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SHORT SCIENTIFIC COMMUNICATION

Otitis media with effusion: A possible role for *Helicobacter pylori?*

Tanya Fancy, MD, Peter H. Mathers, PhD, and **Hassan H. Ramadan, MD, MSc,** Morgantown, WV

OBJECTIVES: To compare *Helicobacter pylori* prevalence rates in the nasopharynx of pediatric patients with and without otitis media with effusion (OME).

STUDY DESIGN: Prospective, controlled.

METHODS: The study group consisted of patients undergoing adenoidectomy for persistent OME. A control group of patients with no history of OME undergoing adenoidectomy was simultaneously enrolled. As a substudy, middle ear effusion samples were also analyzed for *Helicobacter pylori*. Polymerase chain reaction assays were run by using primers against the *Helicobacter pylori* gene.

RESULTS: *Helicobacter pylori* was detected in the adenoids of 10 of 45 study group patients, and 6 of 37 controls ($P = 0.49$). Thirty-two percent of middle ear aspirates were positive for *Helicobacter pylori*.

CONCLUSION: The current study confirms the presence of *Helicobacter pylori* in the nasopharynx and middle ear space, but our results do not support a role for this bacterium in the pathogenesis of otitis media with effusion.

Sponsorships or competing interests that may be relevant to this article are disclosed at the end, preceding the references.

There has been a heightened interest in recent literature in the role of *Helicobacter pylori* as one of the organisms implicated in otitis media with effusion (OME). Because *H pylori* has been discovered in anatomical sites such as the adenoids, which are in proximity to the Eustachian tube, it naturally follows that this bacterium may play a role in middle ear disease.

Thus far, there are very limited definitive data on *H pylori* and OME in children, and even more scant literature on the US pediatric population. Our goal was to determine whether this bacterium is indeed one of the middle ear pathogens that needs to be considered when evaluating a child with refractory middle ear disease.

We used polymerase chain reaction (PCR) to determine whether the prevalence rates of *H pylori* in the adenoids of children with OME differ from those in the adenoids of a control group of children with no history of OME.

METHODS

This prospective study was conducted at a tertiary care hospital over a 4-month period from January to April 2007. The institutional review board granted approval for the study.

The study group included patients aged younger than 18 years undergoing adenoidectomy (with or without ventilation tube placement) for management of chronic or recurrent OME. A total of 45 patients were enrolled in this group.

The control group included 37 pediatric patients with no history of OME, undergoing adenoidectomy (usually as part of an adenotonsillectomy procedure) for an indication of obstructive sleep apnea or chronic adenotonsillitis during the same 4-month period.

Although not the focus of the study, middle ear fluid was also sampled from the study group and analyzed for *H pylori* if the patients were undergoing ventilation tube placement as part of their procedure. To supplement the numbers for this limb of the study, middle ear fluid was also collected from an additional 39 children who were undergoing ventilation tube (VT) placement during the time frame of our study (Fig 1).

Adenoid tissue biopsies were taken with cup forceps and laryngeal mirror prior to performing adenoidectomy. Each biopsy included the surface and core.[1]

Middle ear fluid was aspirated without blood contamination using a JUHN Tym-Tap Sterile Disposable Middle Ear Fluid Aspirator/Collector (Xomed Surgical Products, Jacksonville, FL) under sterile conditions. If present bilaterally, middle ear fluid was collected from both sides and considered one sample representative of the patient.

Primer-3 (5'-TGGCGTGTCTATTGACAGCGAGC-3') and primer-4 (5'-CCTGCTGGGCATACTTCACCAG-3') targeting the 303-bp fragment, which codes for the *H pylori* 26-kDa SSA gene, were selected for amplification.[2]

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A positive sample was one that produced a single band of this size on gel electrophoresis. *Helicobacter pylori* ATCC strain 43504 was used as a positive control with each set of reactions. Sterile water was used as a negative control in each experiment. Patient samples that demonstrated a single band of the appropriate size were run a second time to confirm a positive result.

RESULTS

Gender distribution was similar in both groups ($P = 0.12$). The mean age of all the patients was 4.4 years, with a range from 8 months to 13.3 years.

PCR assays for *Helicobacter pylori* were positive in the adenoid tissue of 10 of the 45 (22.2%) study group patients and 6 of 37 (16.2%) control group patients (Table 1). The difference in *H pylori* prevalence rates between these groups was not statistically significant ($P = 0.49$). The presence of *H pylori* in the nasopharynx (in either group) was not associated with a history of reflux ($P = 0.60$).

Twenty-three (31.5%) of all 73 middle ear samples were *H pylori*–positive. Of the 10 study group patients with *H pylori* present in their adenoids, four also had this organism present in the middle ear.

Table 1
Results of *H pylori* PCR assay on adenoid specimens of the study and control groups

	H pylori positive	*H pylori* negative
Study group adenoid	10	35
Control group adenoid	6	31

DISCUSSION

The nasopharynx has been implicated as a source of the common organisms causing middle ear disease in the pediatric age group. Adenoidectomy is therefore a common procedure as part of the treatment of OME.[1] The goal of the current study was to determine whether *H pylori*, through its colonization of the nasopharynx, might have a role to play in pediatric middle ear disease.

H pylori prevalence in the adenoids in our study was 22.2 percent in the study group and 16.2 percent in the control group. Total prevalence in the nasopharynx (study and control groups combined) was 19.5 percent. These prevalence rates vary from zero to 64 percent in the literature, depending on the technique used to isolate *H pylori.*[1,3] Although the current study does demonstrate the presence of *H pylori* in the nasopharynx (and middle ear fluid) of children with OME, the prevalence rates do not appear differ from those in the nasopharynx of children without OME ($P = 0.49$).

H pylori presence in middle ear effusions in the current study was 31.5 percent. This result is in concordance with other studies in which prevalence rates between 16.3 and 35 percent have been reported with the use of PCR.[3,4] It may be of value to include an age-matched control group for the middle ear limb of the study to help confirm our findings in the nasopharynx.

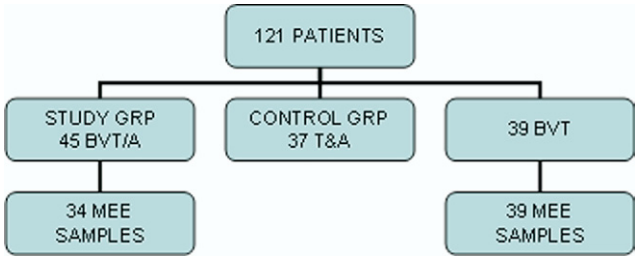

Figure 1 Distribution of patients in the study. *BVT/A,* Bilateral ventilation tubes with adenoidectomy; *T&A,* adenotonsillectomy; *BVT,* bilateral ventilationtubes only; *MEE,* middle ear effusion.

CONCLUSIONS

It is important to investigate all the pathogens implicated in refractory OME so that treatment is complete and targets the responsible organism. The current study confirms the presence of *H pylori* in the nasopharynx and middle ear space, but our results do not support a role for this bacterium in the pathogenesis of OME.

AUTHOR INFORMATION

From the Department of Otolaryngology–Head and Neck Surgery, West Virginia University (Drs Fancy and Ramadan) and the West Virginia University School of Medicine (Dr Mathers).

Corresponding author: Hassan H. Ramadan, MD, Department of Otolaryngology–Head and Neck Surgery, Room 2222, Health Sciences Center South, PO Box 9200, Morgantown, WV 26506.

E-mail address: hramadan@hsc.wvu.edu.

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AUTHOR CONTRIBUTIONS

Hassan Ramadan: supervisor, data analysis, manuscript review and editing; **Tanya Fancy:** sample and data collection, sample analysis and processing, data analysis, manuscript preparation, first author; **Peter Mathers:** supervisor, sample processing and analysis.

FINANCIAL DISCLOSURES

Hassan Ramadan: investigator: Acclarent; **Tanya Fancy:** none; **Peter Mathers:** none.

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