Journal of Molecular and Pharmaceutical Sciences

Original Research Article

Hba1c and fructosamine as dual biomarkers of glycemic control on pediatric type 1 diabetes during Ramadan fasting

Touaitia Rahima [a*], Boussahia Riadh [b]

[a] *Department of Natural and Life Sciences, Faculty of Exact Sciences and Natural and Life Sciences, Echahid Cheikh Larbi Tebessi University, Tebessa 12000, Algeria*

[b] *EL NOUR Medical Laboratory, Cheria, Tebessa 12000, Algeria*

Abstract

Glycated hemoglobin (HbA1c) is the standard biomarker for long-term glycemic control in type 1 diabetes (T1D), but it fails to capture short-term fluctuations. Fructosamine, which reflects glycemia over 2–3 weeks, may provide complementary information during periods of rapid metabolic change, such as Ramadan fasting. To evaluate short- and medium-term glycemic control in pediatric patients with T1D before and after Ramadan by comparing HbA1c and fructosamine.

Methods: This observational study enrolled 18 children (<18 years) with T1D at EL NOUR Medical Laboratory, Tebessa. Blood samples were collected one week before and one week after Ramadan. Fasting plasma glucose (FPG), HbA1c, and fructosamine were measured using validated enzymatic and colorimetric assays. Paired comparisons were performed, and correlation analyses were reported with the correlation coefficient, sample size, p-value, and 95% confidence interval. Mean HbA1c increased significantly from 8.69 ± 1.51% before Ramadan to 9.34 ± 1.46% after Ramadan (p = 0.03), indicating a deterioration in glycemic control over the study period. Mean fructosamine increased from 406.02 ± 91.03 µmol/L to 426.21 ± 82.14 µmol/L; however, this change was not statistically significant (p = 0.09). Five participants showed lower post-Ramadan fructosamine concentrations, reflecting heterogeneous short-term glycemic responses that should be interpreted cautiously. Pearson's correlation analysis demonstrated a moderate, positive, and statistically significant association between HbA1c and fructosamine levels (r = 0.61, 95% CI: 0.20–0.84, p = 0.007, n = 18). In this small observational cohort, Ramadan fasting was associated with a significant increase in HbA1c among children with T1D, suggesting deterioration in overall glycemic control. Fructosamine showed heterogeneous short-term responses without reaching statistical significance, supporting its potential role as a complementary biomarker rather than a replacement for HbA1c. Larger prospective studies including mid-Ramadan measurements, CGM metrics, insulin regimen data, pubertal status, and fasting adherence are needed.

Keywords:

Type 1 diabetes, children, HbA1c, fructosamine, Ramadan, glycemic control

★Received May 10, 2026; accepted June 10, 2026

*Corresponding author

Email address: rahima.touaitia@univ-tebessa.dz (Touaitia Rahima)

Cited as: Touaitia R, Boussahia R. Hba1c and fructosamine as dual biomarkers of glycemic control on pediatric type 1 diabetes during Ramadan fasting. J. Mol. Pharm. Sci. 05 (01), 2026, 53-62.

1. Introduction

Type 1 diabetes (T1D) is a chronic autoimmune disorder characterized by the progressive destruction of pancreatic β-cells, leading to absolute insulin deficiency and necessitating lifelong insulin replacement therapy [1], [2], [3], [4]. While T1D predominantly manifests in children and adolescents, it can develop at any age, with incidence rates increasing globally, particularly among younger age groups [5], [6]. The pathophysiology involves an interplay of genetic susceptibility and environmental triggers, which initiate an autoimmune response targeting insulin-producing β-cells, ultimately resulting in insulin deficiency [7], [8], [9], [10], [11].

Managing T1D poses significant challenges due to the necessity of maintaining precise glycemic control amid varying metabolic demands during growth and development. Effective glycemic management is critical to prevent acute complications such as diabetic ketoacidosis and severe hypoglycemia, both of which can have immediate life-threatening consequences [12], [13], [14]. Furthermore, chronic hyperglycemia contributes to microvascular complications including retinopathy, nephropathy, and neuropathy as well as macrovascular diseases, which remain the leading causes of morbidity and premature mortality in this population [15], [16], [17], [18].

Advances in insulin formulations, delivery systems such as insulin pumps, and continuous glucose monitoring (CGM) technologies have significantly improved the ability to achieve near-normal glycemic targets and reduce the burden of hypoglycemia [19], [20], [21], [22], [23]. Despite these technological improvements, maintaining stable glycemic control remains difficult, particularly during periods of physiological stress, lifestyle changes, or cultural practices such as fasting, which can disrupt metabolic homeostasis and insulin requirements [24], [25], [26], [27], [28], [29]. Understanding these challenges and optimizing monitoring strategies are essential to improve health outcomes in children and adolescents living with T1D.

Glycated hemoglobin (HbA1c) remains the clinical gold standard for monitoring long-term glycemic control, as it reflects average blood glucose concentrations over the preceding two to three months by measuring the non-enzymatic glycation of hemoglobin molecules [30]. Its widespread use is supported by strong evidence linking HbA1c levels to the risk of both microvascular and macrovascular diabetes complications [31], [32], [33], [34], [35]. However, HbA1c has important limitations. It does not capture short-term glycemic variability or acute glucose fluctuations, which can significantly impact patient outcomes, including hypoglycemic episodes and glucose toxicity [36], [37]. This limitation is particularly relevant during periods of rapid metabolic changes, such as illness, therapy adjustments, or lifestyle modifications including fasting [38], [39]. Additionally, HbA1c measurements can be affected by conditions altering red blood cell turnover or hemoglobin variants, potentially leading to inaccurate readings [40], [41]. Consequently, there is growing interest in identifying complementary biomarkers that more accurately reflect shorter-term glycemic control, enabling timely clinical interventions. Biomarkers such as fructosamine and 1,5-anhydroglucitol have been explored for this purpose, as they provide insights into average glycemia over shorter periods ranging from 1 to 3 weeks [42], [43], [44]. Incorporating these markers alongside HbA1c may optimize diabetes management by capturing both chronic and acute glycemic trends.

One clinically significant context in which glycemic monitoring becomes particularly challenging is Ramadan, the ninth month of the Islamic lunar calendar, during which fasting from dawn to sunset is observed by millions of Muslims worldwide [45], [46]. Although children with T1D are religiously exempt from fasting, many elect to participate due to cultural, familial, or personal convictions [45], [47]. Ramadan fasting involves prolonged periods of abstinence from food and drink, followed by altered meal timing and composition during pre-dawn (suhoor) and post-sunset (iftar) meals. These modifications disrupt normal nutritional intake, physical activity patterns, and insulin administration schedules, thereby imposing unique metabolic stresses on glucose homeostasis [48], [49]. Such changes substantially increase the risk of both hypoglycemic and hyperglycemic episodes, as well as diabetic ketoacidosis, particularly in pediatric populations who may have less physiological resilience and variable adherence to treatment [50], [51]. Consequently, Ramadan fasting demands intensified glucose monitoring, individualized insulin regimen adjustments, and comprehensive patient education to mitigate risks and ensure safe observance. Recent guidelines underscore the importance of integrating advanced monitoring tools, including continuous CGM and complementary biochemical markers, to optimize metabolic control during Ramadan [49], [52], [53].

In this clinical setting, fructosamine has gained attention as a complementary biomarker to HbA1c for assessing short-term glycemic control. Fructosamine reflects average blood glucose levels over the preceding 2–3 weeks by measuring glycated serum proteins, primarily albumin, thus providing a more immediate snapshot of glycemic fluctuations compared with HbA1c's 2–3 months window [54], [55]. This

shorter timeframe may be particularly valuable during periods of rapid metabolic change or lifestyle modification, such as Ramadan fasting. Evidence from Ramadan-related diabetic populations suggests that fructosamine may help capture recent glycemic changes when used alongside established markers, although pediatric data remain limited [57].

Despite emerging evidence supporting fructosamine as a valuable short-term glycemic marker, data on its utility specifically in pediatric populations with type 1 diabetes during Ramadan fasting remain limited. Most available studies focus on adults, pregnant women, or CGM-derived outcomes, whereas fewer studies have evaluated the combined interpretation of HbA1c and fructosamine in children. A deeper understanding of this dual-marker approach could enhance clinical decision-making by providing both medium-term and recent glycemic information during a period of marked lifestyle change.

The present study therefore aimed to compare HbA1c and fructosamine levels in pediatric patients with T1D before and after Ramadan fasting. Its scientific contribution lies in evaluating whether the combined use of a conventional medium-term marker (HbA1c) and a shorter-term marker (fructosamine) may provide a more nuanced assessment of glycemic control in children during Ramadan, a context characterized by abrupt changes in meal timing, sleep patterns, insulin administration, and physical activity.

2. Materials and Methods

This comparative observational study was conducted between February and April 2025 at EL NOUR Laboratory in Cheria, Tebessa. The study enrolled 18 children aged under 18 years with a confirmed diagnosis of T1D. Inclusion criteria comprised a documented diagnosis of T1D, age below 18 years, and regular clinical follow-up at the participating medical facility. Exclusion criteria were the presence of other diabetes types (e.g., type 2 diabetes, monogenic diabetes), known hemoglobinopathies, or any medical conditions affecting protein metabolism that could potentially interfere with fructosamine measurements.

Available baseline variables included age and sex. Detailed clinical characteristics such as diabetes duration, insulin regimen, body mass index (BMI), CGM use, number of fasting days, and adherence to Ramadan fasting were not systematically recorded in the source files and were therefore not included in the primary analysis.

Written informed consent was obtained from parents or legal guardians of all participants, and verbal assent was collected from children when developmentally appropriate. The study protocol was reviewed and approved by the local ethics committee at EL NOUR Laboratory, in accordance with the Declaration of Helsinki.

Blood samples were collected at two time points: within one week prior to the start of Ramadan fasting and within one week following the end of Ramadan. To assess different glycemic parameters, three types of blood collection tubes were employed: heparin tubes for fasting plasma glucose, EDTA tubes for HbA1c, and dry tubes for serum fructosamine analysis.

Analytical procedures were performed utilizing a suite of validated instruments to ensure accurate and reliable measurement of glycemic markers. Biochemical assays, including fasting plasma glucose and fructosamine, were conducted on the Mindray BS-240Pro automated analyzer, employing enzymatic colorimetric methods consistent with manufacturer protocols. Hematological parameters were assessed using the Mindray BA-88A analyzer. Point-of-care glucose levels were monitored with the SD BIOSENSOR F200 device to provide immediate capillary glucose readings. HbA1c analysis was carried out on the Lifotronic GH-900Plus platform, utilizing a high-performance methodology standardized against reference protocols. All assays were calibrated regularly, and stringent quality control procedures including the use of internal controls and participation in external proficiency testing were implemented throughout the study to maintain assay precision and accuracy.

Statistical analysis was performed using paired pre- and post-Ramadan values. Continuous variables were expressed as mean ± standard deviation (SD). Pre- and post-Ramadan comparisons were performed using paired statistical tests after assessment of data distribution. Pearson's correlation analysis was used to evaluate the relationship between HbA1c and fructosamine, and results were reported with the correlation coefficient (r), sample size (n), p-value, and 95% confidence interval (CI). A p-value < 0.05 was considered statistically significant.

3. Results

In this cohort of 18 children with type 1 diabetes, the age distribution was skewed toward school-age participants, with 55.6% between 6–12 years, 33.3% between 12–18 years, and 11.1% between 1–6 years (Figure 1). The available baseline characteristics were limited to age and sex, which should be considered when interpreting subgroup-related findings.

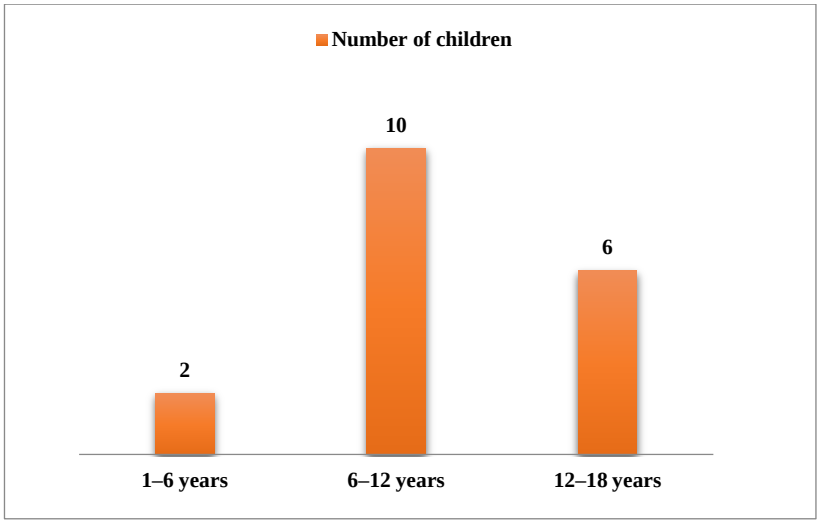

Figure 1: Age distribution of participants.

The sex distribution was equal, with males and females each representing 50% of the cohort. Reported contextual factors at or around the time of diabetes diagnosis are presented in Figure 2. These factors were descriptive only and were not interpreted as causal determinants of type 1 diabetes.

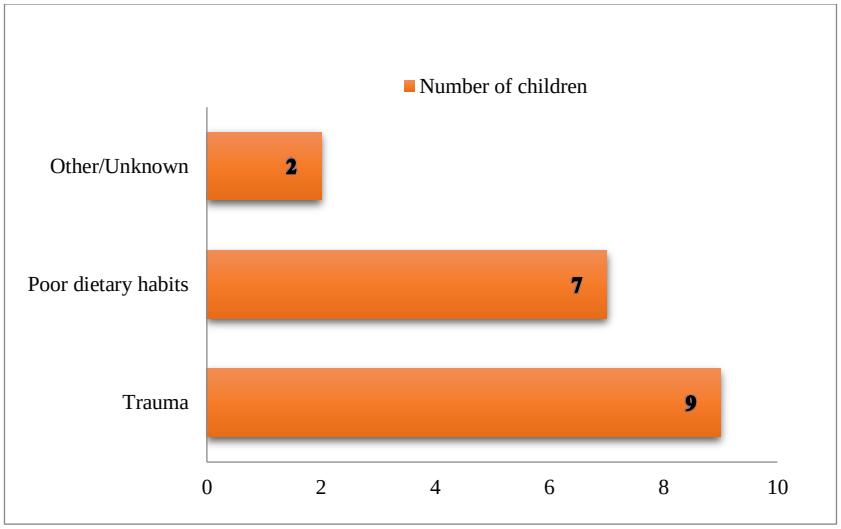

Figure 2: Reported contextual factors at or around diagnosis.

Quantitative analysis revealed an increase in mean fasting plasma glucose (FPG) from 2.16 ± 1.01 g/L prior to Ramadan to 2.41 ± 0.81 g/L post-Ramadan. This change was not statistically significant. Mean HbA1c rose significantly from 8.69% ± 1.51% before Ramadan to 9.34% ± 1.46% after Ramadan (p = 0.03), suggesting deterioration in overall glycemic control during the fasting period (Table 1, Figure 3).

Table 1: Glycemic markers before and after Ramadan in children with T1D

Marker	Pre-Ramadan (Mean ± SD)	Post-Ramadan (Mean ± SD)	*p*-value
FPG (g/L)	2.16 ± 1.01	2.41 ± 0.81	>0.05 (ns)
HbA1c (%)	8.69 ± 1.51	9.34 ± 1.46	0.03*
Fructosamine (µmol/L)	406.02 ± 91.03	426.21 ± 82.14	0.09 (ns)

FPG: fasting plasma glucose; ns: non-significant; *p < 0.05.

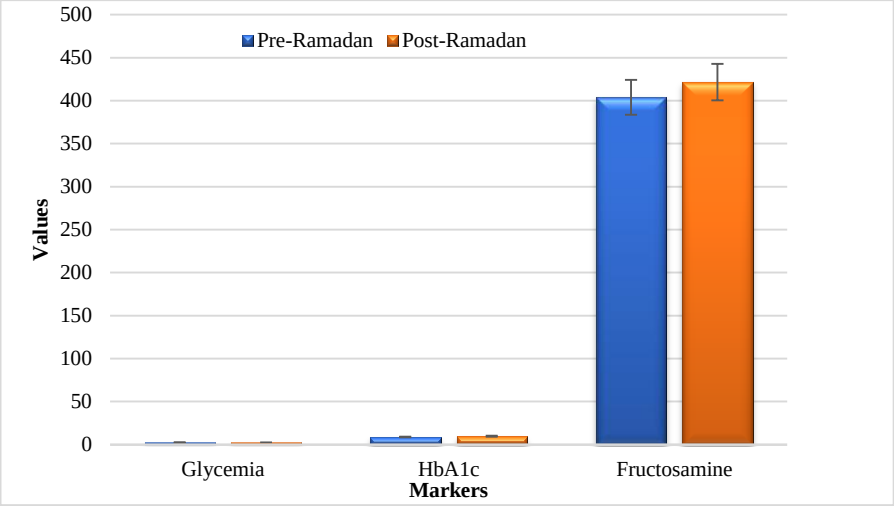

Figure 3: Mean FPG, HbA1c, and fructosamine levels before and after Ramadan, with standard deviations.

Fructosamine concentrations exhibited greater variability (Figure 4). The mean fructosamine value increased from 406.02 ± 91.03 µmol/L pre-Ramadan to 426.21 ± 82.14 µmol/L post-Ramadan; however, this change was not statistically significant ($p = 0.09$). Notably, five participants demonstrated reductions in fructosamine levels after Ramadan. This finding suggests heterogeneous short-term responses but should not be interpreted as definitive evidence of improved glycemic control because of the small sample size and lack of detailed fasting-adherence and treatment-adjustment data.

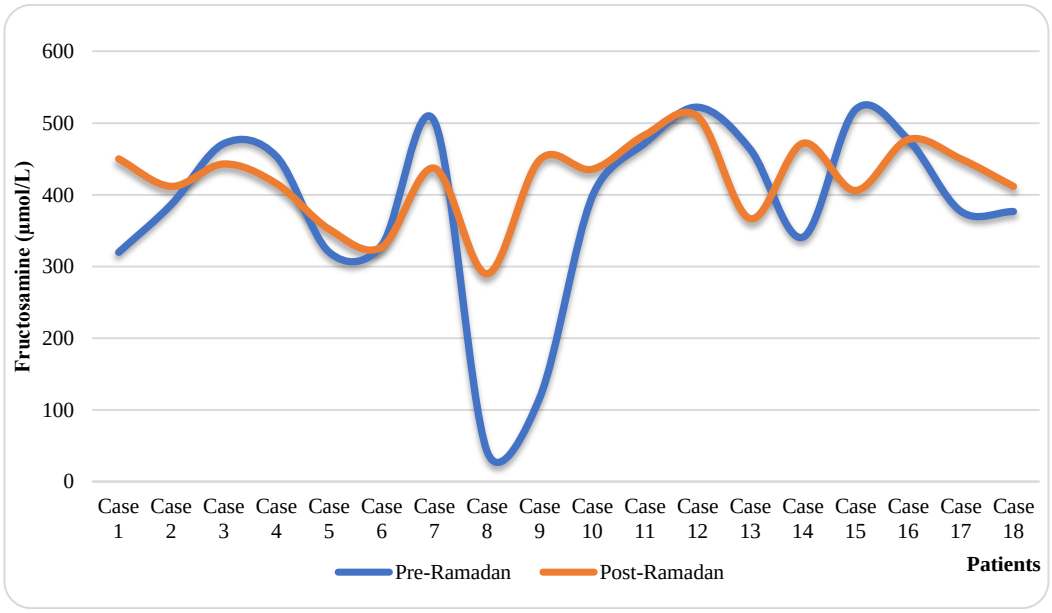

Figure 4: Individual fructosamine levels before and after Ramadan, showing variability across participants.

Pearson's correlation analysis demonstrated a moderate positive and statistically significant correlation between HbA1c and fructosamine levels ($r = 0.61$, 95% CI: 0.20–0.84, $p = 0.007$, $n = 18$). The corresponding coefficient of determination was $R^2 = 0.372$. This result suggests that higher HbA1c levels were associated with higher fructosamine concentrations in the study population.

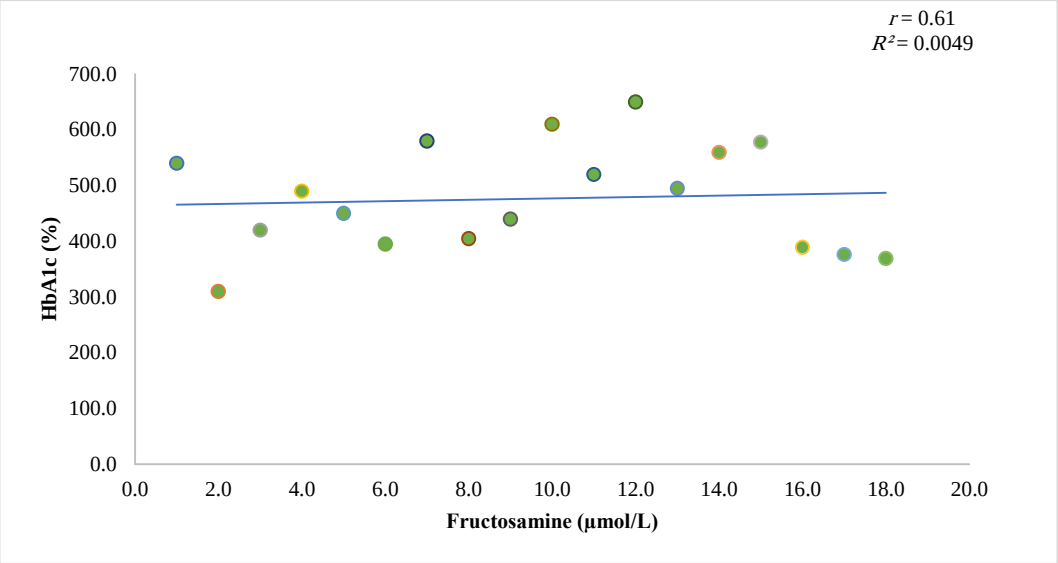

Figure 5: Scatter plot showing the relationship between HbA1c and fructosamine levels with regression line. Pearson correlation: (r = 0.61, 95% CI: 0.20–0.84, p = 0.007, n = 18).

4. Discussion

This study's finding of a significant increase in HbA1c post-Ramadan corroborates earlier reports indicating that fasting during Ramadan can adversely affect long-term glycemic control in patients with T1D, particularly when accompanied by disruptions in routine meal timing, physical activity, and insulin regimens [54], [55]. Recent systematic reviews have reinforced that glycemic instability during Ramadan is common among children and adolescents with T1D, especially in contexts lacking structured pre-Ramadan education or personalized treatment adjustments [56], [57], [58], [59]. For example, a multicenter observational study by [60] reported that inadequate preparation and support during Ramadan fasting increased the risk of both hyperglycemia and hypoglycemia in pediatric patients, emphasizing the critical role of individualized care plans.

The variable fructosamine responses observed in this study suggest that this biomarker may provide complementary short-term information during Ramadan fasting. Because fructosamine reflects average glucose exposure over approximately 2–3 weeks, it may be useful for identifying recent glycemic changes that are less readily captured by HbA1c [55], [61]. However, the non-significant post-Ramadan change in fructosamine observed in this small cohort requires cautious interpretation. Therefore, these findings should be considered exploratory and hypothesis-generating rather than confirmatory.

The moderate and statistically significant positive correlation observed between HbA1c and fructosamine in this study (r = 0.61, 95% CI: 0.20–0.84, p = 0.007, n = 18) supports their complementary roles in glycemic assessment. HbA1c remains essential for evaluating longer-term glycemic exposure and estimating the risk of chronic diabetes-related complications [67]. Fructosamine, in contrast, reflects more recent glycemic changes and may provide additional information on short-term metabolic trends, potentially helping clinicians identify early deterioration or improvement and adjust management more promptly [55].

This dual-marker approach may be particularly relevant in pediatric patients, who frequently experience rapid metabolic and behavioral changes that influence glucose regulation [68], [69]. Ramadan introduces additional complexity by imposing abrupt alterations in dietary intake, insulin timing, sleep, and physical activity, thereby increasing the risk of glycemic excursions [49]. Our findings suggest that fructosamine could be considered as an adjunctive marker during Ramadan, while emphasizing that its clinical utility requires confirmation in larger and better-characterized pediatric cohorts.

Nevertheless, several limitations merit consideration. First, the modest sample size restricts statistical power and generalizability. Second, fructosamine was measured only before and after Ramadan; an additional mid-Ramadan measurement, ideally at approximately two-week intervals, would better demonstrate its capacity to detect rapid glycemic changes compared with HbA1c. Third, detailed clinical and lifestyle data, including diabetes duration, insulin regimen, BMI, CGM use, number of fasting days, dietary intake, physical activity, insulin dose adjustments, and adherence to Ramadan fasting, were not

systematically available. Fourth, although the cohort was balanced by sex, the limited sample size and absence of pubertal staging, such as Tanner-stage assessment, precluded subgroup analyses by sex or pre-/post-pubertal status. Future prospective multicenter studies should incorporate these variables together with CGM metrics and structured Ramadan education interventions to better delineate fasting impacts and refine the clinical utility of fructosamine.

In summary, this study adds preliminary evidence supporting fructosamine as a potentially useful adjunct to HbA1c for monitoring short-term glycemic changes during Ramadan fasting in pediatric T1D. The findings should be interpreted cautiously because fructosamine changes did not reach statistical significance and because key clinical variables were unavailable. Tailored education, individualized therapeutic adjustments, and close monitoring remain essential to minimize risks and optimize glycemic control during this period.

5. Conclusion

Fructosamine may represent a useful complementary biomarker to HbA1c for assessing short-term glycemic changes in children with type 1 diabetes during periods of lifestyle modification such as Ramadan fasting. In this small observational cohort, HbA1c increased significantly after Ramadan, suggesting deterioration in overall glycemic control, whereas fructosamine showed heterogeneous individual responses without reaching statistical significance. These findings support the potential value of a dual-marker approach but should be interpreted cautiously due to the limited sample size and absence of detailed clinical and lifestyle data. Larger prospective studies incorporating mid-Ramadan fructosamine measurements, CGM metrics, insulin regimen data, pubertal status, and fasting adherence are needed to confirm these preliminary observations.

Acknowledgements

We thank the EL NOUR Laboratory team and the families who participated in this study.

Conflict of Interest: None declared.

Funding: This study received no external funding.

List of Abbreviations

Abbreviation	Full Term
T1D	Type 1 Diabetes
HbA1c	Glycated Hemoglobin
CGM	Continuous Glucose Monitoring
EDTA	Ethylenediaminetetraacetic Acid
SD	Standard Deviation
r	Pearson's Correlation Coefficient
mmol/L	Millimoles per Liter
µmol/L	Micromoles per Liter
FPG	Fasting Plasma Glucose
BMI	Body Mass Index

References

[1] T. A. Addissouky, M. M. A. Ali, I. E. T. El Sayed, and Y. Wang, 'Type 1 diabetes mellitus: retrospect and prospect', *Bull. Natl. Res. Cent.*, vol. 48, no. 1, p. 42, Apr. 2024, doi: 10.1186/s42269-024-01197-z.

[2] J. Lucier and P. M. Mathias, 'Type 1 Diabetes', in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2025. Accessed: Aug. 23, 2025. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK507713/

[3] M. S. Popoviciu, N. Kaka, Y. Sethi, N. Patel, H. Chopra, and S. Cavalu, 'Type 1 Diabetes Mellitus and Autoimmune Diseases: A Critical Review of the Association and the Application of Personalized Medicine', *J. Pers. Med.*, vol. 13, no. 3, p. 422, Feb. 2023, doi: 10.3390/jpm13030422.

[4] S. Roy, P. Pokharel, and J. D. Piganelli, 'Decoding the immune dance: Unraveling the interplay between beta cells and type 1 diabetes', *Mol. Metab.*, vol. 88, p. 101998, Oct. 2024, doi: 10.1016/j.molmet.2024.101998.

[5] A. R. Kahkoska and D. Dabelea, 'Diabetes in Youth: A Global Perspective', *Endocrinol. Metab. Clin. North Am.*, vol. 50, no. 3, pp. 491–512, Sep. 2021, doi: 10.1016/j.ecl.2021.05.007.

[6] N. Kandemir *et al.*, 'Epidemiology of type 1 diabetes mellitus in children and adolescents: A 50□year, single□ center experience', *J. Diabetes*, vol. 16, no. 5, p. e13562, Apr. 2024, doi: 10.1111/1753-0407.13562.

[7] K. I. Aamodt and A. C. Powers, 'The pathophysiology, presentation and classification of Type 1 diabetes', *Diabetes Obes. Metab.*, vol. 27, no. S6, pp. 15–27, 2025, doi: 10.1111/dom.16628.

[8] M. Z. Banday, A. S. Sameer, and S. Nissar, 'Pathophysiology of diabetes: An overview', *Avicenna J. Med.*, vol. 10, no. 4, pp. 174–188, Oct. 2020, doi: 10.4103/ajm.ajm_53_20.
[9] M. Du *et al.*, 'Advances in the Pathogenesis and Treatment Strategies for Type 1 Diabetes Mellitus', *Int. Immunopharmacol.*, vol. 148, p. 114185, Feb. 2025, doi: 10.1016/j.intimp.2025.114185.
[10] P. Houeiss, S. Luce, and C. Boitard, 'Environmental Triggering of Type 1 Diabetes Autoimmunity', *Front. Endocrinol.*, vol. 13, p. 933965, Jul. 2022, doi: 10.3389/fendo.2022.933965.
[11] M. A. Atkinson, G. S. Eisenbarth, and A. W. Michels, 'Type 1 diabetes', *The Lancet*, vol. 383, no. 9911, pp. 69–82, Jan. 2014, doi: 10.1016/S0140-6736(13)60591-7.
[12] S. Azar, N. Maroun Abou Jaoude, A. Kędzia, and E. Niechciał, 'Barriers to Type 1 Diabetes Adherence in Adolescents', *J. Clin. Med.*, vol. 13, no. 19, p. 5669, Jan. 2024, doi: 10.3390/jcm13195669.
[13] M. Bahal, V. Pande, J. Dua, and S. Mane, 'Advances in Type 1 Diabetes Mellitus Management in Children', *Cureus*, vol. 16, no. 8, p. e67377, 2024, doi: 10.7759/cureus.67377.
[14] B. Bombaci *et al.*, 'Psychological and Clinical Challenges in the Management of Type 1 Diabetes during Adolescence: A Narrative Review', *Children*, vol. 11, no. 9, p. 1085, Sep. 2024, doi: 10.3390/children11091085.
[15] M. Kropp *et al.*, 'Diabetic retinopathy as the leading cause of blindness and early predictor of cascading complications—risks and mitigation', *EPMA J.*, vol. 14, no. 1, pp. 21–42, Mar. 2023, doi: 10.1007/s13167-023-00314-8.
[16] S. K. Kunutsor *et al.*, 'Glycaemic control and macrovascular and microvascular outcomes: A systematic review and meta-analysis of trials investigating intensive glucose-lowering strategies in people with type 2 diabetes', *Diabetes Obes. Metab.*, vol. 26, no. 6, pp. 2069–2081, 2024, doi: 10.1111/dom.15511.
[17] B. Roy, 'Pathophysiological Mechanisms of Diabetes-Induced Macrovascular and Microvascular Complications: The Role of Oxidative Stress', *Med. Sci.*, vol. 13, no. 3, p. 87, Sep. 2025, doi: 10.3390/medsci13030087.
[18] M. Zakir *et al.*, 'Cardiovascular Complications of Diabetes: From Microvascular to Macrovascular Pathways', *Cureus*, vol. 15, no. 9, p. e45835, 2023, doi: 10.7759/cureus.45835.
[19] A. M. Almurashi, E. Rodriguez, and S. K. Garg, 'Emerging Diabetes Technologies: Continuous Glucose Monitors/Artificial Pancreases', *J. Indian Inst. Sci.*, vol. 103, no. 1, pp. 205–230, Jan. 2023, doi: 10.1007/s41745-022-00348-3.
[20] R. Cardona-Hernandez *et al.*, 'New therapies towards a better glycemic control in youths with type 1 diabetes', *Pharmacol. Res.*, vol. 195, p. 106882, Sep. 2023, doi: 10.1016/j.phrs.2023.106882.
[21] S. Y. Kwon and J. S. Moon, 'Advances in Continuous Glucose Monitoring: Clinical Applications', *Endocrinol. Metab.*, vol. 40, no. 2, pp. 161–173, Apr. 2025, doi: 10.3803/EnM.2025.2370.
[22] L. Morales-Dopico and S. A. MacLeish, 'Expanding the horizon of continuous glucose monitoring into the future of pediatric medicine', *Pediatr. Res.*, vol. 96, no. 6, pp. 1464–1474, Nov. 2024, doi: 10.1038/s41390-024-03573-x.
[23] J. H. Yoo and J. H. Kim, 'Advances in Continuous Glucose Monitoring and Integrated Devices for Management of Diabetes with Insulin-Based Therapy: Improvement in Glycemic Control', *Diabetes Metab. J.*, vol. 47, no. 1, pp. 27–41, Jan. 2023, doi: 10.4093/dmj.2022.0271.
[24] K. Islam *et al.*, 'Diabetes Mellitus and Associated Vascular Disease: Pathogenesis, Complications, and Evolving Treatments', *Adv. Ther.*, vol. 42, no. 6, pp. 2659–2678, 2025, doi: 10.1007/s12325-025-03185-9.
[25] P. Małkowska, 'Positive Effects of Physical Activity on Insulin Signaling', *Curr. Issues Mol. Biol.*, vol. 46, no. 6, pp. 5467–5487, Jun. 2024, doi: 10.3390/cimb46060327.
[26] F. Nurkolis *et al.*, 'New insight on dietary strategies to increase insulin sensitivity and reduce diabetes prevalence: an expert perspective and recommendation', *Discov. Food*, vol. 5, no. 1, pp. 1–32, Dec. 2025, doi: 10.1007/s44187-025-00422-6.
[27] W. Shkorfu, A. Fadel, M. Hamsho, Y. Ranneh, and H. M. Shahbaz, 'Intermittent Fasting and Hormonal Regulation: Pathways to Improved Metabolic Health', *Food Sci. Nutr.*, vol. 13, no. 8, p. e70586, 2025, doi: 10.1002/fsn3.70586.
[28] U. S. A. Syeda, D. Battillo, A. Visaria, and S. K. Malin, 'The importance of exercise for glycemic control in type 2 diabetes', *Am. J. Med. Open*, vol. 9, p. 100031, Jun. 2023, doi: 10.1016/j.ajmo.2023.100031.
[29] X. Zhao, X. An, C. Yang, W. Sun, H. Ji, and F. Lian, 'The crucial role and mechanism of insulin resistance in metabolic disease', *Front. Endocrinol.*, vol. 14, p. 1149239, Mar. 2023, doi: 10.3389/fendo.2023.1149239.
[30] J. Xie *et al.*, 'Adjusted glycated albumin is a novel indicator of glycemic control in patients with macroalbuminuria in diabetic kidney disease', *Sci. Rep.*, vol. 15, no. 1, p. 13812, Apr. 2025, doi: 10.1038/s41598-025-98641-5.
[31] G. Sartore, E. Ragazzi, R. Caprino, and A. Lapolla, 'Long-term HbA1c variability and macro-/micro-vascular complications in type 2 diabetes mellitus: a meta-analysis update', *Acta Diabetol.*, vol. 60, no. 6, pp. 721–738, Jun. 2023, doi: 10.1007/s00592-023-02037-8.
[32] A. G. Tabák *et al.*, 'Risk of Macrovascular and Microvascular Disease in Diabetes Diagnosed Using Oral Glucose Tolerance Test With and Without Confirmation by Hemoglobin A1c: The Whitehall II Cohort Study', *Circulation*, vol. 146, no. 13, pp. 995–1005, Sep. 2022, doi: 10.1161/CIRCULATIONAHA.122.059430.
[33] J. K. Tan, G. H. Lim, N. N. Mohamed Salim, S. Y. Chia, J. Thumboo, and Y. M. Bee, 'Associations Between Mean HbA1c, HbA1c Variability, and Both Mortality and Macrovascular Complications in Patients with Diabetes Mellitus: A Registry-Based Cohort Study', *Clin. Epidemiol.*, vol. 15, pp. 137–149, Jan. 2023, doi: 10.2147/CLEP.S391749.

[34] T.-E. Wu, Y.-W. Su, and H.-S. Chen, 'Mean HbA1c and HbA1c variability are associated with differing diabetes-related complications in patients with type 2 diabetes mellitus', *Diabetes Res. Clin. Pract.*, vol. 192, p. 110069, Oct. 2022, doi: 10.1016/j.diabres.2022.110069.
[35] X. Zhang *et al.*, 'Diabetes□Related Macrovascular Complications Are Associated With an Increased Risk of Diabetic Microvascular Complications: A Prospective Study of 1518 Patients With Type 1 Diabetes and 20 802 Patients With Type 2 Diabetes in the UK Biobank', *J. Am. Heart Assoc.*, vol. 13, no. 11, p. e032626, Jun. 2024, doi: 10.1161/JAHA.123.032626.
[36] S. Lazar, D.-V. Reurean-Pintilei, I. Ionita, V.-F. Avram, A. Herascu, and B. Timar, 'Glycemic Variability and Its Association with Traditional Glycemic Control Biomarkers in Patients with Type 1 Diabetes: A Cross-Sectional, Multicenter Study', *J. Clin. Med.*, vol. 14, no. 7, p. 2434, Apr. 2025, doi: 10.3390/jcm14072434.
[37] M. C. Nwadiugwu, D. R. Bastola, C. Haas, and D. Russell, 'Identifying Glycemic Variability in Diabetes Patient Cohorts and Evaluating Disease Outcomes', *J. Clin. Med.*, vol. 10, no. 7, p. 1477, Jan. 2021, doi: 10.3390/jcm10071477.
[38] American Diabetes Association, 'Standards of Medical Care in Diabetes—2022 Abridged for Primary Care Providers', *Clin. Diabetes*, vol. 40, no. 1, pp. 10–38, Jan. 2022, doi: 10.2337/cd22-as01.
[39] Z. Oganesova, J. Pemberton, and A. Brown, 'Innovative solution or cause for concern? The use of continuous glucose monitors in people not living with diabetes: A narrative review', *Diabet. Med.*, vol. 41, no. 9, p. e15369, 2024, doi: 10.1111/dme.15369.
[40] S. Bakhtiari, N. E. Timbrell, and S. M. D'Almeida, 'Abnormally Low HbA1c Caused by Hemolytic Anemia, a Case Report and Literature Review', *Br. J. Biomed. Sci.*, vol. 81, p. 13898, Jan. 2025, doi: 10.3389/bjbs.2024.13898.
[41] Z. Chen, L. Shao, M. Jiang, X. Ba, B. Ma, and T. Zhou, 'Interpretation of HbA1c lies at the intersection of analytical methodology, clinical biochemistry and hematology (Review) Corrigendum in /10.3892/etm.2023.11885', *Exp. Ther. Med.*, vol. 24, no. 6, pp. 1–11, Dec. 2022, doi: 10.3892/etm.2022.11643.
[42] E. Feskens *et al.*, 'Potential Markers of Dietary Glycemic Exposures for Sustained Dietary Interventions in Populations without Diabetes', *Adv. Nutr.*, vol. 11, no. 5, pp. 1221–1236, Sep. 2020, doi: 10.1093/advances/nmaa058.
[43] M. Migała, J. Chałubińska-Fendler, and M. Zielińska, '1,5-Anhydroglucitol as a Marker of Acute Hyperglycemia in Cardiovascular Events', *Rev. Diabet. Stud. RDS*, vol. 18, no. 2, pp. 68–75, Jun. 2022, doi: 10.1900/RDS.2022.18.68.
[44] H. Xu, R. Chen, X. Hou, N. Li, Y. Han, and S. Ji, 'The clinical potential of 1,5-anhydroglucitol as biomarker in diabetes mellitus', *Front. Endocrinol.*, vol. 15, Oct. 2024, doi: 10.3389/fendo.2024.1471577.
[45] M. M. Hassanein, W. Hanif, R. Malek, and A. Jabbar, 'Changes in fasting patterns during Ramadan, and associated clinical outcomes in adults with type 2 diabetes: A narrative review of epidemiological studies over the last 20 years', *Diabetes Res. Clin. Pract.*, vol. 172, p. 108584, Feb. 2021, doi: 10.1016/j.diabres.2020.108584.
[46] R. K. Ochani, A. Shaikh, S. Batra, G. Pikale, and S. Surani, 'Diabetes among Muslims during Ramadan: A narrative review', *World J. Clin. Cases*, vol. 11, no. 26, pp. 6031–6039, Sep. 2023, doi: 10.12998/wjcc.v11.i26.6031.
[47] A. Tootee and B. Larijani, 'Ramadan fasting and diabetes, latest evidence and technological advancements: 2021 update', *J. Diabetes Metab. Disord.*, vol. 20, no. 1, pp. 1003–1009, May 2021, doi: 10.1007/s40200-021-00804-4.
[48] T. Ali and N. Lessan, 'Chrononutrition in the context of Ramadan: Potential implications', *Diabetes Metab. Res. Rev.*, vol. 40, no. 2, p. e3728, 2024, doi: 10.1002/dmrr.3728.
[49] M. Hassanein *et al.*, 'Diabetes and Ramadan: Practical guidelines 2021', *Diabetes Res. Clin. Pract.*, vol. 185, Mar. 2022, doi: 10.1016/j.diabres.2021.109185.
[50] N. EL-Mohandes, G. Yee, B. S. Bhutta, and M. R. Huecker, 'Pediatric Diabetic Ketoacidosis', in *StatPearls*, Treasure Island (FL): StatPearls Publishing, 2025. Accessed: Sep. 01, 2025. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK470282/
[51] S. Foti Randazzese *et al.*, 'Severe Diabetic Ketoacidosis in Children with Type 1 Diabetes: Ongoing Challenges in Care', *Children*, vol. 12, no. 1, p. 110, Jan. 2025, doi: 10.3390/children12010110.
[52] A. Al Hayek, W. M. Al Zahrani, and M. A. Al Dawish, 'Implications of the Glycemia Risk Index in Assessing Metabolic Control and its Correlation With Therapy-Related Quality of Life During-Ramadan Fasting in Adults With Type 2 Diabetes', *J. Diabetes Sci. Technol.*, p. 19322968251321860, Feb. 2025, doi: 10.1177/19322968251321860.
[53] D. A. Diaz-Rizzolo *et al.*, 'Biological vs. Chronological Overnight Fasting: Influence of Last Evening Meal on Morning Glucose in Dysglycemia', *Nutrients*, vol. 17, no. 12, p. 2026, Jan. 2025, doi: 10.3390/nu17122026.
[54] L. J. de Oliveira Andrade, A. M. V. Bittencourt, L. F. M. de Brito, L. M. de Oliveira, and G. C. M. de Oliveira, 'Estimated average blood glucose level based on fructosamine level', *Arch. Endocrinol. Metab.*, vol. 67, no. 2, pp. 262–265, Jan. 2023, doi: 10.20945/2359-3997000000589.
[55] V. Gounden, M. Ngu, C. Anastasopoulou, and I. Jialal, 'Fructosamine', in *StatPearls [Internet]*, StatPearls Publishing, 2023. Accessed: Sep. 01, 2025. [Online]. Available: https://www.ncbi.nlm.nih.gov/sites/books/NBK470185/
[56] E. Bernier *et al.*, 'Trimester-Specific Serum Fructosamine in Association with Abdominal Adiposity, Insulin Resistance, and Inflammation in Healthy Pregnant Individuals', *Nutrients*, vol. 14, no. 19, p. 3999, Jan. 2022, doi: 10.3390/nu14193999.
[57] A. Ekhzaimy *et al.*, 'Fructosamine as an Index of Short-Term Glycemic Control in Pregnant Women with Diabetes: Before, During and After Ramadan', *Int. J. Womens Health*, vol. 14, pp. 435–444, Mar. 2022, doi: 10.2147/IJWH.S351654.

[58] A. M. Alguwaihes, E. Y. Alyusuf, A. Alrajeh, M. Alotaibi, and M. E. Al-Sofiani, 'Altered Ramadan fasting glycemic profiles of adults with type 1 diabetes reveal strong evidence of underestimated insulin adjustments: a 3-year observational study in Arab settings', *Front. Endocrinol.*, vol. 16, May 2025, doi: 10.3389/fendo.2025.1399990.
[59] M. Bencharif, I. Sersar, M. Bentaleb, F. Z. Boutata, and Y. Benabbas, 'Ramadan Fasting and NCDs-Example of the Diabetes', *Front. Nutr.*, vol. 9, Mar. 2022, doi: 10.3389/fnut.2022.787571.
[60] S. H. Ahmed *et al.*, 'Ramadan and Diabetes: A Narrative Review and Practice Update', *Diabetes Ther.*, vol. 11, no. 11, pp. 2477–2520, Nov. 2020, doi: 10.1007/s13300-020-00886-y.
[61] S. A. Beshyah, K. Hafidh, and H. Abusahmin, 'Diabetes and Ramadan Fasting (2024): A Thematic Overview', *J. Diabetes Endocr. Pract.*, vol. 08, pp. 157–165, May 2025, doi: 10.1055/s-0045-1809319.
[62] O. Safari *et al.*, 'Ramadan fasting among adolescents with type 1 diabetes: a systematic review and meta-analysis', *BMC Endocr. Disord.*, vol. 25, p. 45, Feb. 2025, doi: 10.1186/s12902-025-01835-1.
[63] S. T. Teoh, S. Hussain, and J. Y. H. Hong, 'Glycaemic Changes Among Children and Adolescents With Type 1 Diabetes Mellitus Before and During Ramadan Fasting Using Continuous Glucose Monitoring', *J. ASEAN Fed. Endocr. Soc.*, vol. 37, no. 2, pp. 49–59, Sep. 2022, doi: 10.15605/jafes.037.02.08.
[64] R. Malek, S. Hannat, A. Nechadi, F. Z. Mekideche, and M. Kaabeche, 'Diabetes and Ramadan: A multicenter study in Algerian population', *Diabetes Res. Clin. Pract.*, vol. 150, pp. 322–330, Apr. 2019, doi: 10.1016/j.diabres.2019.02.008.
[65] A. Al Hayek and M. A. Al Dawish, 'Improvement of Glycemia Risk Index and Continuous Glucose Monitoring Metrics During Ramadan Fasting in Type 1 Diabetes: A Real-World Observational Study', *J. Diabetes Sci. Technol.*, p. 19322968241301750, Nov. 2024, doi: 10.1177/19322968241301750.
[66] H. Patel and Anuradha N, 'Comparative analysis of fructosamine and HbA1c as a glycemic control marker in Type 2 diabetes patients in a tertiary care hospital study', *Asian J. Med. Sci.*, vol. 14, no. 10, pp. 73–78, Oct. 2023, doi: 10.3126/ajms.v14i10.53962.
[67] N. A. ElSayed *et al.*, '2. Classification and Diagnosis of Diabetes: Standards of Care in Diabetes—2023', *Diabetes Care*, vol. 46, no. Suppl 1, pp. S19–S40, Jan. 2023, doi: 10.2337/dc23-S002.
[68] B. E. Marks and J. I. Wolfsdorf, 'Monitoring of Pediatric Type 1 Diabetes', *Front. Endocrinol.*, vol. 11, Mar. 2020, doi: 10.3389/fendo.2020.00128.
[69] M. Nevo-Shenker, M. Phillip, R. Nimri, and S. Shalitin, 'Type 1 diabetes mellitus management in young children: implementation of current technologies', *Pediatr. Res.*, vol. 87, no. 4, pp. 624–629, Mar. 2020, doi: 10.1038/s41390-019-0665-4.