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ORIGINAL RESEARCH

Possible Role of *Helicobacter pylori* in Ear Nose and Throat Diseases

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Background: *Helicobacter pylori* is assumed to cause many gastric and extragastric diseases. We aimed to assess the possible association role of *H. pylori* in Otitis media with effusion (OME), nasal polyps and adenotonsillitis.

Patients and Methods: A total of 186 patients with various ear, nose and throat diseases were included. The study comprised 78 children with chronic adenotonsillitis, 43 children with nasal polyps and 65 children with OME. OME patients were assigned to two subgroups: those who have and those who did not have adenoid hyperplasia. Among the patients with bilateral nasal polyps, 20 individuals had recurrent nasal polyps and 23 had de novo nasal polyps. Patients who have chronic adenotonsillitis were divided into three groups: those with chronic tonsillitis and those who underwent tonsillitis, those with chronic adenoiditis and adenoidectomy was performed, and those with chronic adenotonsillitis and underwent adenotonsillectomy. In addition to examination of *H. pylori* antigen in stool samples of all included patients, real-time polymerase chain reaction (RT-PCR) for detection of *H. pylori* in the effusion fluid was performed, additionally, Giemsa stain was used for detection of *H. pylori* organism within the tissue samples when available.

Results: Frequency of *H. pylori* in effusion fluid was 28.6% in patients with OME and adenoid hyperplasia, while in those with OME it was only 17.4% with a p value of 0.2. Nasal polyp biopsies were positive in 13% patients of denovo, and 30% patients with recurrent nasal polyps, p=0.2. De novo nasal polyps were more prevalent in the positive stools than recurrent ones, p=0.7. All adenoid samples were negative for *H. pylori*, only two samples of tonsillar tissue (8.3%) were positive for *H. pylori*, and stool analysis was positive in 23 patients with chronic adenotonsillitis.

Conclusion: Lack of association between *Helicobacter pylori* and occurrence of OME, nasal polyposis or recurrent adenotonsillitis.

Keywords: *Helicobacter pylori*, otitis media with effusion, nasal polyps, adenotonsillitis

Introduction

Helicobacter pylori is a spiral-shaped Gram-negative microaerophilic bacillus. It is known to be one of the most harmful bacteria. It affects more than half of the entire world population. The prevalence of *H. pylori* infection is increasing with age; it reaches about 75% by the age of 10 years.[1] *H. pylori* is assumed to cause many gastric diseases such as chronic and acute gastritis, gastric ulcers, lymphoma and adenocarcinoma.[2–4] In addition to its gastrointestinal effects, it can cause head and neck disorders, atherosclerosis, lung, hepato-biliary, hematological, and intestinal diseases.[5] *H. pylori* is primarily found in the stomach, and numerous studies have linked it to disorders of the upper airways.[6–9] Patients with

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gastroesophageal reflux may develop OME due to damaging action of gastric acid on mucosal lining of Eustachian tube leading to oedema and Eustachian tube dysfunction.[10,11]

The initial *H. pylori* reservoir is the stomach, although *H. pylori* has also been found in tonsils, adenoids, dental plaque, and laryngeal squamous carcinomas.[12–14] Tonsil and adenoid tissues have *H. pylori* infection, according to Lin et al.[15] Numerous individuals with GERD also have *H. pylori* infection, exposing their nasal mucosa to contact with bacteria that can lead to sinonasal disorders and nasal polyps.[16,17] *H. pylori* was recently found in tonsils, adenoids, nasal polyp tissues, and middle ear fluid.[1]

There are numerous methods for detection of *H. pylori*; invasive and noninvasive methods. The first technique for diagnosing *H. pylori* was histology.[18] Noninvasive methods such as serology, urea breath test (UBT) and stool antigen tests. Invasive methods using biopsies for histological examination using stain like Giemsa, Warthin-Starry, acridine orange, Dieterle, *H. pylori* silver stain, Gimenez, McMullen, and immunostaining. Giemsa staining is frequently used to identify *H. pylori*. The haematoxylin and eosin stain aids in determining the degree of bacterial-induced inflammation. Giemsa stain is more practical for identifying *H. pylori* due to its ease of use, low cost, high sensitivity, and ease of preparation.[18,19] Sensitivity of Giemsa stain in the detection of *H. pylori* is 100%, while its specificity is 85.7%, which makes it better than H&E stains. In the study done by Kocsmár et al, Giemsa revealed an overall sensitivity of 83.3% and a specificity of 98.8%.[20,21] Also, PCR can be used for detection of *H. pylori* using samples from gastric juice, gastric biopsy, dental plaque, saliva, and stools. Moreover, it was reportedly used to find H. pylori in middle ear effusion collected.[22,23] We aimed to study the possible role of *H. pylori* in diseases of Ear, Nose and Throat such as: OME, bilateral nasal polyps and chronic adenotonsillitis.

Patients and Methods

This study, which was conducted on 186 patients hospitalized at the ENT Department of Qena University Hospitals, Egypt, between April 2020 and February 2022, is a prospective cohort study. 65 patients with OME, 43 individuals with bilateral nasal polyps, and 78 patients with chronic adenotonsillitis were included in the current study. The study was approved by the Institutional Review Board of Faculty of Medicine-South Valley University, Qena, Egypt, and the ethical approval code is 4/54/7/2020. The current study complies with the Declaration of Helsinki.

Informed Consent was obtained from patients, and parents of children participate in the study after explaining the objectives and steps of the research. A written consent was obtained from the patients or their guardians on tissue samples.

Full history taking was done including: ENT symptoms, upper gastrointestinal symptoms (epigastric pain, discomfort, dyspepsia, indigestion, heart burn, regurgitation, etc.) then clinical examination and full laboratory investigations were done.

Stool samples for all patients were collected to detect *H. pylori* antigen using rapid urease test.

The pathologists and clinical pathologists were not aware of the history of GERD and the results of the stool antigen test when evaluating many histopathological investigations and other laboratory testing.

Methodology of OME

Diagnosis of OME based on observation of its clinical signs using otoscopy and confirmed by type B tympanometry up to 3 months after treatment, absent acoustic reflex using tympanometry with evidence of average air bone gap greater than 20 dB using audiometry. For all patients, lateral neck radiography was performed in the extension position to confirm the diagnosis of adenoid hyperplasia. The patients were divided into two groups; the first group consisted of 42 OME patients who also had adenoid hyperplasia, and the second group consisted of 23 OME patients who did not. All patients had surgical myringotomy and tympanostomy tube placement, and samples of effusion were taken while maintaining sterilization. Those who have adenoid hyperplasia had adenoidectomy. Myringotomy incisions are produced at the antero-inferior quadrant of the ear drum after the outer ear canal has been sterilised with a 70% alcohol solution. Middle ear fluid samples were taken in sterile containers and then sent to the microbiology lab for RT-PCR. To check for *H. pylori* antigen, stool samples from every patient were collected utilizing the InstaTest, RapiCard, One-Step *H. pylori* Antigen.

DNA Extraction and Real Time-PCR After Thawing Samples

Using the DNA Extraction Kit, DNA has been extracted (Qiagen, Hilden, Germany). Following that, the amplification was carried out using the BioDetect *H. pylori* Kit (Qiagen, Hilden, Germany).

The mixed reaction included two oligonucleotides with primer-function for PCR with an HP-specific fluorescent-labeled DNA probe. This allowed for real-time reaction monitoring by observing the increase in fluorescence for each sample at the end of each PCR cycle. To ensure the success of the PCR reaction, the kit includes both a positive control related to the specific target of *Helicobacter pylori* and a negative amplification control. A negative outcome in the amplification of foreign gene indicates the presence of PCR inhibitors in the extraction of highly degraded DNA. The technique also provided the amplification of an exogenous gene as an internal control in each mixture to confirm the accurate extraction of the nucleic acid. The total mix (Total Mix), which contained the reagents and had previously been centrifuged (45 µL), was then added to each extracted DNA sample (5µL); this combination also contained the hot start polymerase enzyme, which can only be activated after denaturation at 95°C for 10 min. Real-Time PCR Thermal Cycler was used for the test. The initial amplification cycle consisted of one repetition in one step at a temperature of 95°C for 10 min. The second cycle was split into two phases, with the first step consisting of 50 repeats at 95°C for 6 min and 30s and the second step consisting of 1 min at 56°C for the acquisition of real-time fluorescence data. Two fluorophores, FAM fluorophore for HP positive and JOE/VIC fluorophore for amplification of internal control (and extraction), were used to obtain the sample's amplification traces. The thermal cycler programme was used to evaluate the data after that (Figure 1).

Methodology of Nasal Polyps, Tonsils and Adenoids

Forty-three patients with bilateral nasal polyps were included in this study; they were classified as 23 patients with de novo bilateral nasal polyps and 20 patients with recurrent bilateral nasal polyps. For each of these patients, a sinuscopy and biopsies were performed. When samples were being processed, they were placed in 10% formalin.

Methodology of Tonsils and Adenoids

This study included 78 patients presented with tonsillitis, adenoiditis or adenotonsillitis; 38 (48.7%) males and 40 (51.3%) females. From those patients 24 patients underwent tonsillectomy, 26 patients underwent adenoidectomy and 28 patients underwent adenotonsillectomy. Operations were conducted under general anesthesia. Biopsies were collected under sterile conditions, using direct punch forceps. Samples were put in 10% formalin till sample processing.

Sample Processing

Following the fixation of samples in 10% formalin, paraffin-embedded blocks were cut into thin sections and stained with hematoxylin and eosin to prepare them for microscopic analysis. Giemsa dye was used to detect *H. pylori* on a different slide. On dry, spotless microscopic glass, thin slices of the specimen were cut. The slides were hydrated with distilled

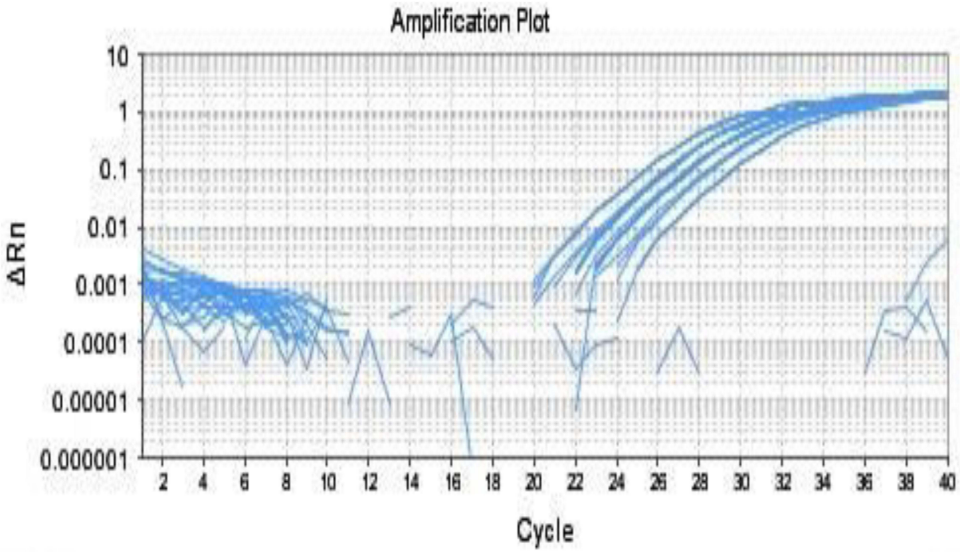

Figure 1 Representative picture for *H. pylori* amplification by RT-PCR. ΔRn: delta-normalized reporter (ratio of the fluorescence emission intensity of the reporter dye to the fluorescence emission intensity of the passive reference dye).

water after being deparaffinized. Fresh *H. pylori* solutions were incubated with the sections for 15–30 min. Giemsa stain solution/distilled water (1:20) with 12.5% methanol was used to make *H. pylori*-solutions. Glass cover was placed over the mounted slides. These slides are ready to be examined under a microscope. Stool examination for *H. pylori* utilizing the InstaTest, RapiCard, One-Step *H. pylori* Antigen.

Statistical Analysis

The statistical programme for social sciences (SPSS) version 26 was used to handle and analyze the data. Frequencies, means, and standard deviation will be used for descriptive statistics, followed by chi-square and independent sample t tests for analytical statistics. If the p-value is less than 0.05, then the values are deemed significant.

Results

Findings of the Included OME Group

The study included 65 children with OME classified into two groups age and sex matched; Group A was (OME and adenoid hyperplasia) and included 18 males and 24 females with the mean age 7.4±1.7 years and group B was (OME only) and included 13 males and 10 females with the mean age 8.1±2.2 years with no statistically significant difference. GIT symptoms were reported in 3(7.1%) patients in group A and in 4(17.4%) patients in group B with no statistically significant difference (Table 1). *H. pylori* were detected by RT-PCR in 12 (28.6%) patients in group A (OME and adenoid hyperplasia) and in 4(17.4%) patients in group B (OME only) with no.

In stool, *H. pylori* Ag was positive in 19 (45.2%) patients in group A, and in 7(30.4%) patients in group B with no statistically significant difference (P value 0.1) (Table 2, Figure 2). *H. pylori* Ag in effusion was positive in 16 patients (24.6%) of all the studied patients. *H. pylori* Ag in stool was positive in 26 patients (40%) of all cases. All patients with positive *H. pylori* Ag in effusion had positive *H. pylori* Ag in stool. The association between age and *H. pylori*, there is

Table 1 Comparison Between Demographic Data in Both Groups of OME

Variables		Group A (Effusion with Adenoids) N = 42	Group B (Effusion without Adenoids) N= 23	P value
Age (years, mean±SD)		7.4±1.7	8.1±2.2	0.1
Sex (No., %)	• Males • Females	18(42.9%) 24(57.1%)	13(56.5%) 10(43.5%)	0.3
GIT symptoms(No., %)	• Yes • No	3(7.1%) 39(92.9%)	4(17.4%) 19(82.6%)	0.2

Abbreviations: OME, Otitis media with effusion; SD, standard deviation; GIT, gastrointestinal tract.

Table 2 Comparison Between the Frequency of *H. pylori* Ag in Middle Ear Effusions and Stool in Both Groups of OME

Variables		Group A (Effusion with Adenoids) N = 42	Group B (Effusion without Adenoids) N= 23	P value	Odds Ratio
PCR of effusion	Positive	12(28.6%)	4(17.4%)	0.2	2.1
	Negative	30(71.4%)	19(82.6%)		
Sample of stool	Positive	19(45.2%)	7(30.4%)	0.1	2.2
	Negative	23(54.8%)	16(69.6%)		

Abbreviations: OME, Otitis media with effusion; PCR, polymerase chain reaction.

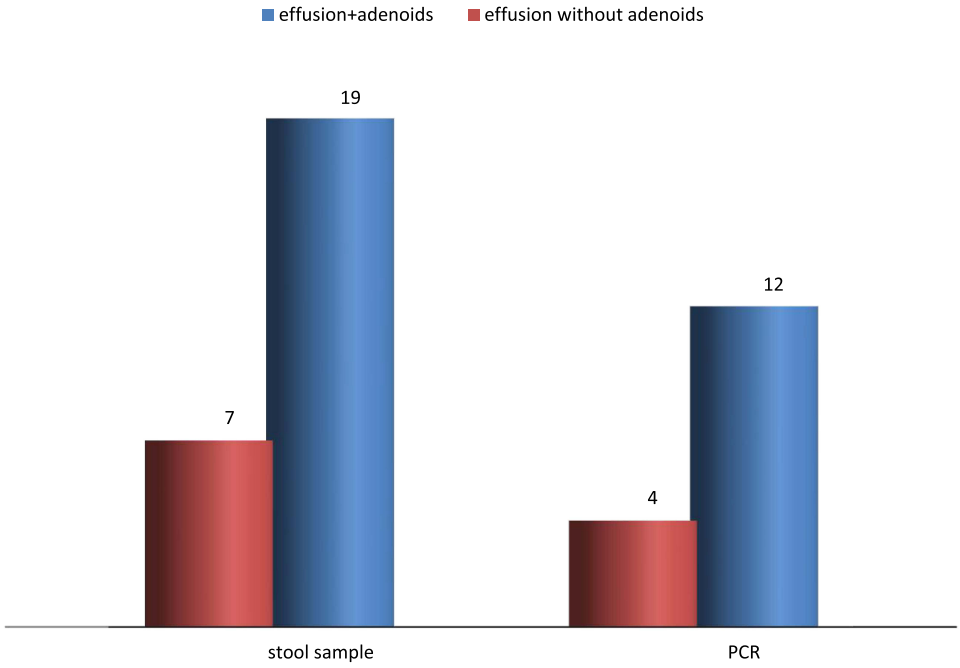

Figure 2 Comparison between both groups as regard to presence of *H. pylori* in effusion and stool.

an increased incidence of *H. pylori* in age 3–7 years and decrease in age 8–12 years (OR=6, CI 1.9–18.3). The association between sex and *H. pylori*, there is an increased incidence of *H. pylori* in females and a decreased frequency in males (OR=0.6, CI 0.2–1.6) (Table 3).

Results of the Included Patients with Nasal Polyps

This group of patients included 43 patients diagnosed with bilateral nasal polyps. The mean age was 43.1±15.8 years including 17 males (39.5%) and 26 females (60.5%). The de novo nasal polyps group included 9 males and 14 females with mean age (38.9±12.4 years). The recurrent nasal polyps group included 8 males and 12 females with mean age (47.9 ± 18.2 years) no statistically significant difference between both groups (Table 4 and Table 5).

In the de novo polyps group, 15 patients (65.2%) had GIT symptoms, but in the recurrent polyps group, 13 patients (65%) had GIT symptoms, with a P value of 0.9, which is not statistically significant (Table 5).

Table 3 Relation Between Both Groups of OME as Regard Presence of *H. pylori* According to Age, Sex by Multivariate Analysis

Variables		Group A (Effusion with Adenoids) N = 42	Group B (Effusion without Adenoids) N= 23	Odds Ratio	95% CI	P value
PCR	• Positive	12(28.6%)	4(17.4%)	1.9	0.534–6.67	0.4
	• Negative	30(71.4%)	19(82.6%)			
Sex (No., %)	• Males	18(42.9%)	13(56.5%)	0.577	0.2–1.6	0.1
	• Females	24(57.1%)	10(43.5%)			
Age range (No., %)	• 3–7 years	32(76.2%)	8(34.8%)	6	1.9–18.3	0.3
	• 8–12 years	10(23.8%)	15(65.2%)			

Abbreviations: OME, Otitis media with effusion; CI, confidence interval; PCR, polymerase chain reaction.

Table 4 Demographic Data of Patients with Nasal Polyps

Variables		Patients with Nasal Polyps (N=43)
Age (years, mean±SD)		43.1±15.8
Sex (No., %)	• Males • Females	17(39.5%) 26(60.5%)
Subgroups (No., %)	• Denovo nasal polyps • Recurrent polyposis	23(53.5%) 20(46.5%)

Table 5 Comparison Between Results of Presence of *H. pylori* in Both Groups of Nasal Polyps

Variables		Denovo Nasal Polyps (N=23)	Recurrent Polyposis (N=20)	P value
Age (years, mean±SD)		38.9±12.4	47.9± 18.2	0.06
Sex (No., %)	• Males	9(39.1%)	8(40%)	0.9
	• Females	14(60.9%)	12(60%)	
H. pylori in the biopsy (No., %)	• Negative	20(87%)	14(70%)	0.2
	• Positive	3(13%)	6(30%)	
H. pylori antigen in stool (No., %)	• Negative	9(39.1%)	9(45%)	0.7
	• Positive	14(60.9%)	11(55%)	
GIT symptoms (No., %)	• Negative	8(34.8%)	7(35%)	0.9
	• Positive	15(65.2%)	13(65%)	

Abbreviation: GIT, gastrointestinal tract.

In terms of the presence of *Helicobacter pylori* antigen in stool, it was positive in 14 patients (60.9%) with de novo bilateral nasal polyps and in 11 patients (55%) with recurrent bilateral nasal polyps, with a p value of 0.7 that is not statistically significant (Table 5).

Biopsies of nasal polyps were positive for *Helicobacter pylori* in 3(13%) patients of group A (de novo), and 6(30%) patients with recurrent bilateral nasal polyps with no statistically significant difference in p value 0.2 (Table 5).

There is a positive correlation between the presence of *H. pylori* antigen in the stool and the presence of the organism in biopsies of nasal polyps R (0.528), also there is a positive correlation between the presence of GIT symptoms and the presence of the organism in biopsies of nasal polyps R (0.455). Lastly, there is a positive correlation between the presence of *H. pylori* antigen in the stool and the presence of GIT symptoms R (0.764) (Table 6, Figure 3).

Table 6 Correlation Between Different Results of Presence of *H. pylori* in Patients of Nasal Polyps

Variables	R	P value
Positive biopsy for *H. pylori* and *H. pylori* antigen in stool	0.528	0.000
Positive biopsy for *H. pylori* andGIT symptoms	0.455	0.002
H. pylori antigen in stool andGIT symptoms	0.764	0.000

Abbreviation: GIT, gastrointestinal tract.

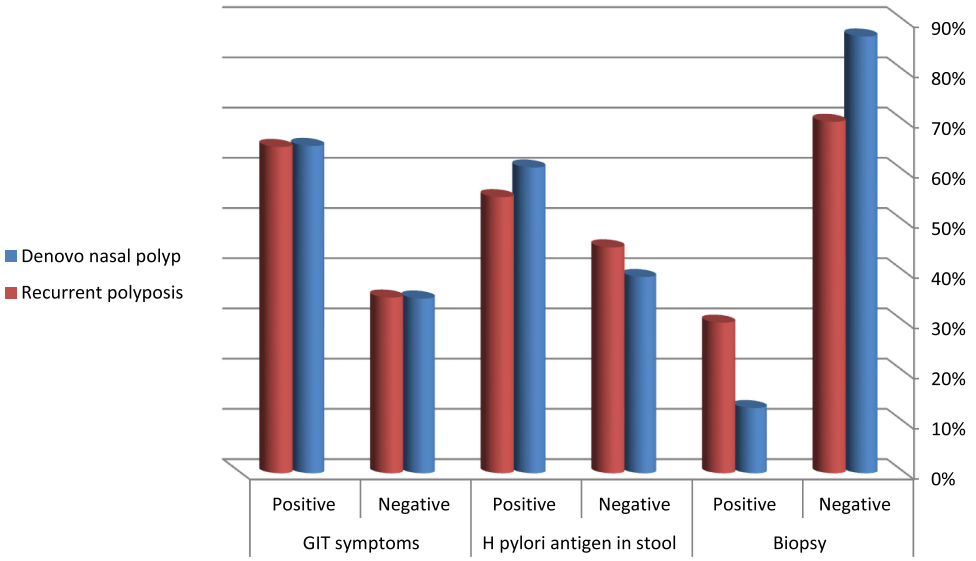

Figure 3 Comparison between both groups as regard to presence of GIT symptoms, presence of *H. pylori* in nasal polyps' biopsy and stool.

Results of Chronic Adenotonsillitis

This study included 78 children with their mean age was 7.53±2.28 years. The first group included 24 patients who have chronic tonsillitis and were subjected to tonsillectomy, second group included 26 patients with chronic adenoiditis who underwent adenoidectomy, and the third group included 28 patients with chronic adenotonsillitis who underwent adenotonsillectomy (Table 7).

All samples of adenoid tissue were negative for *H. pylori*, and only two samples of tonsillar tissue (8.3%) were positive for *H. pylori* organism. Regarding stool analysis, it was positive in 22 patients, 5 patients with tonsillitis, 10 with adenoiditis, and 7 with adenotonsillitis. Regarding GIT symptoms, it was positive in 20 patients; 5 patients with tonsillitis, 9 with adenoiditis, and 6 with adenotonsillitis (Table 8, Figures 4–6).

There is a positive correlation between presence of *H. pylori* antigen in stool and presence of GIT symptoms (r=0.937, p=0.000) (Table 9).

Discussion

Helicobacter pylori has been classified as a class I carcinogen in humans by The Working Group of the World Health Organization International Agency for Research on Cancer.[24] OME, nasal polyps, were asserted to be one of the extragastric manifestations of *H. pylori* infection. *H. pylori* is recognized to have numerous gastric and extragastric manifestations. Tonsils and adenoids were claimed to be a reservoir for this organism.[2,25]

Table 7 Demographic Data of Patients with Chronic Adenotonsillitis

Variables		Patients with Adenotonsillar Hypertrophy (N=78)
Age (years, mean±SD)		7.53±2.28
Sex (No., %)	• Males	38(48.7%)
	• Females	40(51.3%)
Subgroups (No., %)	• Tonsillitis	24(30.8%)
	• Adenoditis	26(33.3%)
	• Adenotonsillitis	28(35.9%)

Table 8 Results of *H. pylori* Detection in Different Groups of Adenotonsillar Hypertrophy

Variables (No., %)		Tonsillitis (N=24)	Adenoiditis (N=26)	Adenotonsillitis (N=28)	P value
Microscopic examination for *H. pylori*	• Positive	2(8.3%)	0(0%)	0(0%)	0.09
	• Negative	22(91.7%)	26(100%)	28(100%)	
Stool analysis for *H. pylori* antigen	• Positive	5(20.8%)	10(38.5%)	7(25%)	0.3
	• Negative	19(79.2%)	16(61.5%)	21(75%)	
GIT symptoms	• Positive	5(20.8%)	9(34.6%)	6(21.4%)	0.2
	• Negative	19(79.2%)	17(65.4%)	22(78.6%)	

It is proved that *H. pylori* needs a microaerophilic environment for its replication, which may explain the role of *H. pylori* in the pathogenesis of many upper GIT, and URT problems. It is known that *H. pylori* are colonized by multiplying inside the epithelium of the mucosal layer of the stomach. The mucosal side of the stomach has a pH of 7. The middle ear fluid has a pH that ranges from 7.0 to 9.0. *H. pylori* infection in the stomach causes goblet cell hyperplasia and mucosal metaplasia, and the same pathological changes are reported in OME.[26,27] Hearing loss is due to OME affecting a high percentage of children. In this study, the presence of *H. pylori* antigen was assessed in middle ear fluid and also in the stool.

In this study *H. pylori* was positive in patients with OME with adenoid hyperplasia and in patients with OME without adenoid hyperplasia with no statistically significant difference, which means that *Helicobacter pylori* has no role in the pathogenesis of OME, and adenoids does not act as a reservoir for bacterial colonization, this is similar to results recorded by six original research papers, including four prospective cohort studies, two randomized controlled studies, and 27 controls, were used in a systematic review by Sudhoff et al to examine the presence of *H. pylori* in the middle ear of patients with otitis media with effusion. They discovered a weak correlation between the presence of *H. pylori* and otitis media with effusion.[28] Yilmaz et al discovered HP by real-time polymerase chain reaction in 16 out of 34 ear effusion samples (47%) with RT-PCR.[29] According to Bilter et al, there is no correlation between *H. pylori* and OME. They employed PCR and culture on 28 effusion samples, and all of the results were negative.[30]

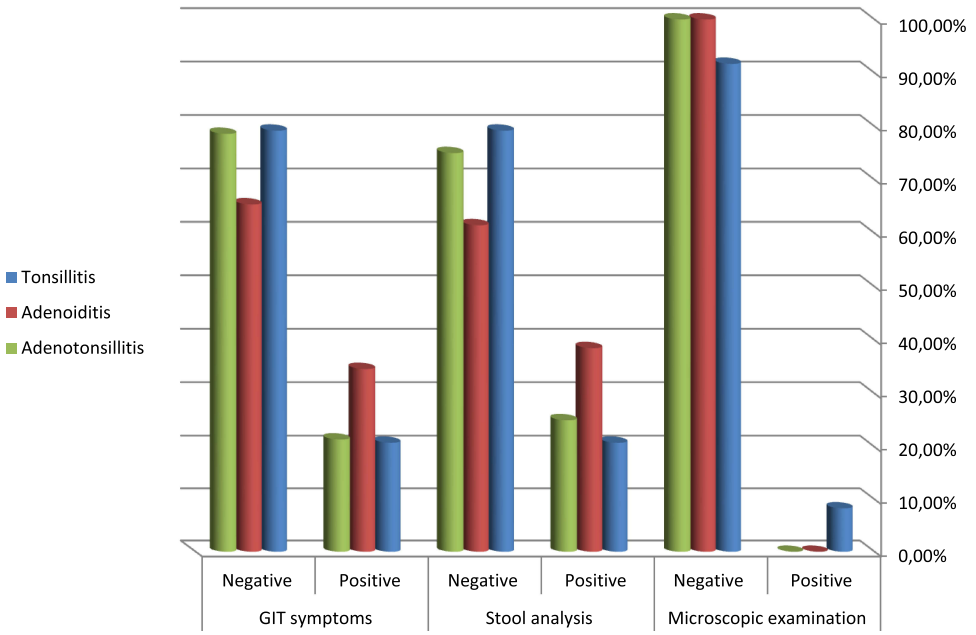

Figure 4 Comparison between the three groups as regard to presence of GIT symptoms and presence of H. pylori in both tissues' biopsies and stool.

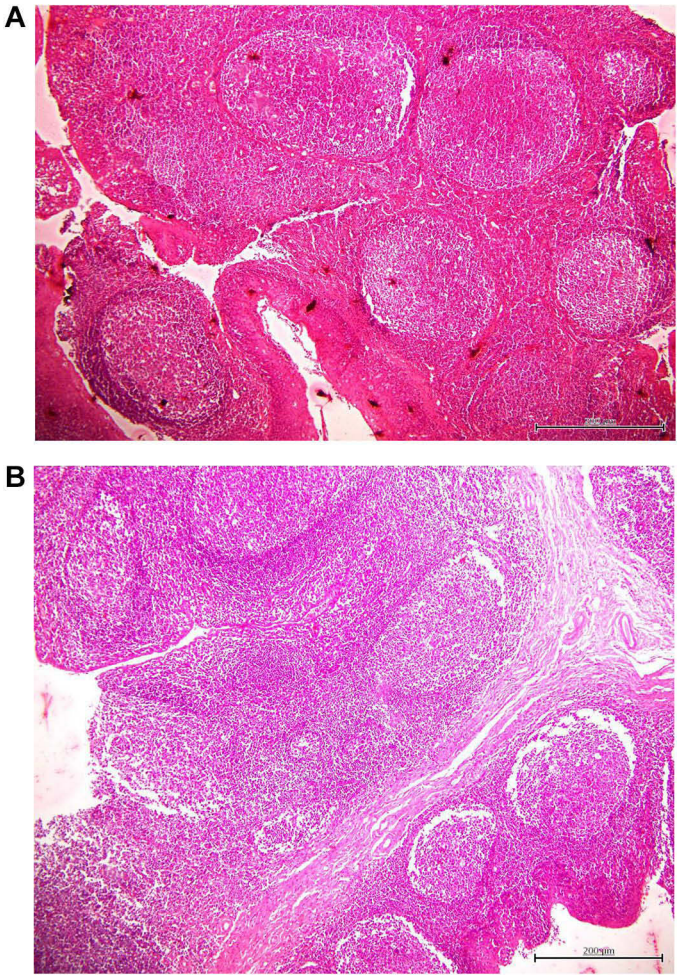

Figure 5 H & E stain showing tonsil with reactive follicles and positive for H pylori organism.

Moreover, there was no statistically significant difference between patients with OME who had positive *H. pylori* Ag in their stool and those who were negative. *H. pylori* may be involved in OME, according to the results of another study by Mel-Hennawi and Ahmed,[31] which contrasts with the findings of this study. According to Fancy and his colleagues, *H. pylori* has no role in the pathophysiology of OME. In patients with middle ear effusion and adenoid hyperplasia, they used PCR to find *H. pylori* in adenoid tissue. With no discernible difference, they found *H. pylori* in the adenoid tissue of patients with OME in 22.2% of cases and in 16.2% of cases with adenoid hyperplasia without middle ear effusion.[32] Saki et al, who investigated the frequency of *H. pylori* in OME patients. He looked at two groups; the first one had 84 patients who had been admitted for an adenoidectomy and myringotomy. Ninety-one individuals who underwent an adenoidectomy alone make up the second group. 25% of patients in the first group and 19.8% of patients in the second group had *H. pylori* in their adenoid samples. *H. pylori* was detected in 42.8% of the patient effusion samples from the first group.[33] Khasawneh et al observed that a considerable number (38.6%) of patients with OME had a history of gastric acid reflux; this study only reported GIT signs in 10% of patients; this difference may be attributable to the different age groups of the two studies.[23]

In the current study *Helicobacter pylori* Ag was positive in stool in all patients with OME positive effusion to *H. pylori* Ag. This raises the importance of detection of *H. pylori* Ag in stool in patients with OME. Still, several studies and larger numbers of patients are needed to prove such association. Also, no relation was found between upper GIT symptoms and OME with or without adenoid.

In this study, most patients with nasal polyps were in the second to fourth decades. This is similar to the results of several studies reported that second to fourth decade patients were the most commonly affected age group.[17,34,35]

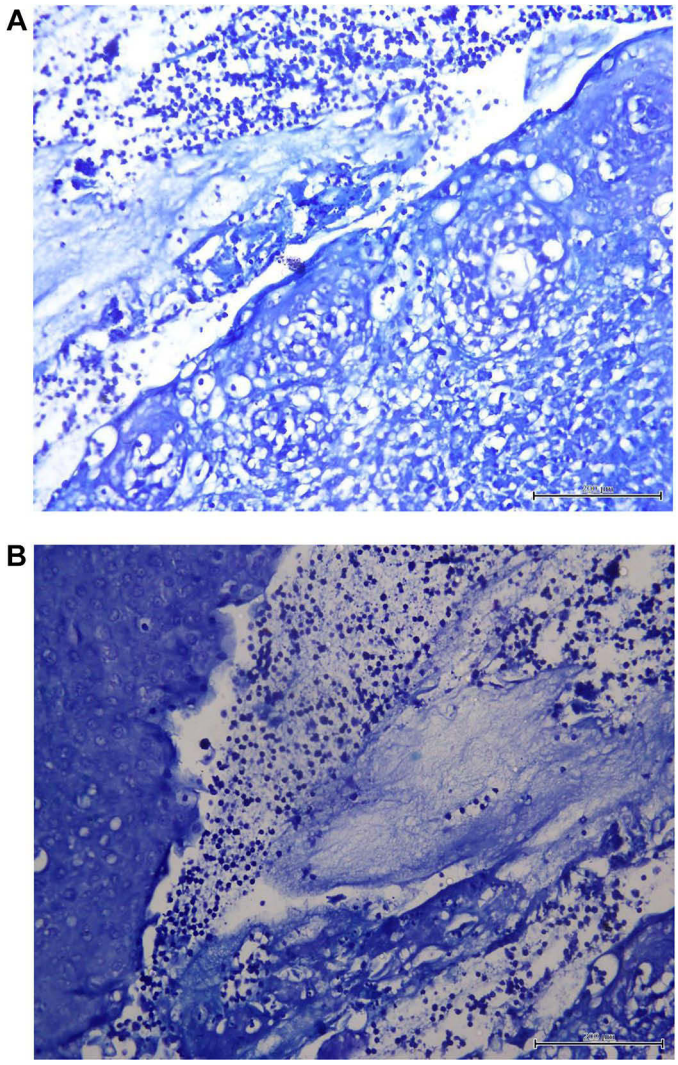

Figure 6 Giemsa stain x 400 showing *H. pylori* organism in mucous film at surface of tonsil.

In this study, females were more likely to have bilateral nasal polyps (female: male ratio of 1.5:1). According to Aref et al and Bakari et al, the female-to-male ratio was 1.5:1 and 1.2:1, respectively.[17,36] Yet, according to Bansal et al, males are more likely to develop nasal polyps.[37]

This study found *H. pylori* bacteria positive biopsies in some cases of de novo nasal polyps and some cases of recurrent nasal polyps Aref et al obtained results that were remarkably similar.[17] Cvorovic and Al-Abbasi reported different outcomes in their research on nasal polyps using modified Giemsa staining, reporting 26% and 35% positive.[38,39] Moreover, H & E was positive in 21.4% of patients, according to Bansal et al.[37]

Table 9 Correlation Between Different Results for *H. pylori* Detection in Patients with Adenotonsillar Hypertrophy

Variables	R	P value
Microscopic *H. pylori* detection and stool analysis for *H. pylori* antigen detection	0.079	0.5
Microscopic *H. pylori* detection and GIT symptoms	0.090	0.4
Stool analysis for *H. pylori* antigen detection andGIT symptoms	0.937	0.000

Abbreviation: GIT, gastrointestinal tract.

Stool analysis for the presence of *H. pylori* antigen was carried out to determine its relevance to the patients' gastrointestinal health. The results were positive in more than half of the included patients who had bilateral de novo nasal polyps or recurrent nasal polyps. This is in agreement with Aref et al,[17] who obtained results (52.5%) and (60%) that were remarkably similar.

According to the results of the current investigation, patients with positive biopsies also had positive stool antigen tests. This concurs with Aref et al[17] and Bansal et al.[37] Also, this study found no statistically significant link between *H. pylori* and bilateral nasal polyps, concurring with Aref et al and Al-Abbasi's findings that there was no clear link between the two.[17,40] Nevertheless, nasal polyps and *H. pylori* have been linked in a significant way, according to Bansal et al.[37]

Adenotonillar hypertrophy results revealed that all samples of adenoid tissue and most samples of tonsillar tissue were negative for the *H. pylori* bacteria. Studies on the colonization of *H. pylori* in adenoid and tonsillar tissues have produced a variety of outcomes. According to Unver et al report of 57.9% positive CLO tests on samples taken from patients who underwent adenotonsillectomy,[41] and when checking for the presence of *H. pylori* using PCR, Cirak et al discovered 30% positive findings.[42–46] Vilarinho et al, on the other hand, discovered no positive results using PCR-DEIA assays.[42] Additionally, just two samples (2%) produced positive findings on the fast urease test, while Güçlü et al discovered no positive growth in culture.[43] According to Hwang et al, tonsillar tissue colonization with *Helicobacter pylori* was not found to be more common with chronic or recurring infections.[44] On a study done by Wu et al 2022 reported that the presence of *Helicobacter pylori* in patients with chronic tonsillitis was significantly higher than the control group of children. They suspected that *H. pylori* had a role in chronic tonsillitis.[45]

Study's Limitation

Single-center study is the main study limitation, so further study in various centers are recommended to confirm the findings of the current research.

Conclusion

There is no indication that *H. pylori* played a role in the emergence of OME. There is no connection between the pathogenesis of bilateral nasal polyposis, either de novo or recurrent, and *H. pylori*. Chronic adenotonsillitis and *H. pylori* adenotonsillar colonization did not significantly correlate.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no competing interests in this work.

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