**Helicobacter pylori in Children Who Are Prone to Upper Respiratory Tract Infections**

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**Objective:** To examine the presence of *Helicobacter pylori* infection in children with recurrent upper respiratory infections, including acute otitis media.

**Design:** A prospective clinical trial.

**Setting:** Tertiary referral center.

**Patients:** A total of 20 children who were prone to upper respiratory infections and acute otitis and who were admitted for elective adenoidectomy and/or tympanostomy were included in the study.

**Main Outcome Measures:** Samples of adenoid tissue and middle ear fluid were cultured for *H pylori*.

Serologic analysis and fecal antigen detection testing were also carried out to determine the presence of *H pylori*.

**Results:** The results of serologic and antigen detection tests were positive for *H pylori* in 4 children (20%), but cultures of adenoid tissue and middle ear fluid samples were negative for *H pylori* in all children.

**Conclusion:** An association between *H pylori* infection and recurrent upper respiratory infections and otitis media could not be established.


*Helicobacter pylori* colonizes gastric mucosa and can be found in gastric juice. Reflux of gastric juice has been suggested to be associated with glue ear and chronic sinusitis in children. It has also been suggested that tonsil and adenoid tissues are potential reservoirs of *H pylori* infection. These observations raise the question as to whether *H pylori* infection might have a role in frequent respiratory tract infections (FRTIs) and recurrent acute otitis media (rAOM) in respiratory infection-prone children.

The aim of the present study was to examine the presence of *H pylori* by culture of samples of adenoid tissue and middle ear fluid from children with FRTIs and rAOM. Furthermore, *H pylori* infection was studied by fecal antigen detection testing and by determination of serum antibody titers.

The study included 20 children (14 boys; age range, 1-7 years [median age, 2 years]) from refugee families of African origin who had been admitted to the Helsinki University Central Hospital, Helsinki, Finland, for elective adenoidectomy and/or tympanostomy between October 2002 and June 2003. All the children were admitted because of FRTIs and rAOM. A child was considered to have FRTIs and rAOM if he or she had experienced at least 9 upper respiratory tract infections and at least 4 episodes of AOM during the year before admittance. All the children underwent adenoidectomy under general anesthesia, and the adenoid tissue sample was sent for *H pylori* culture. Myringotomy was performed in the children with suspected effusion in the middle ear cavity. Middle ear effusions (8 children, 12 ears) were collected for *H pylori* culture. Serum samples were obtained from all 20 children during the anesthesia. The parents were asked to bring a fecal sample from their children for *H pylori* antigen detection, and 10 such samples were available. The study was accepted by the local ethical committee, and an informed written consent was obtained from the parents or guardians.

**CULTURE**

Samples were cultured on Brucella agar plates (Becton Dickinson, Sparks, Md) supplemented with horse blood (7%). Selective Brucella agar plates contained 7% horse blood, 0.5% yeast extract, 0.5% tryptone, 1% Brucella broth, and 0.5% L-cysteine HCl.
SEROLOGIC TESTS

Serum samples were stored at −20°C until analyzed for IgG and IgA antibodies to H pylori with an in-house enzyme immunoassay. The antigen used was an acid glycine extract from H pylori strain NCTC 11637. The lower limits for the raised titers were 700 for IgG and 70 for IgA antibodies. In a series of adult outpatients, the assay showed a specificity of 93% and a sensitivity of 100% when compared with gastric histologic analysis. The results of this test were seropositive in 94% of the children whose H pylori infection was verified by gastric biopsy–based methods (culture, histologic analysis, or biopsy urease test).

Fecal samples from 4 (20%) of the 20 children were positive for H pylori on serologic analysis and/or antigen detection testing; 3 of the 4 children were younger than 5 years and 1 was 7 years old. The results were positive in 3 of 10 fecal samples. Two of the 3 children whose samples were fecal antigen positive (aged 1 year 9 months and 7 years) had positive results on the fecal antigen tests and 1 child (aged 3 years) had positive results only on the monoclonal antibody test. The samples from 2 of these 3 children were positive for H pylori on serologic testing, and the sample from 1 child was seronegative. The fecal sample from 1 child (aged 3 years) whose results were seronegative was not available. The cultures of 20 adenoid samples and 12 middle ear fluid samples were all negative for H pylori.

In the present study, we evaluated the possible role of H pylori infection in respiratory infection–prone children. In this series, the samples from only 20% of the children with FRTIs and rAOM were H pylori positive on serologic testing and/or fecal antigen detection, and all H pylori cultures of adenoid tissue and middle ear fluid samples were negative, thus showing no association between H pylori infection and FRTI and rAOM.

Our findings do not agree with those of some earlier studies that suggested that adenoid tissue acted as a reservoir of H pylori. In those studies, more than 20% of adenoid biopsy specimens from children with either recurrent or chronic tonsillitis were positive for H pylori on polymerase chain reaction analysis. Although the sensitivity of H pylori culture of gastric biopsy specimens is usually lower than that of other diagnostic tests, such as polymerase chain reaction analysis, high sensitivity figures can be obtained, and as the specificity is 100%, it is a very convincing method with which to demonstrate the existence of live bacteria. Although the detection of H pylori by polymerase chain reaction analysis of samples of adenoid tissue may suggest that this tissue acts as a reservoir of H pylori, it may also represent transient rather than persistent colonization. Several studies have shown that H pylori may exist transiently in the mouth. The transient existence of H pylori in the oral cavity seems to be associated with H pylori colonization in the stomach, but it is not known whether this reflects exposure to H pylori via an oral route or the presence of gastroesophageal reflux and the origin of this bacterium from the gastric lesions.

Because the prevalence of H pylori is low and decreasing among Finnish children, we studied children only from refugee families of African origin. Among African children living in Finland, H pylori infection is common, and 63% of the children with gastrointestinal symptoms are seropositive for H pylori. In the present study, we found that samples from only 20% of the children with FRTIs and rAOM were H pylori positive on either serologic testing or fecal antigen detection. Since our study was conducted, several microbial treatments (often amoxicillin) have been made available that may suppress the growth of H pylori, but they have not been of sufficient strength to completely eradicate the bacteria. The levels of serum antibodies in children infected with H pylori, however, do not decrease until there is proper eradication of the bacteria. This low rate of positivity for H pylori further suggests that H pylori infection has no role in FRTIs in children.

Although some authors who have used modern microbiological techniques have suggested that H pylori may have a role in upper respiratory tract infection, to our knowledge no one has been able to culture the bacteria and we were not able to confirm H pylori colonization in samples of adenoid tissue or middle ear fluid from children with FRTIs and rAOM.
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REFERENCES