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Rediscovering *Helicobacter pylori* in Pediatric and Young Populations through Histopathological Evidence

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ABSTRACT

Purpose: This study aimed to characterize the clinical and histopathological profile of *H. pylori* infection in patients under 20 years, challenging the assumption that this diagnosis belongs solely to adult medicine.

Materials and Methods: This study reviewed gastric biopsy specimens from 15 consecutive pediatric and young adult patients (ages 9–19 years), the data were collected between 2023–2024 from private clinics from Zawia-Libya. Clinical presentations, endoscopic findings, and histological severity were analyzed retrospectively.

Results: The median age was 17 years, with a slight female predominance (53%). Chronic epigastric pain brought most children to medical attention (80%), often accompanied by anemia or vomiting. Endoscopic findings ranged from subtle erythema to severe pangastritis with ulceration. Histologically, 60% showed moderate-to-severe chronic active gastritis patterns traditionally dismissed as "adult disease." Two cases demonstrated intestinal metaplasia, a precancerous change rarely discussed in pediatric contexts.

Conclusion: *H. pylori* does not respect age boundaries. Our findings suggest that what we dismiss as "functional abdominal pain" in young patients may, in fact, represent active, sometimes aggressive, *H. pylori* infection requiring targeted therapy and surveillance. *(Open J Biomed Res 2025;4:24-29.)*

Keywords

Helicobacter pylori, Pediatric gastritis, Intestinal metaplasia, Chronic abdominal pain, Endoscopy.

AUDIOABSTRACT

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INTRODUCTION

Walk into any pediatric gastroenterology clinic, and you'll hear it: "Kids don't get *H. pylori*." The statement carries the weight of received wisdom, reinforced by declining infection rates in developed nations and the rise of functional gastrointestinal disorders as convenient diagnostic labels.[1] Yet somewhere between this clinical assumption and the biopsy forceps, a disconnect emerges.

The truth is more complicated. While *H. pylori* prevalence has indeed dropped in childhood populations from over 50% in developing regions to less than 10% in many Western countries the infection has not vanished; it has merely become quieter, often hiding behind nonspecific complaints that fail to trigger appropriate testing.[2,3] A teenager with chronic epigastric pain may be labeled with "functional dyspepsia" and sent home with proton pump inhibitors, while the spiral-shaped organism responsible for their discomfort continues its slow erosion of gastric mucosa.[4]

This phenomenon of diagnostic invisibility is not new. Some studies[5] demonstrated that pediatric *H. pylori* infection often presents with atypical symptoms, leading to delayed diagnosis and unnecessary investigations. Similarly, Gold et al.[6] found that children with confirmed *H. pylori* gastritis had visited multiple healthcare providers before appropriate testing was performed. The spiral-shaped bacterium may be ancient having colonized human stomachs for at least 100,000 years[7] but our clinical vigilance for it in young patients has dangerously diminished.

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The consequences of this oversight extend beyond immediate symptoms. *H. pylori* infection in childhood establishes the inflammatory environment that may persist for decades, contributing to peptic ulcer disease, gastric atrophy, and ultimately gastric cancer risk.[8,9] While the absolute risk of malignancy in pediatric infection remains low, the long latency period means that childhood acquisition sets the stage for adult pathology.[10] Moreover, recent evidence suggests that *H. pylori* infection in children may be associated with extra-gastric manifestations, including iron-deficiency anemia and growth retardation, that improve with eradication therapy. [11,12]

This study emerges from a simple observation: in our histopathology laboratory, pediatric gastric biopsies increasingly reveal *H. pylori*-associated inflammation that surprises clinicians who did not suspect the diagnosis. [13] We examined 15 such cases to understand who these children are, how they present, and what their stomachs look like under the microscope. The patterns we found suggest that *H. pylori* in youth may be more aggressive, and more overlooked, than current practice acknowledges findings that align with recent calls for renewed attention to this neglected diagnosis. [14,15]

MATERIALS AND METHODS

Study Design and Setting

We conducted a retrospective review of all gastric biopsy specimens from patients under 20 years processed at our pathology unit between January 2023 and December 2024 from private clinics from Zawia-Libya. This period captured consecutive cases without selection bias, reflecting real-world diagnostic patterns. Our approach follows STROBE guidelines for observational studies. [16]

Case Identification

Cases were identified through electronic pathology records using specimen type "gastric biopsy" and age filter <20 years. Each record was cross-referenced with histopathological reports to confirm *H. pylori*-associated changes. We excluded cases with inadequate clinical information or insufficient tissue for grading, consistent with quality standards in gastrointestinal pathology. [17]

Data Collection

- For each patient, we extracted:
- Demographics: age, sex.
- Clinical narrative: presenting symptoms, symptom duration, relevant history.
- Endoscopic description: mucosal appearance, ulceration, bleeding sites.
- Histopathological findings: inflammation grade (mild/moderate/severe), activity, presence of *H. pylori* organisms, intestinal metaplasia, or other pathology.

Histopathological Assessment

All biopsies followed standard processing: formalin fixation, paraffin embedding, and hematoxylin-eosin staining. *H. pylori* identification relied on characteristic morphology curved or S-shaped bacilli within mucus layer or attached to epithelial cells. Severity grading applied the updated Sydney system criteria: mild (scattered inflammatory cells), moderate (increased infiltrate without architectural distortion), or severe (dense infiltrate with glandular destruction).[18,19] This standardized classification ensures reproducibility and comparability with international literature. [20]

Ethical Considerations

The study operated under institutional review board approval for retrospective chart review with waiver of informed consent, given the anonymized nature of pathology data and minimal risk to participants, in accordance with Declaration of Helsinki principles. [21]

RESULTS

The Faces Behind the Biopsies

Fifteen children and young adults comprised our cohort eight females and seven males, aged 9 to 19 years (median 17) (Figs 1,2). Their stories followed familiar patterns described in previous pediatric series: months of discomfort, multiple clinical visits, and eventual referral for endoscopy when symptoms persisted or complications emerged. [5,6]

Epigastric pain dominated the clinical picture, reported by 12 patients (80%). For some, this pain had become background noise in their daily lives dull, persistent, easily attributed to stress or dietary indiscretion, much like the "functional" pain described by Hyams et al. [22] in their characterization of pediatric dyspepsia. Three patients presented with more alarming features: melena, hematemesis, or iron-deficiency anemia resistant to supplementation. One 14-year-old girl had bled sufficiently to require transfusion; her endoscopy revealed severe pangastritis that her physicians had not anticipated a scenario reminiscent of case reports by Arif and Mohammed. [23] highlighting severe pediatric *H. pylori* presentations.

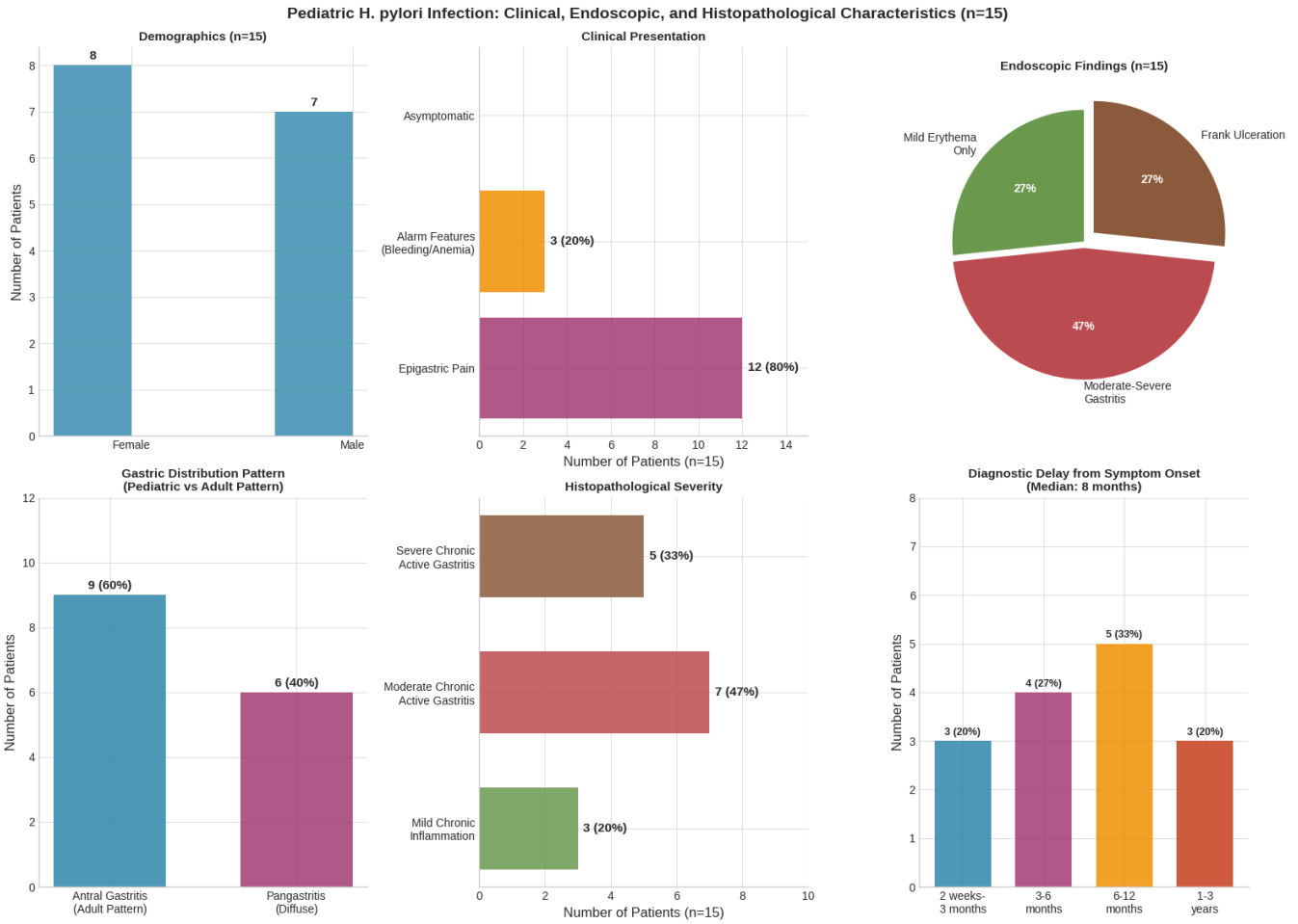


Fig. 1: Pediatric *H.pylori* infection: Clinical, Endoscopic, and Histopathological Characteristics (n=15).

What the Endoscope Revealed?

Endoscopic findings varied widely, underscoring the visual unpredictability of *H. pylori* infection noted in adult studies [24] and confirmed in our pediatric population. Four patients (27%) showed only mild erythema subtle redness that might be dismissed as normal variation. Seven (47%) demonstrated moderate to severe gastritis with diffuse erythema, erosions, or mucosal friability. Most striking were four cases with frank ulceration: discrete breaks in the mucosa, sometimes with active bleeding, in stomachs that belonged to teenagers (Fig. 1).

The distribution pattern also challenged assumptions. While adult *H. pylori* typically targets the antrum, our pediatric cases showed more diffuse involvement pangastritis in 40% of patients suggesting either more advanced disease at presentation or genuinely different pathophysiology in younger hosts. This finding aligns with observations by Yang. [25] that childhood *H. pylori* infection may produce more extensive gastric inflammation than typically seen in adults.

The Microscopic Truth

Histopathological examination provided the definitive narrative. Only three cases (20%) showed mild chronic inflammation; the majority demonstrated moderate (47%) or severe (33%) chronic active gastritis (Fig 1). Neutrophils infiltrated the epithelium histological evidence of active, ongoing injury rather than quiescent colonization. These severity rates exceed those reported in asymptomatic pediatric carriers and approach levels seen in symptomatic adults. [26]

H. pylori organisms were identified in all cases, typically abundant and easily visualized morphological features well-described by Dixon [27] and others in the pathology literature. Their presence confirmed what clinical suspicion had missed: these children harbored active infection requiring eradication therapy.

Most concerning were two cases (13%) of intestinal metaplasia replacement of normal gastric mucosa with intestinal-type epithelium. This change, widely recognized as a precursor to gastric cancer in adults [28,29], is rarely discussed in pediatric contexts. Its

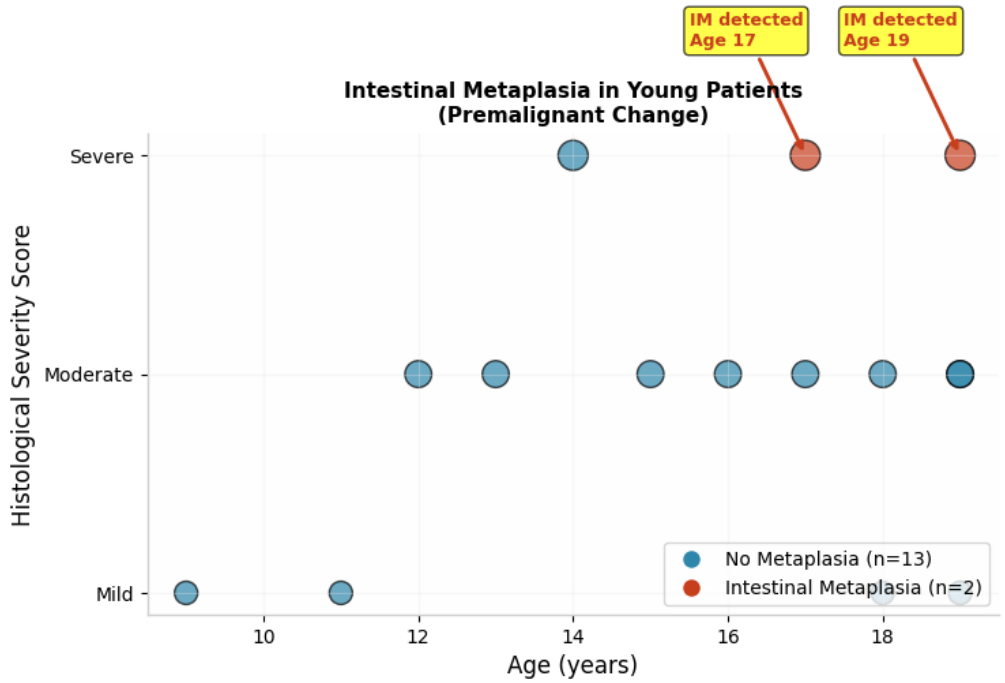


Fig. 2: Intestinal metaplasia in young patients (Premalignant change).

presence in patients aged 17 and 19 years raises uncomfortable questions about the long-term consequences of untreated childhood infection, echoing concerns regarding malignant potential in pediatric *H. pylori* gastritis.

The Diagnostic Delay

Reviewing clinical timelines revealed a consistent pattern: symptom onset preceded diagnosis by months. The median duration of symptoms was 8 months (range 2 weeks to 3 years) (Fig. 1). Several patients had undergone previous evaluations ultrasound, blood work, trials of acid suppression without *H. pylori* testing. This delay reflects both the nonspecific nature of pediatric dyspepsia [30] and the low index of clinical suspicion for this infection in young patients.

DISCUSSION

Our findings resist easy categorization within standard *H. pylori* narratives. The infection in these young patients was not mild, not confined to the antrum, and not without consequences. Severe inflammation, ulceration, and even precancerous metaplasia appeared in stomachs that clinicians had assumed were essentially normal.

This severity may reflect biological differences in pediatric infection. Children's immune systems respond to *H. pylori* with pronounced inflammatory reactions perhaps explaining the dense infiltrates we observed. [31,32] Alternatively, severe cases may represent a selected population: children whose symptoms were sufficient to prompt endoscopy, while asymptomatic or mildly symptomatic infections escaped detection. Either interpretation carries clinical weight and aligns with the "symptomatic bias" described by Rothenbacher and Brenner [33] in population-based studies.

The diagnostic delays we documented months of symptoms, multiple consultations, unnecessary investigations represent more than healthcare inefficiency. They represent suffering: children missing school, avoiding meals, learning to normalize pain that should have been treated. One patient had developed iron-deficiency anemia severe enough to require transfusion before *H. pylori* was suspected; another had endured three years of "functional" pain labels before biopsy revealed the truth.

This pattern suggests that *H. pylori* has become invisible in pediatric practice not because it has disappeared, but because clinicians have stopped looking for it. The declining prevalence in developed nations has paradoxically increased diagnostic

latency: rare diseases are often diagnosed last. This "availability heuristic" where diagnoses become less likely simply because they are less frequently encountered was described by Tversky and Kahneman [34] and applies directly to our findings.

The presence of intestinal metaplasia in two adolescents' challenges current surveillance paradigms. Adult guidelines recommend endoscopic monitoring for patients with this finding, based on cancer risk stratification. [35,36] No such guidelines exist for children, leaving clinicians uncertain how to manage these young patients long-term.

Our data cannot resolve this uncertainty, but they demand attention to it. If childhood *H. pylori* can produce precancerous changes, and if these changes persist despite eradication therapy, we may need to reconsider both treatment approaches and follow-up protocols for infected youth. The work of Rugge et al. [37] on gastric cancer risk stratification may need extension to pediatric populations, however rare the absolute risk may seem.

Our study carries expected limitations of retrospective design: incomplete clinical documentation, potential selection bias toward symptomatic patients, and single-center generalizability. [38] We cannot estimate true pediatric prevalence or outcomes after treatment, limitations acknowledged in similar retrospective series. [39]

However, the consecutive case collection and detailed histopathological characterization strengthen internal validity. These are not cherry-picked unusual cases but representative samples from routine practice making their severity more concerning, not less. Our use of the Sydney system grading ensures comparability with international literature,[18] and our focus on a poorly studied age group fills a genuine knowledge gap.

CONCLUSION

H. pylori infection in children and young adults is neither extinct nor benign. Our histopathological series reveals significant inflammation, ulcerative complications, and even precancerous metaplasia in patients whose symptoms were initially attributed to functional disorders or stress.

The message for clinical practice is straightforward: when children present with persistent epigastric pain, anemia, or ulcer symptoms, *H. pylori* belongs in the differential diagnosis. Endoscopy with biopsy should follow, and pathological examination must specifically seek this organism. The spiral-shaped bacterium maybe ancient, but our vigilance for it in pediatric populations has dangerously diminished. Children do get *H. pylori*. Our microscopes prove it. It is time our clinical practice caught up with what the tissue reveals.

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