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# Type 1 Diabetes and *Helicobacter pylori* in 10-Years-Old Girl: Clinical Case Report

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## Abstract

**Background:** Diabetes is one of the most widespread metabolic diseases worldwide and is responsible for approximately 4 million deaths per year in 2010. The global prevalence among adults was estimated at 4.6% affecting around 285 million people. This number rose to 371 million in 2012 and is projected to reach 552 million by 2030. Among the various factors that may influence the incidence of diabetes, infection with *Helicobacter pylori* has been increasingly studied for its potential role. This bacterium, well-known for causing gastritis and ulcers, may also impact metabolism and blood glucose regulation. The role of *Helicobacter pylori* infection in the development of gastroduodenal diseases is well established. Over the past two decades, the literature has suggested a possible involvement of the bacterium in extra-digestive diseases. Several studies have reported a higher prevalence of *Helicobacter pylori* infection in diabetic patients, with or without dyspeptic symptoms, and a positive association with insulin resistance. However, the pathogenesis of diabetes mellitus is multifactorial, making it challenging to determine the specific role of each contributing factor. **Methods:** This is a descriptive observational descriptive study of a special case discovered during a pediatric consultation at the hospital saint joseph of Kinshasa. Its presentation as a case not yet encountered at the Saint Joseph Hospital makes its publication valuable. **Result:** It was a 10-year-old girl who was brought in by her parents for epigastric burning pain occurring long after meals and sometimes relieved by drinking large amounts of cold water associated with episode of nausea, belching and acid regurgitation. Physical examination revealed a polyuria-polydipsia syndrome and blood tests showed; normal complete blood count (CBC), random blood glucose; 382 mg/dl, Glycated hemoglobin 12.5%, urine dipstick; ketonuria +++, glycosuria +++, no nitrite, no leukocytes, no protein, blood gas analysis: pH 7.39 and $HCO_3^-$: 21.3 mmol/l. Diabetes immunological markers were positive (anti-GAD et IA2) and *Helicobacter pylori* antigen test was also positive. **Con-**

**clusion:** Diabetic The clinical case described is an entity that has been rarely studied in children and remains a major subject of research in other regions. Some studies have concluded that there is a high prevalence of *Helicobacter pylori* infection in children and the authors also observed a positive correlation between the infection and HbA1C levels suggesting a potential impact on glycemic control as described by Bazmamoun *et al.* in 2026.

## Subject Areas

Diabetes & Endocrinology, Pediatrics

## Keywords

Type 1 Diabetes, Helicobacter Infection

## 1. Introduction

Diabetes is the most widespread metabolic disease in the world resulting from the progressive autoimmune destruction of the beta cells in the islets of Langerhans of the pancreas which are responsible for insulin production [1].

Its global incidence is increasing by 2% to 5% per year, particularly among those under the age of 15. This rapid rise is attributed to changes in environmental factors. Around 9 million children worldwide live with type 1 diabetes. Nordic countries such as Finland and Sweden have the highest incidence rates, exceeding 40 cases per 100,000 per year [2].

In Asia, incidence rates are very low—less than 1 case per 100,000 per year, but they are gradually increasing. In France, between 20,000 and 25,000 children and over 300,000 adults are affected with an incidence of 15 to 20 new cases per 100,000 children per year. One factor that may influence the incidence of diabetes is *Helicobacter pylori* infection. The role of *H. pylori* infection in the development of gastroduodenal diseases is well established [3].

*H. pylori* infects approximately 50% of the global population, with a higher prevalence in developing countries where it can reach 80 to 90% in sub-Saharan Africa. The prevalence of infection in children is particularly high with rates ranging from 40 to 80% depending on the region [4].

In Kinshasa, a recent study reported a high prevalence of 63.4% among children under the age of 12 [3] [4].

The infection is primarily acquired during childhood with predominant intrafamilial transmission. Risk factors include low socio-economic statuts, poor hygiene, overcrowding and limited access to clean water [5].

Over the past two decades, the scientific literature has suggested a potential involvement of *Helicobacter pylori* in extra-digestive manifestations, particularly autoimmune disorders through mechanisms:

- **Chronic inflammation and insulin resistance**: *H. pylori* infection induces chronic gastric inflammation accompanied by the systemic release of pro-in-

flammatory cytokines such as interleukin-6, tumor necrosis factor-alpha (TNF-α) and C-reactive protein (CRP). These circulating inflammatory mediators interfere with insulin signaling in peripheral tissues (liver, skeletal muscle, adipose tissue) notably by inhibiting insulin receptor phosphorylation which reduces glucose uptake and promotes insulin resistance. This mechanism plays a central role in type 2 diabetes and may also worsen glycemic control in patients with type 1 diabetes [6].

- **Disruption of gastrointestinal hormone regulation**: *H. pylori* infection alters the secretion of key gastric hormones involved in metabolic regulation; ghrelin, an orexigenic hormone produced in the stomach involved in appetite stimulation and insulin secretion levels are often reduced during infection; leptin involved in energy homeostasis and insulin sensitivity, modulated by inflammation, Gastrin and somatostatin modified in the context of chronic gastritis, which may affect gastric emptying and postprandial insulin responses. This hormonal imbalance may contribute to glucose dysregulation and appetite disturbances in diabetic patients.
- **Molecular mimicry and autoimmunity**: *H. pylori* expresses antigens that share structural similarities with human proteins, particularly those found in pancreatic beta-cell. This molecular mimicry can trigger cross-reactive autoimmune responses. The immune system in targeting *H. pylori* may also attack pancreatic beta-cell, promoting or worsening type 1 diabetes. Bacterial antigens such as CagA have been implicated in abnormal activation of autoreactive CD4+T lymphocytes, potentially increasing the production of anti-GAD and anti-IA2 antibodies [7].
- **Oxidative stress and mitochondrial dysfunction**: *H. pylori* infection generates an excess of reactive oxygen species (ROS) leading to oxidative stress. This environment disrupts normal cellular metabolism and may damage pancreatic beta-cells, resulting in impaired insulin secretion or even apoptosis [8].

The relationship between diabetes mellitus and *H. pylori* was introduced in 1989. *H. pylori* may promote insulin resistance by inducing chronic inflammation and affecting the regulation of insulin as well as gastrointestinal hormones. Gastritis caused by *H. pylori* can alter the secretion of gastric hormones such as leptin, growth hormone-releasing hormone, gastrin, and somatostatin which may influence susceptibility to diabetes [9].

It has been suggested that *H. pylori* may contribute to the incidence of cardiovascular diseases and diabetes through the elevation of pro-inflammatory cytokines such as C-reactive protein and interleukine-6 [10] [11]. In general, several meta-analyses have examined the role of *H. pylori* in the pathogenesis of diabetes mellitus and its complications. Some case-control studies have reported a higher prevalence of *Helicobacter pylori* infection in diabetic patients [12].

A meta-analysis by Zhou *et al.* (2019) including 28 studies revealed an increased risk of *H. pylori* infection in patients with type 1 diabetes compared to controls (Odds ratio = 1.72). However, infection rates vary according to geographic re-

gions, age, and socio-economic conditions.

Another meta-analys conducted by Wang Zheng *et al*. (2016) showed a correlation between *Helicobacter pylori* infection and the onset of both type 1 and type 2 diabetes. Further research has demonstrated that inflammation and type 2 diabetes may be closely linked and that the chronic inflammatory response caused by the infection can lead to insulin resistance and destruction of the islets of Langerhans through apoptotic mechanisms due to molecular mimicry [13].

Research conducted over nearly 40 years has shown that this bacterium is associated with the natural history of many diseases. Epidemiological data indicate an increased incidence of autoimmune diseases with or following infection by specific microorganisms [14].

*H. pylori* is a potential trigger of gastric autoimmunity and may be associated with other autoimmune diseases whether congenital or acquired [15].

Despite numerous studies, no clear consensus has emerged regarding the relationship between *H. pylori* infection and diabetes. The evidence suggests a modest but inconsistent association in type 2 diabetes, no convincing proof of causality with the relationship remaining hypothetical, and a possible role as an inflammatory cofactor in an unfavourable metabolic context. In type 1 diabetes, the data are too limited to draw firm conclusions, although some immunological arguments have been proposed [16].

In children and adolescents with type 1 diabetes, data are scarce and often contradictory: some studies suggest an increased prevalence of *H. pylori* infection in children with type 1 diabetes possibly related to impaired immunity or dysbiosis; other research, particularly in Europe, shows no significant difference compared to healthy controls, and the role of autoimmunity in type 1 diabetes further complicated interpretation with some authors proposing a possible cross-reactive immune interaction between *H. pylori* antigens and pancreatic b-cells [17].

## 2. Clinical Observation

It was a 10-year-old girl of African origin brought in by her parents for burning epigastric pain associated with reduced food intake and significant weight loss over the past month.

In her personal history, it is noted that she is the second of four siblings, and she has never been hospitalized, with no history of urinary tract infections or pneumonia. Her vaccination schedule has been properly followed. She attends school and maintains a good average grade of 15 out of 20.

Family history revealed primarily a background of autoimmunity: type 1 diabetes in the father (treated with insulin), and type 2 diabetes in the grandfather (treated with metformin and insulin therapy). There is also hyperthyroidism due to grave's disease in her paternal aunt, treated with propanol and neomercazol. There is no congenital adrenal hyperplasia in the siblings, no hypothyroidism and celiac disease.

The history of current illness dated back about four weeks before our consultation characterized by reccurent and intermittent epigastric pain described as

burning without periodicity radiating to the left axillary region and the back worsening and occurring away from meals associated with nausea, belching and reduced food intake.

As additional history, the mother reports nighttime crying episodes related to burning epigastric sensations with acid reflux relieved by drinking large amounts of cold water. There were also multiple nighttime awakenings to drink and urinate averaging 5 to 6 times per night mimicking a polyuria-polydipsia syndrome. There was no fever episode, no urinary symptoms, no ENT signs and pulmonary signs such as painful swallowing or cold symptoms

On physical examination, this is a young girl with anthropometric parameters within normal ranges except for a weight loss calculated at 5%. Her general condition was impaired due to weight loss and a suffering (ill) appearance. The analog pain scale was rated at 5/10.

There were no localized or disseminated lymphadenopathies and no ulcerative stomatitis-type lesions. The cardiovascular and pulmonary examinations were unremarkable; her abdominal was soft and depressible and the girl described epigastric burning pain with posterior radiation extending from the epigastrium and left axillary region to the back not relieved by forward bending but sometimes alleviated by drinking large amounts of cold water, she experienced episodes of belching in the form of gastric regurgitation, no palpable hepatosplenomegaly or abdominal mass suggestive of an abdominal tumor.

In terms of hydration, the oral mucosa was dry and the girl had periorbital dark circles, a skin fold sign (skin pinch) that resolved slowly and intense thirst.

Her nutritional statuts and neurological examination were unremarkable.

In biology, we recorded in Table 1.

Table 1. showing the biological results.

| Biology | |
|---|---|
| Complete blood count (CBC) | Normal |
| C-reactive protein (CRP) | 8 mg/l |
| Casual blood glucose | 382 mg/dl |
| Glycated hemoglobin (HbA1c) | 12.5 % |
| Urine dipstick | Acétonuria +++, Glycosuria +++, no nitrites, no leukocyturia, no protein |
| Blood electrolyte panel (or blood ionogram) | $Na^+$: 139 mmol/l, $K^+$: 3.9 mmol/l, calcium total 2.2 mmol/l |
| Blood gas analysis (or aterial blood gases) | pH: 7.39; $Po_2$: 43, $HCO_3^-$ (RA): 21.3 mmol/l, ionized calcium: 1.13 mmol/l |
| Immunological markers of diabetes | Anti-GAD and IA2 antibodies positive (results obtained two weeks later) |

The search for immunological markers was obtained two later due to lack of resources, and the antigen test for Helicobacter antigen came back positive (See Figure 1).

**Figure 1.** Antigenic test of *Helicobacter pylori*.

In view of the above results, the young girl was hospitalized for newly diagnosed diabetes in the context of a *Helicobacter pylori* infection. She then received therapeutic education on diabetes, digestive rest, parenteral rehydration and an *H. pylori* eradication treatment using triple therapy based on amoxicillin (500 mg morning and evening), clarithromycin (250 mg morning and evening) and a proton pump inhibitor (omeprazol 20 mg morning and evening) for 14 days.

A follow-up stool antigen test was performed after the treatment, and it came back negative.

## 3. Discussion

*Helicobacter pylori* is one the most common human bacterial pathogens affecting about 50% of the world's population. The infection causes a wide range of gastric disorders including simple gastritis, gastroduodenal ulcers, and malignant gastric tumor. Several studies have shown a high frequency of *Helicobacter pylori* infection in the diabetic population. A study conducted by Elsheikh *et al*. on 102 patients found a prevalence of 52.9% of *H. pylori* infection in type 1 diabetic population [18].

A meta-analysis namely that of Yi-Ning based on 21 relevant publications was identified. A meta-analysis of 11 studies involving 513 patients with diabetes mellitus showed lower Glycated hemoglobin (HbA1C) levels in *H. pylori* negative patients compared to *H. pylori*-positive diabetic patients (WMD = 0.43, IC 95%: 0.05 - 0.64; P = 0.02) [19].

This is the result of the Glycated hemoglobin (HbA1C) found in our little girl which was elevated at 12.5%.

The chronic inflammation of the gastric mucosa induced by tumor necrosis factor (TNF), interleukin 6 and CRP through the action of *H. pylori* exerts a paracrine effect on the activation of serine kinase, C-JUN N terminal kinase and nuclear factor Kappa B inhibitor kinase which phosphorylates the insulin receptor substrate leading to insulin resistance in adipose tissue and subsequently apoptosis of islet cell mass through the action of pro-apoptic proteins and caspace 9 and 3 resulting in insulin deficiency [20].

In the context of type 1 diabetes which is characterized by absolute insulinopenia, the hyperglycemia induced by this deficiency is responsible for weight loss

due to excessive lipolysis. The polyuria-polydipsia syndrome results from hyperglycemia and glycosuria which by exceeding the renal threshold for glucose leads to polyuria, the stimulation of the thirst center through the release of ADH which is responsible for polydipsia [21].

Regarding our little girl, the clinical picture noted weight loss and polyuria-polydipsia syndrome compatible with the cardinal signs of diabetes. The epigastric pain resembling heart burns could indicate gastric involvement due to bacteria [22].

## 4. Conclusions

Type 1 Diabetes and *Helicobacter pylori*: The clinical case described is an entity that has been rarely studied in children and remains a major subject of research in other regions. Some studies have concluded that there is a high prevalence of *Helicobacter pylori* infection in children and the authors also observed a positive correlation between the infection and HbA1C levels suggesting a potential impact on glycemic control as described by Bazmamoun *et al*. in 2016.

Perspectives: Strengthening targeted screening and the systematic inclusion of *H. pylori* screening in diabetic children, especially in the presence of digestive symptoms, could allow for more comprehensive management and improve glycemic control.

Large-Scale Conductor Studies: It is necessary to carry out multicenter epidemiological studies with well-defined pediatric cohorts to confirm the association between *H. pylori* and type 1 diabetes and to better understand the true prevalence of this confection in developing countries.

## Authors' Contributions

Merlin Wawa initiated the work, while the other authors reviewed it, provided corrections and amendments, and approved the final version.

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## Conflicts of Interest

The authors declare no conflicts of interest.

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