

Helicobacter pylori Infection and Its Impact on Biochemical and Hematological Parameters in Children with Type 1 Diabetes Mellitus

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Abstract

Background: *Helicobacter pylori* (*H. pylori*) is a prevalent chronic bacterial infection of the gastrointestinal tract, especially in developing regions, yet its extra-gastric consequences in chronic diseases remain insufficiently understood.

Objectives: This case control-study evaluated the influence of *H. pylori* infection on biochemical and hematological parameters in children with type 1 diabetes (T1D).

Methods: This case-control study enrolled 120 children, including 60 patients with T1DM and 60 apparently healthy, non-diabetic controls, age-matched controls. Participants were recruited from diabetes clinics in 2016. Interviews were completed, and blood samples were analyzed for serum *H. pylori* IgG using competitive ELISA, alongside various biochemical and hematological measurements.

Results: Children with T1D had lower household income, a stronger family history of diabetes, and higher rates of neuropathy and gastritis than healthy controls ($P < 0.05$). Significant differences were observed in several biochemical and hematological measures. *H. pylori* seropositivity was notably more prevalent among children with T1D than among non-diabetic participants (40.0% vs. 21.7%, $P = 0.030$). Within the diabetic group, infected children demonstrated higher levels of glycated hemoglobin, glucose, insulin, total cholesterol, triglycerides, LDL-C, ALT, and white blood cell counts. In contrast, HDL-C, red blood cell counts, and hemoglobin concentrations were lower in seropositive children. Overall, the findings suggest that *H. pylori* infection may be associated with poorer glycemic control and greater metabolic and hematological abnormalities in pediatric patients with T1D.

Conclusion: *H. pylori* infection was associated with unfavorable biochemical and hematological changes in children with T1D. Further prospective studies should evaluate whether eradication improves metabolic control.

Keywords: Biochemical Alterations, Children, *H. pylori*, Type 1 Diabetes

1. Background

T1D is the most prevalent chronic metabolic disorder among children and adolescents and results from autoimmune-mediated destruction of pancreatic β -cells, which are responsible for insulin production. Progressive loss of insulin secretion leads to persistent hyperglycemia and lifelong dependence on exogenous insulin to prevent severe complications, including cardiovascular disease, peripheral neuropathy, diabetic kidney disease, and retinopathy.^{1,2} Nevertheless, effective glycemic management can substantially reduce the risk of these complications and improve long-term outcomes in children with T1D.³ Insulin is a key regulator of metabolic homeostasis, coordinating carbohydrate, lipid, and protein metabolism. Consequently, insulin deficiency in T1D produces

widespread metabolic disturbances. Poorly controlled or untreated disease is characterized by increased circulating glucose, ketone bodies, free fatty acids, and branched-chain amino acids, largely as a result of enhanced hepatic gluconeogenesis and glycogenolysis, increased lipolysis, and accelerated proteolysis.⁴⁻⁶ In addition to these metabolic abnormalities, hematological alterations have been documented in children with T1D.⁷

H. pylori is a spiral-shaped, Gram-negative bacterium that commonly causes persistent infection of the gastrointestinal tract, with a particularly high burden in developing countries. Its global prevalence was estimated to be 43.1% during 2011–2022.⁸ Growing evidence has linked *H. pylori* infection not only to gastrointestinal disorders but also to several extragastric conditions,

including diabetes mellitus (DM), as well as cardiovascular and hematological diseases.⁹⁻¹² Studies have reported a greater prevalence of *H. pylori* infection among children with T1D than among their non-diabetic counterparts.^{13,14} Furthermore, T1D patients with *H. pylori* infection have been reported to exhibit higher glycated hemoglobin (HbA1c) and glucose concentrations, hyperinsulinemia, and dyslipidemia compared to uninfected patients.^{15,16} Anemia has also been associated with *H. pylori* infection in these patients.¹⁷ Collectively, these observations suggest that *H. pylori* infection may contribute to worsening metabolic and hematological disturbances in individuals with diabetes. Although findings from eradication studies may further clarify this relationship, such interventions are beyond the scope of the present investigation.

Despite increasing interest in the relationship between *H. pylori* and diabetes, evidence concerning its occurrence and clinical implications in children with T1D remains limited, particularly in resource-constrained settings. To our knowledge, no previous study has investigated the association between *H. pylori* infection and T1D among children in the Gaza Strip, Palestinian Territories. Previous research in this region has focused primarily on patients with type 2 diabetes, with only two published studies addressing *H. pylori* infection in this population.^{18,19}

2. Objectives

This study is the first to compare *H. pylori* infection and its impact on biochemical and hematological parameters in children with T1D and non-diabetic children in the Gaza Strip, Palestinian Territories.

3. Methods

3.1. Study Design, Sample Size and Sampling

This case-control study enrolled 120 children, including 60 patients with T1DM (cases) and 60 apparently healthy, non-diabetic controls. T1D was confirmed in accordance with the diagnostic criteria established by the World Health Organization (WHO).²⁰ The diabetic participants were recruited in 2016 from the diabetes units of Al-Ranteesy Hospital and the Palestinian Medical Relief Center in the Gaza Strip, whereas control participants were recruited from the general population. The two groups were matched by age and body mass index. The required sample size was estimated using the EPI-INFO statistical package, version 3.5.1, assuming a 95% confidence level, 80% statistical power, a conservative expected proportion of 50%, and an odds ratio greater than 2. The initial calculation yielded 54 participants per group for a 1:1 case-to-control ratio. To account for potential non-response or incomplete participation, the sample was increased to 60 children in each group. Children aged 18 years or younger were eligible. Participants were excluded if they were older than 18

years or had type 2 diabetes, previous gastrointestinal surgery, chronic diseases that could affect the study outcomes or hematological indices, goiter, peptic ulcer disease, diabetic vasculopathy complications, or recent use of antibiotics, anti-inflammatory agents, prokinetic medications, proton pump inhibitors, drugs that alter gastric acid secretion, hormone replacement therapy, or corticosteroids during the preceding two months.

3.2. Questionnaire Interviews and Patients' Records

A structured, study-specific questionnaire was developed and administered through face-to-face interviews conducted by the researcher. Its content was adapted from previously published studies investigating diabetes mellitus in the Gaza Strip, with appropriate modifications to suit the objectives of the present study.^{19,21} Most items were formulated as binary yes/no questions.²² To establish content validity, the questionnaire was reviewed by specialists in relevant medical disciplines who assessed the items for clarity, relevance, and appropriateness. Reliability and internal consistency were evaluated using Cronbach's alpha. Before the main data collection, the questionnaire was pilot tested among 10 participants who were excluded from the final study sample. The pilot assessment was used to evaluate the instrument's clarity and comprehensibility and to make any necessary revisions. The questionnaire addressed sociodemographic characteristics, including age, monthly household income, and family history of diabetes; clinical characteristics, such as self-reported complications, gastritis, peptic ulcer disease, and diabetes duration; and information regarding dietary practices and adherence to prescribed medications.

3.3. Specimen Collection and Processing

Following an overnight fast of 12 hours, 8 ml of venous blood was collected from each diabetic and non-diabetic child by a trained nurse. Approximately 3 ml of the sample was transferred into an EDTA-containing vacutainer tube for HbA1c and complete blood count analyses. The remaining blood was transferred into a plain plastic tube without anticoagulant and allowed to clot at room temperature for 20 minutes. The clotted samples were then centrifuged at 3,000 rpm for 10 minutes using a Rotina 46 centrifuge (Hettich, Japan). The resulting serum was carefully separated and stored appropriately until biochemical and serological analyses were performed.

3.4. *H. pylori*, Biochemical and Hematological Assay

Serum *H. pylori* IgG antibodies were quantitatively assessed using a competitive enzyme-linked immunosorbent assay (ELISA; Monobind, USA; Cat. No. 1425-300 IgG; 96-well format).²³ HbA1c was measured in whole-blood samples using a colorimetric assay (Stanbio Laboratory, Texas, USA).²⁴ Serum glucose concentrations were

determined by the glucose oxidase–peroxidase method using Labkit reagents (Spain).²⁵ Insulin levels were analyzed by microparticle enzyme immunoassay with the Abbott IMx Insulin assay.²⁶ Total cholesterol and triglycerides were quantified using cholesterol oxidase–peroxidase and glycerol phosphate oxidase–peroxidase methods, respectively, with BioSystems reagents (Spain).²⁷ High-density lipoprotein cholesterol (HDL-C) was measured using a precipitation-based assay,²⁸ whereas low-density lipoprotein cholesterol (LDL-C) was estimated using the Friedewald equation.²⁹ Serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were determined using an optimized ultraviolet method in accordance with International Federation of Clinical Chemistry and Laboratory Medicine recommendations, with DiaSys reagents.³⁰ Urea and creatinine concentrations were measured using the urease–glutamate dehydrogenase/UV and alkaline picrate methods, respectively, with BioSystems reagents.³¹ Hematological indices, including white blood cell (WBC) and red blood cell (RBC) counts, hemoglobin (Hb), hematocrit (HCT), and platelet counts, were determined using a Cell-Dyne 1800 automated hematology analyzer (Sequoia-Turner Corporation, California, USA) with appropriate controls and calibrators.

3.5. Statistical Analysis

Data were entered and analyzed using SPSS for Windows, version 24.0 (Statistical Package for the Social Sciences, IBM Corp., Chicago, IL, USA). Descriptive statistics and cross-tabulations were performed to summarize the study variables. Differences between categorical variables were assessed using the chi-square (χ^2) test. When fewer than 20% of cells had an expected frequency below 5, Yates' continuity correction was

applied. Continuous data were presented as mean $\pm$ standard deviation (SD), and comparisons between two independent groups were conducted using the independent-samples t-test. This approach was used, for example, to compare quantitative parameters such as insulin concentrations between diabetic and non-diabetic children. A P -value < 0.05 was considered indicative of statistical significance. The percentage difference between two values was calculated as the absolute difference divided by their mean, multiplied by 100, according to the following equation: Percentage difference = $|V1 - V2| / [(V1 + V2)/2] \times 100$.

4. Results

4.1. Sociodemographic and Clinical Data of the Study Population

Table 1 demonstrates that the mean age did not differ significantly between diabetic and non-diabetic children (11.1 ± 3.9 vs. 11.2 ± 3.8 years, respectively; $P = 0.907$). In contrast, children with diabetes had significantly lower monthly household income and were more likely to report a family history of diabetes ($\chi^2_{(corrected)} = 6.295$, $P = 0.043$; $\chi^2 = 7.502$, $P = 0.006$, respectively). Neuropathy and gastritis were also significantly more frequent among diabetic children, occurring in 10 (16.7%) and 8 (13.3%) participants, respectively, compared with 1 (1.7%) case for each condition among non-diabetic children ($\chi^2_{(corrected)} = 6.405$, $P = 0.011$; $\chi^2_{(corrected)} = 4.324$, $P = 0.038$, respectively). As shown in Table 2, most diabetic children had been diagnosed within the previous five years (40, 66.7%), followed by those with a disease duration of 5–10 years (17, 28.3%). Only 3 (5.0%) had diabetes for more than 10 years. Regarding management, 15 (25.0%) followed a prescribed diet, while 57 (95.0%) reported adherence to their medication regimen.

Table 1. Sociodemographic and Clinical Data of the Study Population (n = 120)

Variable	Controls (N = 60)	Cases (N = 60)	Test	P-value
Age (year)	11.2 $\pm$ 3.8	11.1 $\pm$ 3.9	t	0.118
Family income/month (NIS)				
< 1000	26 (43.3)	19 (31.7)	χ^2	6.295
1000 - 2000	24 (40.0)	38 (63.3)		
> 2000	10 (16.7)	3 (5.0)		
Family history of diabetes				
Yes	22 (36.7)	37 (61.7)	χ^2	7.502
No	38 (63.3)	23 (38.3)		
Retinopathy				
Yes	2 (3.3)	7 (11.7)	χ^2	1.922
No	58 (96.7)	53 (88.3)		
Neuropathy				
Yes	1 (1.7)	10 (16.7)	χ^2	6.405
No	59 (98.3)	50 (83.3)		
Gastritis				
Yes	1 (1.7)	8 (13.3)	χ^2	4.324
No	59 (98.3)	52 (86.7)		
Peptic ulcer				
Yes	1 (1.7)	4 (6.7)	χ^2	0.835
No	59 (98.3)	56 (93.3)		

N: Number, Controls: Healthy non-diabetic children, Cases: Children suffering from T1D, Values are No. (%) except age where values are expressed as mean $\pm$ standard deviation, NIS: New Israeli Shekel (~ 0.27 \$US), * P -value of $\chi^2_{(corrected)}$ test, $P < 0.05$: Significant, $P > 0.05$: Not significant.

Table 2. Duration of Diabetes, Diet and Compliance to Medication among Diabetic Children

Variable	Cases	
	Number (60)	Percentage
Duration of diabetes (year)		
< 5	40	66.7
5 - 10	17	28.3
> 10	3	5.0
Diet		
Yes	15	25.0
No	45	75.0
Compliance to medication		
Yes	57	95.0
No	3	5.0

Cases: Children suffering from T1D.

4.2. Biochemical and Hematological Profile of the Study Population

Table 3 presents the biochemical and hematological profiles of children with diabetes compared with those of non-diabetic controls. Diabetic children had significantly higher mean concentrations of HbA1c, glucose, and insulin than their non-diabetic counterparts ($9.8 \pm 1.8\%$, 169.6 ± 111.9 mg/dl, and 26.7 ± 17.0 mIU/ml vs. $5.0 \pm 0.5\%$, 77.7 ± 7.0 mg/dl, and 10.6 ± 4.7 mIU/ml, respectively), corresponding to differences of 64.8%, 74.3%, and 86.3% ($P = 0.001$ for all). Serum AST and ALT activities were

also significantly elevated in the diabetic group (32.1 ± 7.8 and 26.5 ± 12.4 U/L vs. 22.0 ± 10.5 and 19.5 ± 7.5 U/L), with differences of 37.3% and 30.4%, respectively ($P < 0.001$). In addition, the WBC count was significantly higher among diabetic children ($7.5 \pm 2.4 \times 10^9/L$ vs. $6.7 \pm 1.8 \times 10^9/L$; difference = 11.3%, $P = 0.030$). Conversely, RBC counts and hemoglobin concentrations were significantly lower in diabetic children than in controls ($4.5 \pm 0.4 \times 10^{12}/L$ and 11.7 ± 1.2 g/dl vs. $4.7 \pm 0.5 \times 10^{12}/L$ and 12.4 ± 1.3 g/dl), representing differences of 4.3% and 5.8%, respectively ($P = 0.016$ and $P = 0.005$).

Table 3. Biochemical and Hematological Profile of the Study Population

Parameter	Controls	Cases	% difference	P-value
	(N = 60)	(N = 60)		
HbA1c (%)	5.0 ± 0.5	9.8 ± 1.8	64.8	0.001
Glucose (mg/dl)	77.7 ± 7.0	169.6 ± 111.9	74.3	0.001
Insulin (mIU/ml)	10.6 ± 4.7	26.7 ± 17.0	86.3	0.001
Cholesterol (mg/dl)	164.0 ± 18.4	171.3 ± 26.9	4.5	0.093
Triglycerides (mg/dl)	110.2 ± 28.3	121.8 ± 37.7	10.0	0.064
LDL-C (mg/dl)	88.3 ± 18.4	95.0 ± 25.5	7.3	0.082
HDL-C (mg/dl)	54.6 ± 2.5	53.7 ± 3.3	-1.6	0.080
AST (U/L)	22.0 ± 10.5	32.1 ± 7.8	37.3	< 0.001
ALT (U/L)	19.5 ± 7.5	26.5 ± 12.4	30.4	< 0.001
Urea (mg/dl)	25.3 ± 3.9	26.6 ± 4.5	5.9	0.092
Creatinine (mg/dl)	0.74 ± 0.08	0.76 ± 0.07	2.6	0.108
WBCs $\times 10^9/L$	6.7 ± 1.8	7.5 ± 2.4	11.3	0.030
RBCs $\times 10^{12}/L$	4.7 ± 0.5	4.5 ± 0.4	-4.7	0.016
Hb (g/dl)	12.4 ± 1.3	11.7 ± 1.2	-5.8	0.005
Hct (%)	38.9 ± 4.0	37.8 ± 3.8	-3.8	0.110
PLT $\times 10^9/L$	302.0 ± 76.5	319.2 ± 90.6	5.5	0.268

Controls: Healthy non-diabetic children, Cases: Children suffering from T1D, HbA1c: Glycated hemoglobin, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, WBC: White blood cell, RBC: Red blood cell, Hb: Hemoglobin, Hct: Hematocrit, PLT: Platelet, N.: Number. All values are presented as mean $\pm$ SD, $P < 0.05$: Significant, $P > 0.05$: Not significant.

4.3. Seroprevalence Rate of *H. pylori* and Its Impact on Biochemical and Hematological Profile of Diabetic Children

Table 4 demonstrates a significantly greater seroprevalence of *H. pylori* among children with T1D compared with non-diabetic controls (24/60 [40.0%] vs. 13/60 [21.7%]; $\chi^2 = 4.728$, $P = 0.030$). Children with T1D were approximately 1.8 times more likely to test positive for *H. pylori* IgG. As shown in Table 5, within the T1D group, seropositive children had significantly higher mean HbA1c, glucose, and insulin concentrations than seronegative children ($10.5 \pm 1.7\%$ vs. $9.5 \pm 1.7\%$, 233.8

± 145.2 vs. 146.3 ± 87.9 mg/dl, and 34.2 ± 21.7 vs. 23.9 ± 14.2 mIU/ml, with $P = 0.046$, 0.006, and 0.037, respectively). Children who were seropositive for *H. pylori* had significantly higher concentrations of cholesterol, triglycerides, and LDL-C than seronegative children (186.2 ± 35.0 , 135.5 ± 44.2 , and 107.9 ± 32.9 mg/dl vs. 157.8 ± 19.4 , 99.5 ± 29.4 , and 83.4 ± 18.6 mg/dl, respectively; $P = 0.001$ for all). In contrast, HDL-C levels were significantly lower among seropositive children (51.3 ± 3.7 mg/dl vs. 54.6 ± 2.6 mg/dl, $P = 0.001$). ALT activity was also significantly elevated in children with *H. pylori* infection (29.1 ± 8.5 U/L vs.

24.5 ± 7.3 U/L, $P = 0.045$). Similarly, the WBC count was significantly higher in infected children ($7.8 \pm 1.5 \times 10^9/L$ vs. $6.7 \pm 1.9 \times 10^9/L$, $P = 0.041$). Conversely, RBC and Hb levels were significantly lower in

seropositive children than in seronegative children ($4.6 \pm 0.5 \times 10^{12}/L$ and 11.9 ± 1.1 g/dl vs. $4.9 \pm 0.4 \times 10^{12}/L$ and 13.0 ± 1.4 g/dl, $P = 0.023$ and $P = 0.012$, respectively).

Table 4. Distribution of *H. pylori* Immunoglobulin G among the Study Population

<i>H. pylori</i> IgG	Controls (N = 60)		Cases (N = 60)		χ^2	<i>P</i> -value
	No	%	No	%		
Positive	13	21.7	24	40.0	4.728	0.030
Negative	47	78.3	36	60.0		

Controls: Healthy non-diabetic children, Cases: Children suffering from T1D, IgG: Immunoglobulin G, N: Number, $P < 0.05$: Significant.

Table 5. Biochemical and Hematological Profile in *H. pylori* Positive and *H. pylori* Negative Type 1 Diabetic Children

Parameter	<i>H. pylori</i> positive (N = 24)	<i>H. pylori</i> negative (N = 36)	<i>P</i> -value
HbA1c (%)	10.5 ± 1.7	9.5 ± 1.7	0.046
Glucose (mg/dl)	233.8 ± 145.2	146.3 ± 87.9	0.006
Insulin (mIU/ml)	34.2 ± 21.7	23.9 ± 14.2	0.037
Cholesterol (mg/dl)	186.2 ± 35.0	157.8 ± 19.4	0.001
Triglycerides (mg/dl)	135.5 ± 44.2	99.5 ± 29.4	0.001
LDL-C (mg/dl)	107.9 ± 32.9	83.4 ± 18.6	0.001
HDL-C (mg/dl)	51.3 ± 3.7	54.6 ± 2.6	0.001
AST (U/L)	33.9 ± 9.8	30.4 ± 7.0	0.123
ALT (U/L)	29.1 ± 8.5	24.5 ± 7.3	0.045
Urea (mg/dl)	26.9 ± 4.6	25.0 ± 3.7	0.128
Creatinine (mg/dl)	0.77 ± 0.06	0.75 ± 0.07	0.162
WBCs $\times 10^9/L$	7.8 ± 1.5	6.7 ± 1.9	0.011
RBCs $\times 10^{12}/L$	4.6 ± 0.5	4.9 ± 0.4	0.023
Hb (g/dl)	11.9 ± 1.1	13.0 ± 1.4	0.012
Hct (%)	37.4 ± 4.1	38.8 ± 2.9	0.135
PLT $\times 10^9/L$	326.4 ± 98.0	294.3 ± 61.2	0.192

All values are expressed as mean ± Standard deviation, *H. pylori*: *Helicobacter pylori*, HbA1c: Glycated hemoglobin, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, WBC: White blood cell, RBC: Red blood cell, Hb: Hemoglobin, Hct: Hematocrit, PLT: Platelet, $P < 0.05$: Significant, $P > 0.05$: Not significant.

5. Discussion

Previous research on concomitant *H. pylori* infection and T1D remains limited, particularly among children. Although several studies have reported an association between *H. pylori* infection and T1D in pediatric populations,^{9,13,14,32} other investigations have found no such relationship.^{33,34} In the present study, *H. pylori* infection was more prevalent among children with T1D and was associated with significant changes in biochemical and hematological parameters. Although *H. pylori* eradication in children with T1D could help clarify whether the infection contributes to these metabolic abnormalities, evaluating this issue was beyond the scope of the current investigation. To our knowledge, this case-control study is the first to assess the prevalence of *H. pylori* infection and its association with biochemical and hematological abnormalities among children with T1D compared with non-diabetic children in the Gaza Strip, Palestinian Territories.

Sociodemographic and clinical characteristics indicated that children with diabetes had significantly lower monthly household income and a greater family history of diabetes than non-diabetic children. Diabetes is known to disproportionately affect individuals from lower-

income populations.³⁵ In children with T1D, socioeconomic disadvantage has been associated with poorer health outcomes and inadequate glycemic control.³⁶ Therefore, the lower household income observed in our diabetic participants may contribute to an increased risk of diabetes-related complications. This interpretation is consistent with our finding that neuropathy was significantly more prevalent among diabetic children than among controls. A prospective study examining risk factors for complications in individuals with childhood-onset T1D found that lower family income during early adulthood predicted subsequent nerve damage, potentially reflecting the effects of inadequate glycemic control.³⁷ Diabetic neuropathy is a recognized complication of pediatric T1D, and persistent poor glycemic control is an important determinant of its progression.^{38,39} A positive family history is also an established risk factor for T1D.^{40,41} Alhonen and colleagues reported that T1D tends to cluster not only within the nuclear families of affected children but also across their extended families.⁴² Furthermore, gastritis was significantly more common among diabetic children in our study. This finding is compatible with evidence that individuals with T1D are at increased risk of other autoimmune and multisystem

disorders, including gastritis.⁴³

Approximately two-thirds of the diabetic children in our study had been diagnosed with T1D for less than five years, suggesting that the disease is commonly diagnosed during childhood and adolescence. The typical age at presentation is reported to be between 6 and 16 years, although an increasing proportion of cases are now being diagnosed at younger ages.⁴⁴ Although approximately three-quarters of the diabetic children were not following a prescribed diet, most participants reported adherence to their medications. Comparable findings have been reported previously.⁴⁵ Dietary management and glycemic control are closely interconnected components of T1D management. Nevertheless, because dietary patterns and specific food intake were not assessed in detail in this study, this aspect warrants further investigation. Healthcare professionals and nutrition educators should therefore emphasize dietary adherence as an integral component of diabetes management in children with T1D.

The biochemical and hematological findings demonstrated significantly higher levels of HbA1c, glucose, insulin, AST, ALT, and WBCs, together with significantly lower RBC counts and hemoglobin concentrations, among diabetic children compared with non-diabetic controls. Similar abnormalities have been described in individuals with T1D.^{21,46,48} HbA1c is a key biomarker for assessing long-term glycemic control and is routinely used to guide diabetes management, including insulin therapy.⁴⁹ The elevated HbA1c concentrations observed in our diabetic participants are therefore consistent with the poor glycemic control that may contribute to the development of neuropathy. The increased insulin concentrations may reflect the administration of relatively high doses of exogenous insulin to achieve adequate metabolic control. However, elevated insulin levels should not necessarily be attributed solely to treatment, as poor glycemic control may also be accompanied by hepatic dysfunction and increased liver transaminase concentrations in children and adolescents with T1D.⁵⁰

The increased WBC count may reflect hyperglycemia-associated oxidative stress, which can promote pro-inflammatory cytokine production, activate inflammatory signaling pathways, and stimulate immune-cell recruitment.⁵¹ Anemia is also frequently observed in individuals with diabetes and has a multifactorial pathophysiology. One proposed mechanism is reduced erythropoietin production associated with renal microvascular complications, together with sympathetic denervation of the kidneys in the presence of autonomic neuropathy.⁵²

The seroprevalence of *H. pylori* IgG antibodies was significantly higher among diabetic children than among their non-diabetic counterparts (40.0% vs. 21.7%), representing an approximately 1.8-fold higher prevalence in the T1D group. This finding supports a possible

association between *H. pylori* infection and T1D and is consistent with previous research.⁵³ The increased prevalence may be related to greater *H. pylori* colonization in the setting of gastritis and suboptimal glycemic control among diabetic children. Nevertheless, previous studies have reported substantially different prevalence rates, ranging from 11.4% to 60.0% in children with T1D.^{14,33} Such variation may reflect differences in participant characteristics, age distribution, sample size, geographic setting, and diagnostic methods used to identify *H. pylori*.³⁴ Among children with T1D, those who were seropositive for *H. pylori* had significantly higher mean HbA1c, glucose, and insulin concentrations than seronegative participants. These findings suggest a potential interaction between *H. pylori* infection and glycemic regulation and are consistent with previous observations in children and adolescents with T1D.^{53,54} Poor glycemic control, reflected by elevated HbA1c, may increase insulin requirements, potentially explaining the higher insulin concentrations observed in infected participants. A significant positive association between *H. pylori* infection, insulin requirements, and HbA1c has previously been reported among children and adolescents with T1D.⁵³ Cytokines involved in *H. pylori*-associated gastric inflammation may contribute to these metabolic effects by impairing insulin secretion, increasing insulin resistance or anti-insulin activity, and altering carbohydrate metabolism.⁵⁵ Collectively, these mechanisms may contribute to impaired glycemic control and increased insulin requirements in infected individuals.⁵³ Although no significant differences in lipid parameters were observed between diabetic and non-diabetic children overall, diabetic children with *H. pylori* infection had significantly higher cholesterol, triglyceride, and LDL-C levels and lower HDL-C concentrations than their seronegative counterparts. These findings raise the possibility that *H. pylori* infection may contribute to dyslipidemia in children with T1D and are consistent with previous observations in diabetic patients.^{56,57} Several mechanisms may explain this association. *H. pylori*-induced cytokine production may promote endothelial dysfunction and insulin resistance while increasing hepatic gluconeogenesis and triglyceride synthesis.⁵⁸ In addition, inflammatory processes may inhibit lipoprotein lipase activity, promoting lipid mobilization, increasing circulating triglycerides, and reducing HDL-C concentrations.⁵⁹ *H. pylori*-associated inflammation may also enhance lipid peroxidation.⁶⁰ Collectively, these alterations could increase the long-term risk of atherosclerotic cardiovascular disease.

ALT activity was significantly higher in *H. pylori*-seropositive children with T1D than in seronegative diabetic children, suggesting a possible relationship between *H. pylori* infection and hepatic involvement. Previous evidence has identified *H. pylori* infection as a

potential risk factor for liver disease, with increased ALT activity serving as a biomarker of hepatic injury and hepatotoxicity.⁶¹ However, further studies are needed to clarify whether this association represents a direct effect of infection or is mediated through metabolic and inflammatory pathways. Hematological assessment showed significantly higher WBC counts and lower RBC counts and hemoglobin concentrations among *H. pylori*-seropositive diabetic children compared with seronegative participants. Evidence regarding the relationship between *H. pylori* infection and hematological abnormalities specifically in children with T1D remains scarce, highlighting the need for further investigation. Nevertheless, leukocytosis has been reported in patients with type 2 diabetes and concurrent *H. pylori* infection.^{58,62} Increased WBC counts may result from enhanced production of inflammatory cytokines by gastric epithelial cells in response to *H. pylori*.⁶³ Furthermore, *H. pylori* infection has been associated with reduced hemoglobin and RBC counts as well as vitamin B12 and iron deficiencies.⁶⁴ These findings suggest that *H. pylori* infection may aggravate the anemia observed in children with T1D. Prospective studies are warranted to determine whether successful eradication of *H. pylori* can improve metabolic and hematological abnormalities in this population.

This study has limitations, including the relatively small number of patients who regularly attended the diabetes clinics and the refusal of many participants to provide blood samples. Future research should use more accurate and reliable diagnostic methods to detect active *H. pylori* infection, such as the ¹³C-urea breath test, stool antigen testing, or endoscopic biopsy-based diagnostic techniques.

6. Conclusion

Neuropathy and gastritis were significantly more prevalent among diabetic children than among non-diabetic children. Diabetic children also exhibited significantly higher levels of HbA1c, glucose, insulin, AST, ALT, and WBC count, together with lower RBC count and hemoglobin levels, compared with their non-diabetic peers. The seroprevalence of *H. pylori* was significantly higher among diabetic children than among non-diabetic children. Within the diabetic group, *H. pylori* seropositivity was significantly associated with higher levels of HbA1c, glucose, insulin, cholesterol, triglycerides, LDL-C, ALT, and WBC count, as well as lower levels of HDL-C, RBC count, and hemoglobin. These findings indicate an association between *H. pylori* seropositivity and the clinical and biochemical characteristics observed in diabetic children; however, the cross-sectional design does not permit conclusions regarding causality or temporal relationships. Further longitudinal studies are warranted to clarify the nature and clinical significance of these associations.

Research Highlights

What Is Already Known?

H. pylori infection is reported more often in children with type 1 diabetes than in healthy peers. It is linked to chronic gastric inflammation, possible nutrient malabsorption, and subtle metabolic changes. Some studies associate it with poorer glycemic control and a higher risk of anemia, adding to the clinical burden in these children.

What Does This Study Add?

Diabetic children showed higher rates of neuropathy, gastritis, *H. pylori* infection, and elevated metabolic and inflammatory markers, including HbA1c, glucose, insulin, AST, ALT, and WBCs, along with lower RBCs and hemoglobin. Among diabetics, *H. pylori*-seropositive children had worse metabolic profiles—higher lipids and liver enzymes—and reduced HDL-C, RBCs, and hemoglobin.

Author Contributions

M.M.Y. conceived and designed the study, supervised the project, and contributed to drafting the original manuscript. S.N.M. and S.S.G. contributed to the interpretation of the results and critically reviewed and revised the manuscript. E.S.SH. conducted the primary sample collection and biochemical analyses and contributed to data curation. M.M.L. contributed to drafting the original manuscript and to the interpretation of the results, as well as reviewing and editing the manuscript. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest Disclosures

All authors declared that they have no conflict of interest.

Ethical Approval

An official letter of request (No. 3622/15) was sent from the Palestinian Ministry of Health to Medical Center Administrations in different Gaza hospitals to facilitate the conduction of the study. The necessary approval to conduct the study was obtained from Helsinki Committee with the ethical approval number PHRC/HC/84/16 in the Palestinian Territories. The participants were given a full explanation about the purpose of the study and assurance about the confidentiality of the information and the participation was optional. Besides that, written informed consent was freely signed by each participant prior to inclusion in the study.

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