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Helicobacter pylori infection in recurrent abdominal pain in children

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Abstract
Helicobacter pylori, previously known as Campylobacter pylori, is a gram-negative, microaerophilic bacterium usually found in the stomach. It is also linked to the development of duodenal ulcers and stomach cancer.
The study was conducted in the Department of Paediatrics in Sri Krishna Medical College and Hospital. Total 80 patients reported with abdominal pain were enrolled into the study. The childrens who fulfilled Apley's criteria of RAP at least three discrete episodes of abdominal pain of sufficient severity, to interrupt normal daily activities or performance, occurring over a period of three or more months) were enrolled.
Helicobacter pylori (HP) infection and Recurrent Abdominal Pain (RAP) are two major childhood challenges presenting a dilemma in diagnosis and treatment. There was no association between HP infection and RAP. There was a significant association between HP infection and epigastric pain, vomiting and IDA in symptomatic children and not in asymptomatic children suggesting that RAP is not an indication for a test-and-treat strategy for HP infection in children.

Keywords: *Helicobacter pylori*, recurrent abdominal pain, children's, etc.

Introduction

Helicobacter pylori (see the image below) is a ubiquitous organism that is present in about 50% of the global population. Chronic infection with H pylori causes atrophic and even metaplastic changes in the stomach, and it has a known association with peptic ulcer disease. The most common route of H pylori infection is either oral-to-oral or fecal-to-oral contact.

In general, patients infected with H pylori are asymptomatic, and no specific clinical signs and symptoms have been described. When signs and/or symptoms are present, they may include the following [1]:

- Nausea
- Vomiting
- Abdominal pain
- Heartburn
- Diarrhea
- Hunger in the morning
- Halitosis (bad breath)

Helicobacter pylori, previously known as Campylobacter pylori, is a gram-negative, microaerophilic bacterium usually found in the stomach. It was identified in 1982 by Australian scientists Barry Marshall and Robin Warren, who found that it was present in a person with chronic gastritis and gastric ulcers, conditions not previously believed to have a microbial cause. It is also linked to the development of duodenal ulcers and stomach cancer. However, over 80% of individuals infected with the bacterium are asymptomatic, and it may play an important role in the natural stomach ecology [2].

More than 50% of the world's population have *H. Pylori* in their upper gastrointestinal tract. Infection is more common in developing countries than Western countries. *H. Pylori*'s helical shape (from which the genus name derives) is thought to have evolved to penetrate the mucoid lining of the stomach [3].

Colonization with *H. Pylori* is not a disease in and of itself, but a condition associated with a number of disorders of the upper gastrointestinal tract. Testing for *H. Pylori* is recommended if peptic ulcer disease or low-grade gastric MALT lymphoma is present, after endoscopic resection of early gastric cancer, first-degree relatives with gastric cancer, and in certain cases of dyspepsia, not routinely. Several ways of testing exist. One can test noninvasively for *H. Pylori* infection with a blood antibody test, stool antigen test, or with the carbon urea breath test (in which the patient drinks 14C-or 13C-labelled urea, which the bacterium metabolizes, producing labelled carbon dioxide that can be detected in the breath). Also, a urine ELISA test with a 96% sensitivity and 79% specificity is available. None of the test methods is completely failsafe. Even biopsy is dependent on the location of the biopsy. Blood antibody tests, for example, range from 76% to 84% sensitivity. Some drugs can affect *H. Pylori* urease activity and give false negatives with the urea-based tests. The most accurate method for detecting *H. Pylori* infection is with a histological examination from two sites after endoscopic biopsy, combined with either a rapid urease test or microbial culture [4].

Once *H. Pylori* is detected in a person with a peptic ulcer, the normal procedure is to eradicate it and allow the ulcer to heal. The standard first-line therapy is a one-week "triple therapy" consisting of proton pump inhibitors such as omeprazole and the antibiotics clarithromycin and amoxicillin. Variations of the triple therapy have been developed over the years, such as using a different proton pump inhibitor, as with pantoprazole or rabeprazole, or replacing amoxicillin with metronidazole

for people who are allergic to penicillin. In areas with higher rates of clarithromycin resistance, other options are recommended. Such a therapy has revolutionized the treatment of peptic ulcers and has made a cure to the disease possible. Previously, the only option was symptom control using antacids, H2-antagonists or proton pump inhibitors alone [5].

An increasing number of infected individuals are found to harbor antibiotic-resistant bacteria. This results in initial treatment failure and requires additional rounds of antibiotic therapy or alternative strategies, such as a quadruple therapy, which adds a bismuth colloid, such as bismuth subsalicylate. For the treatment of clarithromycin-resistant strains of *H. Pylori*, the use of levofloxacin as part of the therapy has been suggested [6].

This study is planned to investigate correlation between *H. Pylori* infection and Recurrent Abdominal Pain (RAP) as well as other GI symptoms in children and thereby to evaluate the clinical significance of *H. Pylori* infection.

Methodology

The study was conducted in the Department of Paediatrics in Sri Krishna Medical College and Hospital. Total 80 patients reported with abdominal pain were enrolled into the study. The childrens who fulfilled Apley's criteria of RAP at least three discrete episodes of abdominal pain of sufficient severity, to interruptnormal daily activities or performance, occurring over a period of three or moremonths) were enrolled. The all study group children's stool microscopy, and urine examination is carried to out. Upper gastrointestinal endoscopy was performed with a fiberoptic pediatric size endoscope (GIF Type PQ 20), after informed consent was obtained from the parents. Endoscopic changes were noted in the esophagus, stomach and duodenum. Multiple biopsy samples were taken endoscopically from duodenum and antrum, and evaluated.

Results & Discussion

The data from the total 80 patients were collected. There are 40 patients of cases and 40 patients of the controlled cases were involved in the study. Table 1 represents the age distribution in the both the study group patients.

Table 1: Age distribution of Case & Control Study group patients

Age	Case Study Group	Control Study Group
Less than 5 years	7	5
5- 8 years	15	19
8 -10 years	8	7
10 – 12 years	10	9
Total	40	40

There are 7 children in case and 5 children in control study group with age less than 5 years. There are 15 children in case and 19 children in control study group with age of 5- 8 years. There are 8 children in case and 7 children in control study group with age of 8 -10 years. There are 10 children in case and 9 children in control study group with age 10 -12 years.

In the case study group there is prevalence of 10 children out of 40 found positive with *H. Pylori* infections.

Table 2: *H. Pylori* positive cases

	Case Study Group	Control Study Group
Total Cases	40	40
H. Pylori positive cases	10	nil

Table 3: Shows the data by the histopathological findings.

Table 3: Number of Cases as per Histopathological findings

Observations	Total Case	*H. Pylori* positive cases
A) Antral Gastritis	18	5
▪ Mild	11	3
▪ Moderate	7	2
▪ Severe	0	0
B) Duodenitis	17	3
▪ Mild	8	1
▪ Moderate	5	1
▪ Severe	4	1
C) Villus Stuning	5	2
Total	40	10

The clinical manifestations of *H. Pylori* infection are not very clearly defined in children. The infection is supposed to be acquired in early childhood, but complex host bacterium relationship determines the clinical manifestations of the disease [7]. In the present study 18 cases of Antral Gastritis. 17 cases of Duodentis and 5 cases with Villus stunting.

There is a proposed link between Hp infection and RAP reported to range from 5- 34% [8, 9]. Estimation of prevalence of Hp in RAP in comparison to asymptomatic pediatric popultion by using endoscopy guided biopsies in the latter is obviously unethical. Therefore prevalence of Hp in asymptomatic children of developing and developed populations have been estimated using serology alone. This varies widely, from 4-75%, higher rates being generally from developing countries [10]. Macarthur *et al*. [11] made a MEDLINE search from January 1983 through July 1994 and found inconsistent prevalence rates of infection in children with recurrent abdominal pain (range, 0-81%; median, 22%). The rates were still lower in children meeting Apley's criteria (range 0-9%; median 6%).

There is a strong correlation between duodenal ulceration and Hp gastritis in children [12]. Duodenal ulcer was not observed in any child in our study. However, a statistically significant association was observed between histopathological evidence of duodenitis and Hp colonization. Relatively few workers have identified Hp associated duodenitis. Madsen *et al*. in a study of 45 adult patients found increased frequency of chronic active duodenitis in patients with Hp compared with those lacking it [13].

Diagnosis with the help of serology as well as RUT are known to be highly sensitive and specific [14]. Histology scores even better. However, sampling error is thought to be a minor difficulty in the his to logical diagnosis of Hp due to its patchy distribution [14]. This could be the cause of false negative result of histology in two cases in this study. On the other hand, these two cases, who had both RUT and serology positive, could be false positive, which is less likely. If histology is regarded as the gold standard then the sensitivity and specificity of RUT/serology in the present study is 100%

and 93%, respectively. These observations are similar to those reported by earlier workers [14].

Conclusion

HP infection and RAP are two major childhood challenges presenting a dilemma in diagnosis and treatment. There was no association between HP infection and RAP. There was a significant association between HP infection and epigastria pain, vomiting and IDA in symptomatic children and not in asymptomatic children suggesting that RAP is not an indication for a test-and-treat strategy for HP infection in children.

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