gases [ABG], random blood glucose [RBG], complete blood count [CBC], glycated hemoglobin [HbA1c], albumin, prothrombin time [PT], international normalized ratio [INR], serum electrolytes [Na and K] and urine analysis data were collected.

Results: The median age of cases was 10 [7-10.5] years and there was a predominance of male gender [60%]. The majority of cases [58%] had severe DKA, [22%] had moderate DKA and [20%] had mild DKA. Albumin showed statistically lower levels among severe DKA patients 3.34 ± 0.52 gm/dl compared to mild DKA patients 3.65 ± 0.57 gm/dl with a sensitivity of 67.2% and a specificity of 85.7% to detect severe DKA cases at a cutoff of 3.05 gm/dl. There was also a significant negative correlation between albumin levels and frequency of DKA in the past 6 months [r=-0.435, p-value=0.001].

Conclusion: Albumin levels were significantly associated with severity of DKA demonstrating significant difference between mild and severe cases of DKA. Additionally, cases with lower levels of albumin had significantly higher frequency of DKA during the last 6 months prior to our study.

Keywords: Albumin; Diabetic Ketoacidosis; Type 1 Diabetes Mellitus.

INTRODUCTION

Type 1 diabetes [T1DM], also known as insulin-dependent diabetes, is an autoimmune disorder that eventually results in the destruction of insulin-producing beta cells in the Langerhans islets of the pancreas. This β cells destruction causes control of blood glucose levels leading to hyperglycemia, ketoacidosis and on the long-term incidence of complications like blindness, renal failure, and cardiovascular disease [1].

Diabetic ketoacidosis [DKA] is the most frequent acute hyperglycemic emergency in children with diabetes mellitus [DM]. It occurs due to either an absolute or relative insulin deficiency, along with an increase in counter-regulatory hormones. This typically leads to the combination of hyperglycemia, metabolic acidosis, and ketosis, often accompanied by varying levels of circulatory volume depletion [2].

Ketoacidosis is a metabolic condition characterized by abnormally high levels of ketone bodies in the blood and urine. These ketone bodies [KBs] can be used as an energy source by the brain, heart, and skeletal muscles when the body is either deficient in or unable to properly utilize glucose [3].

Albumin, which is synthesized in the liver, is minimally stored there and is quickly released into the bloodstream. It is present in the bloodstream, interstitial space, and other bodily fluids. In children, the normal albumin concentration ranges from 2.9 to 5.5 g/dl [4].

Hypoalbuminemia, a condition marked by low levels of albumin in the blood, is a common sign in sick patients. This condition results not only from reduced albumin synthesis but also from protein breakdown, impaired protein uptake, and leakage into the extravascular space [5].

In diabetes, the chronic high blood sugar levels lead to the glycation of proteins, which increases the accumulation of advanced glycation end products [AGEs]. Among plasma proteins, albumin is particularly prone to glycation and is a major contributor to the presence of AGEs in the plasma. This increases the level of glycated albumin from 8-10% in healthy individuals to 20–30% in diabetic patients [6].

PATIENTS AND METHODS

a cross-sectional case control study of patients with This cross-sectional study included 100 patients with DKA recruited from the Pediatric Intensive Care Unit [PICU], Al-Azhar University Hospital in New Damietta from July 2023 to May 2024. Patients were divided into three groups according to venous PH and HCO_3 [7].

Group I [n=20]: Mild DKA where the venous pH of <7.3 and/or a HCO_3 level of <15 meq/L. **Group II [n=22]:** Moderate DKA where the venous pH of <7.2 and/or a HCO_3 level of <10 meq/L. **Group III [n=58]:** Severe DKA where the venous pH is <7.1 with or without a HCO_3 level <5 meq/L.

Inclusion criteria: Both males and females, the age was between 2 and 18 years, patients presented with a picture of DKA and confirmed with laboratory data [including first presentation and already known T1DM] according to Commonly accepted criteria for diabetic ketoacidosis, which are blood glucose greater than 300 mg/dl, arterial pH less than 7.3, serum bicarbonate less than 15 mEq/l, and the presence of ketonemia or ketonuria [8].

Exclusion criteria: Any disease that can affect serum albumin including: Metabolic syndromes, malignancy, nephritic or nephrotic syndromes, chronic kidney disease, chronic liver disease and malnutrition

Ethical consideration: This study was conducted after approval of Faculty of Medicine, Al-Azhar University Research Ethics Committee.

Patient were evaluated regarding age, sex, medical history that included family history, Onset of disease, acute complications including attacks of DKA in the last 6 months, hypoglycemia episodes in the last month. Laboratory investigations included CBC and HbA1c assay using *Sysmex-Yumizen [Horiba]*, ABG analysis using *Gastat-720*, Na and K analysis using *Cobas 311*, PT, INR using *Sysmex* and albumin analysis using *ST200*.

Statistical analysis: The data were analyzed using the IBM Statistical Package for the Social Sciences [SPSS], Version 22.0 [IBM Corp., Armonk, NY]. The significance of the results was assessed at the 0.05 level. The Shapiro-Wilk test was applied to assess the normality of the quantitative data. For non-parametric data, the median and interquartile range [IQR] were used for description, and the Kruskal-Wallis test was employed to compare the three groups. For parametric data, the mean and standard deviation [SD] were reported, and a One-Way ANOVA test was used for group comparisons. Qualitative data were presented as numbers and percentages, with the Chi-Square test used for comparisons between two or more groups. The Monte Carlo test was applied to correct the Chi-Square test when more than 25% of cells had counts less than 5 in tables larger than 2x2. The diagnostic performance of a test was assessed using Receiver Operating Characteristic [ROC] curve analysis, and binary stepwise logistic regression was used to predict independent variables related to a binary outcome.

RESULTS

Table [1] illustrates that patients were significantly younger among moderate and severe DKA groups than that patients with mild DKA group, while there was no significant difference among the groups regarding BMI percentile and sex. It also demonstrates that DKA recurrence and hypoglycemic episodes were significantly higher among severe DKA group compared to the mild and moderate DKA groups. Table [2] shows that RBG were comparable among all groups with no statistically significant difference. Both the anion gap and serum Na^+ were significantly higher among severe DKA group compared to mild DKA groups. As regards K^+, the levels were comparable among all groups with no

statistically significant difference. Table [3] illustrates that PT was significantly longer among severe DKA group compared to mild and moderate DKA group. It also demonstrates that albumin was significantly lower among severe DKA group compared to mild DKA group. Table [4] and figure [1] show the sensitivity and specificity of albumin were 67.2% and 85.7% at a cutoff point of 3.05 gm/dl was associated with severe cases of DKA.

Table [5] shows that considering univariate regression, age, history of DKA in the last 6 months, hypoglycemic episodes in the last month, anion gap, serum Na^+ and albumin were independent predictors of DKA severity. While upon considering multivariate regression analysis, history of DKA in the last 6 months, hypoglycemic episodes, anion gap and albumin were independent predictors of DKA severity.

Table [1]: Medical history of the studied patients

Medical history		Mild DKA [n=20]	Moderate DKA [n=22]	Severe DKA [n=58]	Test of sig.	P-value
Age [years]	Median[IQR]	11 [9.63-13]	9.25 [8-10]	9 [7 -10.25]	KW=11.6 P=0.003	P_1=0.002 P_2=0.789 P_3=0.002
	Min-Max	6-13.5	5-11	3-13		
BMI percentile	Median[IQR]	50 [40-50]	37.5 [25-50]	40 [30-50]	KW=3.89	0.14
	Min-Max	25-70	25-50	25-70		
Sex N [%]	Male	13 [65%]	9 [40.9%]	38 [65.5%]	χ2=4.29	0.117
	Female	7 [35%]	13 [59.1%]	20 [34.5%]		
Family history of DM		9 [45%]	8 [36.4%]	31 [53.4%]	χ2=1.96	0.38
Known diabetic [n=65]		16 [80%]	10 [45.5%]	39 [67.2%]	χ2=5.8	0.06
Newly diagnosed [n=35]		4 [20%]	12 [54.5%]	19 [32.8%]		
History of DKA in last 6 months [n=65]	None	8 [50%]	4 [40%]	6 [15.4%]	MC= 10.59	0.032
	1-3	6 [37.5%]	4 [40%]	14 [35.9%]		
	>3	2 [12.5%]	2 [20%]	19 [48.7%]		
Hypoglycemic episodes during last month [n=65]	0	10 [62.5%]	2 [20%]	5 [12.8%]	MC= 25.98	0.001
	1	3 [18.8%]	4 [40%]	8 [20.5%]		
	2	2 [12.5%]	3 [30%]	16 [41%]		
	3	1 [6.2%]	1 [10%]	8 [20.5%]		
	4	**0 [0%]**	**0 [0%]**	**2 [5.2%]**		

BMI: Body mass index, DKA: Diabetic ketoacidosis, IQR: Interquartile range, KW: Kruskal Wallis test DKA: Diabetic ketoacidosis, DM: Diabetes mellitus, MC: Monte Carlo, χ2: Chi square test, p: p-value >0.05: Non-significant; p-value <0.05: Significant; P1: The difference between mild and moderate DKA groups, P2: The difference between moderate and severe DKA groups, P3: The difference between mild and severe DKA groups.

Table [2]: Laboratory data of the studied patients

Laboratory investigations		Mild DKA [n=20]	Moderate DKA [n=22]	Severe DKA [n=58]	Test of sig.	P-value
RBG [mg/dl]	Mean±SD	516.67 ± 15.81	493.14 ± 62.54	520.47 ± 82.07	F=0.42	**0.66**
	Min-Max	400-530	443-560	400-600		
HbA1c [%]	Median[IQR]	10.5 [10.5-12]	10.7 [8.7-14.5]	11.5 [10.1-12]	KW=918	**0.45**
	Min-Max	9.5-12.4	8.5-14.5	9-16		
PH	Mean±SD	7.23 ± 0.028	7.14 ± 0.03	6.97 ± 0.098	F=94.9 P=0.001	**P_1=0.799; P_2=0.001 P_3=0.002**
	Min-Max	7.2-7.29	7.1-7.19	6.81-7.09		
PCO_2 [mmHg]	Mean±SD	28.05 ± 6.1	29.57 ± 5.09	22.18 ± 7.55	F=12.48 P=0.001	**P_1=0.799; P_2=0.001; P_3=0.002**
	Min-Max	22-39	21.33	15-39		
HCO_3 [mEq/L]	Mean±SD	11.1 ± 0.9	8.34 ±1.18	4.03 ± 1.07	F=418.3 P=0.001	**P_1=0.001; P_2=0.001; P_3=0.001**
	Min-Max	10-12	7-10	3-6		
Anion gap [mEq/L]	Median[IQR]	20 [18.25-22]	21 [18-23]	22 [19-23]	KW=7.1 P=0.001	**P_1=0.51; P_2=0.089; P_3=0.015**
	Min-Max	18-22	18-23	18-25		
Serum Na^+ [mmol/L]	Mean±SD	137.75 ± 3.96	139.32 ± 2.3	140.67 ± 3.3	F=6.25 P=0.001	**P_1=0.27; P_2=0.23; P_3=0.002**
	Min-Max	131-143	135-144	132-146		
Serum K^+ [mEq/L]	Mean±SD	3.9 ± 0.26	3.89 ± 0.53	3.95 ± 0.44	F=0.14	**0.87**
	Min-Max	3.3-4.3	3.3-4.6	3.5-5		

F: One-Way ANOVA, DKA: Diabetic ketoacidosis, IQR: Interquartile range, HbA1c: Glycated hemoglobin, K: Potassium, Na: Sodium, KW: Kruskal Wallis test, p: p-value >0.05: Non-significant; p-value <0.05: Significant; P1: The difference between mild and moderate DKA groups, P2: The difference between moderate and severe DKA groups, P3: The difference between mild and severe DKA groups.

Table [3]: Hematological and coagulation profiles among the studied patients

Laboratory investigations		Mild DKA [n=20]	Moderate DKA [n=22]	Severe DKA [n=58]	Test	P
Hb [gm/dl]	Median[IQR]	14[13.7-14]	14 [11-14]	13.3 [12-14.5]	KW=3.29	**0.19**
	Min-Max	12.5-14.2	11.2-14.9	10.2-14.9		
RBCs [x10^6/mcL]	Mean±SD	5.01 ± 0.45	5.31 ± 0.32	5.14 ± 0.38	F=2.21	**0.12**
	Min-Max	4.37-5.65	3.99-5.73	4.15-5.58		
Platelet count [x10^3/mm^3]	Mean±SD	282.8±61.8	260.7 ± 76.8	259.8 ± 73.9	F=2.87	**0.07**
	Min-Max	160-414	83-350	150-429		
WBC [x10^3/mm^3]	Mean±SD	16.2 ± 5.2	14.5 ± 4.7	15.9 ± 2.8	F=1.29	**0.28**
	Min-Max	9-25	7.5-22	8.9-22		
Neutrophils [x10^3/mm^3]	Mean±SD	9.28 ± 2.7	9.28 ± 2.39	9.1 ± 2.3	F=0.26	**0.77**
	Min-Max	0.51-13.2	0.57-13.2	0.31-13.8		
Lymphocytes [x10^3/mm^3]	Mean±SD	6.3 ± 3.4	5.3 ± 2.1	6.6 ± 1.8	F=2.95	**0.05**
	Min-Max	2.7-12.9	2.5-8.8	4.2-9.9		
PT [Seconds]	Mean±SD	11.78±0.86	11.54 ± 0.51	12.4 ± 1.31	F=5.08 P=0.008	P_1=0.57; P_2=0.008 P_3=0.026
	Min-Max	11-19	11-12	11-14		
INR	Median[IQR]	1 [1-1]	1 [1-1]	1 [1-1]	KW=2.12	0.35
	Min-Max	0.92-1.2	0.95-1.1	0.97-1.3		
Albumin [gm/dl]	Mean±SD	3.65 ± 0.57	3.58 ± 0.48	3.34 ± 0.52	F=4.45 P=0.014	P_1=0.94; P_2=0.076 P_3=0.035
	Min-Max	3-4.9	3-4.7	2.8-4.6		

CBC: Complete blood count, DKA: Diabetic ketoacidosis, F: One-Way ANOVA, Hb: Hemoglobin, INR: International normalized ratio, IQR: Interquartile range, KW: Kruskal Wallis test, PT: Prothrombin time, WBCs: White blood cell, RBCs: Red blood cells, SD: Standard deviation, T: Independent Student T test, U: Mann Whitney test, p: p-value >0.05: Non-significant; p-value <0.05: Significant; P1: The difference between mild and moderate DKA groups, P2: The difference between moderate and severe DKA groups, P3: The difference between mild and severe DKA groups.

Table [4]: Diagnostic value of serum albumin for detecting the severity of DKA

Lab parameter	AUC	P	Sensitivity [%]	Specificity [%]	95% CI	Cutoff
Albumin [gm/dl]	0.345	0.009	67.2	85.7	0.239-0.452	3.05

AUC: Area Under curve, CI: Confidence interval, value, p: p-value >0.05: non-significant; p-value <0.05: significant.

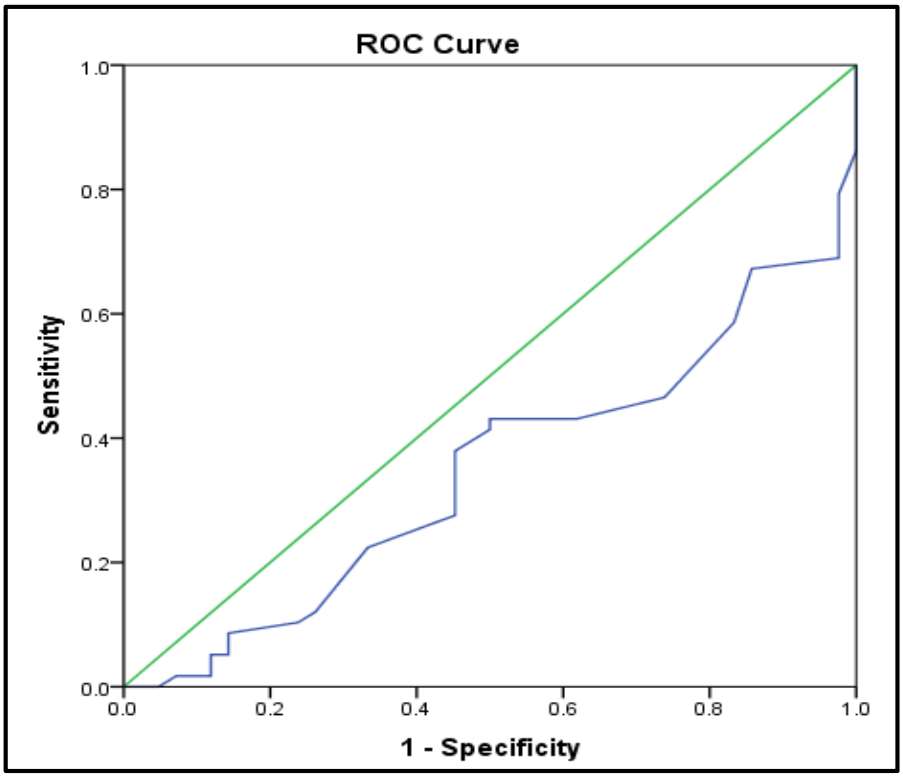

Figure [1]: ROC curve of albumin for prediction of DKA severity

Table [5]: Some risk factors of severity of DKA

Risk factors	Univariate regression			Multivariate regression		
	B	P	OR [95%CI]	β	P	OR [95%CI]
Age [years]	-0.21	0.017	0.82 [0.69-0.96]	-0.15	0.34	0.86 [0.64-1.17]
Anion gap [mEq/L]	0.26	0.009	1.29 [1.07-1.57]	0.41	0.001	1.5 [1.19-1.89]
Hx of DKA in the last 6 months	0.76	0.006	2.14 [1.24-3.69]	1.21	0.01	3.35 [1.32-8.51]
Hypoglycemia in the last month	0.85	0.003	2.34 [1.34-4.08]	0.69	0.04	1.99 [1.04-3.84]
Serum Na^+ [mmol/L]	0.19	0.004	1.22 [1.07-1.39]	0.11	0.45	1.11 [0.84-1.48]
Albumin [gm/dL]	-1.09	0.009	0.34 [0.15-0.76]	-0.2	0.03	0.81 [0.64-0.92]

DKA: Diabetic ketoacidosis, DM: Diabetes mellitus, Hb: Hemoglobin, HbA1c: Glycated hemoglobin, INR: International normalized ratio, RBG: Radom blood glucose, K: Potassium, Na: Sodium, WBCs: White blood cells, PT: Prothrombin time, β: Standardized regression coefficient, OR: Odds ratio, CI: Confidence interval, p: p-value >0.05: Non-significant [NS]; p-value <0.05: Significant[S]; p-value< 0.01: highly significant [HS].

DISCUSSION

This study showed a significant difference regarding age among cases with moderate and severe DKA groups compared to the mild DKA group as cases of moderate and severe DKA had significantly younger age. This comes in agreement with **Ahmed *et al.*** [9] whom performed a retrospective study that included 982 in Sudanese children with T1DM and found that the highest occurrence of severe DKA was observed in preschool-aged children, highlighting the need for increased focus on this age group with T1DM to prevent acute complications and the severe consequences of the disease. In explanation, **Lee *et al.*** [10] have reported that the increased risk of DKA in younger children may be due to more pronounced β-cell dysfunction, indicating that aggressive diabetes and delayed recognition of diabetes symptoms are more common in this age group.

Our study demonstrated that patients with severe DKA had significantly higher frequency of DKA in the last 6 months and hypoglycemic episodes during the last month implying poor glycemic control. This was in compliance with **Pettus *et al.*** [11] whom reported that older patients had lower HbA1c levels compared to younger patients. Those with poor glycemic control experienced the highest annual incidence of hypoglycemia and DKA. This finding challenges the common belief that hypoglycemic events generally result from strict glycemic control; in fact, poor glycemic control may lead to greater fluctuations in glucose levels, resulting in more frequent hypoglycemic episodes. The fear of hypoglycemia may also discourage some patients from aiming for optimal HbA1c control, which can lead to worsened glycemic control and, consequently, an increase in DKA incidence.

The present study showed that both anion gap and sodium were significantly elevated among severe DKA patients compared to mild DKA patients.

Although **Albuali *et al.*** [12] reported a significant association between severe DKA and the anion gap compared to moderate DKA, high blood glucose level, low serum K and high Na levels were associated with severe DKA.

Indeed, serum sodium levels are typically low due to the movement of water from the intracellular to the extracellular space, driven by the osmotic effect of hyperglycemia. Therefore, normal or elevated serum sodium levels suggest severe volume depletion. Serum potassium levels may be elevated as a result of potassium shifting from the intracellular compartment in exchange for acids due to the lack of insulin. Normal or low potassium levels indicate overall depletion of the body's potassium stores, requiring correction before starting insulin therapy [13].

This study showed that the prothrombin time [PT] was significantly longer in the severe DKA group compared to the mild and moderate DKA groups. Additionally, albumin levels were significantly lower in the severe DKA group compared to the mild DKA group. When performing a ROC curve analysis for albumin, a cutoff of 3.05 g/dl demonstrated a sensitivity of 67.2% and a specificity of 85.7% for detecting severe cases of DKA.

These findings align with **Cheng *et al.*** [14], who reported that among 255 diabetic individuals in Taiwan, those with low serum albumin levels were more prone to ketosis, as indicated by a higher prevalence of ketonuria and a trend towards increased ketonemia. This suggests an underlying insulin deficiency. They proposed that serum albumin levels could indirectly reflect a diabetic individual's insulin reserve and, therefore, serve as an indirect marker of glycemic control.

This may be due to the fact that albumin acts as a coregulator of ketogenesis, alongside insulin, glucagon, and other hormones. Albumin might inhibit the mitochondrial enzyme hydroxy-methylglutaryl-CoA synthase, which is the rate-limiting enzyme in ketogenesis, thereby reducing ketone production in insulin-deficient states. In other words, hypoalbuminemia in insulin-deficient conditions removes the inhibition of this enzyme, promoting uncontrolled ketogenesis. When the synthesis of ketone bodies exceeds their breakdown, a diabetic patient develops DKA. A significant proportion of normo-albuminemic patients showed no evidence of ketonuria, clearly indicating that normal albumin levels inhibit ketogenesis [15].

Furthermore, insulin influences the synthesis of liver proteins, including albumin and fibrinogen. It enhances albumin gene transcription and mRNA production in a dose-dependent manner. Conversely, insulin deficiency leads to a reduction in albumin gene transcription and mRNA levels, resulting in decreased albumin production. In patients with T1DM, the absence of insulin leads to a decline in albumin synthesis, which is reversible upon insulin administration [16].

Another hypothesis suggests that hyperglycemia worsens beta cell dysfunction and depletes the insulin secretory reserve. While direct measurement of beta cell function is not readily available, and the proinsulin-to-insulin ratio is often used in research, albumin may serve as a marker of insulin reserve in these patients. Low albumin levels could be associated with insufficient insulin, potentially triggering DKA [17, 18]. However, the measurement of proinsulin is not commonly accessible in hospital labs, and the proinsulin-to-insulin ratio may be influenced by exogenous insulin used to manage hyperglycemia. Serum albumin, which is widely available in hospital laboratories and reflects insulin secretory reserve, may help clinicians determine the appropriate duration of insulin therapy. Furthermore, serum albumin concentration may assist in classifying hyperglycemic individuals based on their risk of ketosis and prognosis. Along with

HbA1c, serum albumin could serve as an additional measure of glycemic control [14].

On the other side, the production of most coagulation factors occurs in the liver as well as albumin. A reduction in coagulation factor production can result in prolonged prothrombin time, which may influence albumin levels [19].

There are **some limitations** that have been encountered in this study. The first limitation of this study is the small sample size and the lack of control group. Second, this study was conducted in a single setting, and so these results cannot be generalized to the whole population. Third, the cross-sectional design of the study does not allow establishing causal relationships. Finally, follow-up of patients and multiple measurements of serum albumin along with frequency of acute complications would provide more comprehensive idea regarding these correlations.

Conclusion: There was a significant negative correlation between serum albumin and frequency of DKA in the last 6 months. Additionally, hypoalbuminemia and young age seem to be risk factors for severity of DKA. DKA can affect the coagulation profile as evidenced by the profile the presence of significant correlation between PT and severity of DKA.

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