Incidence of Streptococcal Carriers in Private Pediatric Practice

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Objective: To determine the incidence of group A β-hemolytic streptococcus (GABHS) carriers in children who are well, in children seen with presumed and documented viral illnesses with sore throat, and in children after treatment of acute GABHS tonsillopharyngitis with 10 days of oral penicillin V potassium, oral cephalosporins, or macrolides.

Methods: Prospective collection of clinical and microbiologic data from October 1996 to June 1997 in a private pediatric practice were obtained from children who were asymptomatic and well, from children with both presumed (and documented) viral sore throats, and from children who had completed a full antibiotic treatment course for acute GABHS throat infections.

Results: The incidence of GABHS carriers was 2.5% among well children (n = 227), 4.4% among children with upper respiratory tract infections including sore throat of presumed viral etiology (n = 296), and 6.9% among children with upper respiratory tract infections including sore throat from whom viruses were isolated (n = 87). Following 10 days' treatment of acute GABHS tonsillopharyngitis, 81 (11.3%) of 718 children treated with penicillin, 22 (4.3%) of 508 children treated with an oral cephalosporin, and 10 (7.1%) of 140 children treated with a macrolide were GABHS carriers (P < .001).

Conclusions: A small percentage of children seen in private pediatric practices who are well or who have apparent viral upper respiratory tract infections with sore throat are GABHS carriers. Penicillin treatment of acute GABHS tonsillopharyngitis results in a higher GABHS carriage rate than occurs following treatment with cephalosporins and macrolides.


CARRIERS OF group A β-hemolytic streptococcus (GABHS) harbor the organism in their nose or throat but display no symptoms of acute infection. The incidence of streptococcal carriers in general pediatric practice is a controversial issue. Estimates of their number vary with the study population and with the methods used. Since diagnosis of GABHS throat infection is based in large part on the finding of the organisms with an appropriate culture in the sick patient, differentiation between the carrier and the individual with bona fide illness is of importance. In this study we sought to determine the incidence of GABHS carriers in well children, and in children seen with both presumed and documented viral illnesses with sore throat. We also examined the incidence of GABHS carriage following an appropriate treatment course for GABHS tonsillopharyngitis with 10 days of oral penicillin V potassium, oral cephalosporins, and oral macrolides.

Results: The incidence of GABHS carriers among 227 well children seen in our practice was 2.5%. Among the 296 children seen with sore throat of presumed viral etiology the incidence was 4.4%, and among the 87 children seen with sore throat and documented viral illness it was 6.9% (P = .16; Table 1). Two thirds of positive GABHS throat cultures in carriers were 3+ or 4+ and one third were 2+ or less for colony counts. No child older than 10 years was a GABHS carrier and the mean age of GABHS carriers was 6.7 ± 1.4 years. The 6 children with concurrent documented viral illness and GABHS isolation had viral cultures positive for adenovirus (n = 1),
**PATIENTS AND METHODS**

This was a prospective study (October 1996 through June 1997) conducted at the Elmwood Pediatric Group, which is a private practice group of board-certified pediatricians, located in the suburbs of Rochester, NY. Our patients are drawn from all socioeconomic groups, although predominantly middle- and upper-middle class families. Four study groups were defined for the purposes of this study. Group 1 included well children. For 31 consecutive weeks, on the same day of the week, approximately 10 randomly selected patients visiting Elmwood Pediatric Group for routine well-child care, allergy injections, or other nonacute illnesses had a throat swab of their oropharynx obtained for isolation of GABHS. Children with any illness referable to the upper respiratory tract were specifically excluded. Group 2 were children with sore throat and apparent viral upper respiratory tract infection (URI). Again, on the same day of the week for 31 consecutive weeks, the Elmwood Pediatric Group care providers collected nose and throat viral cultures from approximately 10 children as a sentinel practice for the National Institutes of Health epidemiological surveillance program under a Vaccine and Treatment Evaluation Unit contract with the University of Rochester. Group 3 included children from group 2 whose viral culture was positive, representing a subset of group 2. Group 4 were children presenting with sore throat, positive rapid antigen detection test results or positive culture confirmed as due to GABHS infection, and who were treated for 10 days with oral penicillin V potassium, oral cephalosporins, or oral macrolides.

**LABORATORY METHODS**

Throat swabs for bacterial isolation were processed by routine in-office culture methods in our office laboratory (Clinical Laboratory Improvement Amendments, level 3 approved, 1992). For patients in group 4, if a rapid GABHS antigen detection test result was positive, then a throat culture was not taken and the rapid test result was considered definitive evidence of the presence of GABHS infection. Throat swabs were plated on sheep blood agar, a bacitracin disk was placed on the inoculum, and the agar was stabbed in several areas. Plates were incubated aerobically at 35°C and examined for 2 successive days for the presence of β-hemolytic streptococci. Isolates with typical streptococcal colony morphologic features that were sensitive to bacitracin were classified as group A. Appropriate positive and negative quality-control cultures were plated daily. The GABHS colony counts were categorized as 4+ if more than 100 colony-forming units were present, and 3+ if 51 to 100, 2+ if 11 to 50, and 1+ if 1 to 10 colony-forming units were present.

**VIRAL CULTURE METHODS**

Nose and throat swabs were placed in viral transport media and stored at 4°C overnight. The next-day cultures were taken to the University of Rochester and processed according to standard techniques in the laboratory of Caroline Breese Hall, MD.

**SYMPTOMS AND SIGNS OF PRESUMED VIRAL URI**

Symptoms and signs of acute infection compatible with the diagnosis of presumed viral URI included sore throat, fever, headache, coryza, cough, malaise, myalgia, tonsillopharyngeal redness, swollen and/or tender anterior cervical lymph nodes, and chest rhonchi.

**SYMPTOMS AND SIGNS OF ACUTE GABHS INFECTION**

Symptoms and signs of acute infection compatible with the diagnosis of GABHS tonsillopharyngitis included sore throat, fever, headache, malodororous breath, facial flush, nausea, vomiting, tonsillopharyngeal redness and/or exudate, palatal petechiae, and swollen and/or tender anterior cervical lymph nodes. Treated patients were seen at follow-up visits 14 to 21 days after initiation of antibiotic therapy. During the follow-up visit, symptoms of GABHS throat infection were again solicited, a limited examination pertaining to the possibility of recurrent throat infection was undertaken, and a throat swab was obtained for routine culture of GABHS.

**ANTIBIOTIC TREATMENT**

Oral penicillin V potassium was administered for 10 days at a daily dose of 20 to 40 mg/kg, 2 or 3 times daily. Various oral cephalosporins were used, including first-, second-, and third-generation drugs, and dosing was according to manufacturers’ recommendations and continued for 10 days. Macrolide therapy consisted of a 10-day course of erythromycin estolate or erythromycin ethylsuccinate, or a 5-day course of azithromycin administered according to manufacturers’ recommendations. Compliance was not actively monitored.

**STATISTICS**

We used χ² analysis to assess differences in GABHS carriage rates among the study groups.

Contrary to the results of our study, others have found that the carrier rate for GABHS in apparently healthy chil-
The bacteriologic method used for isolation of GABHS also is relevant. Many earlier longitudinal surveys relied on highly sensitive methods that are not commonly used in practice. These included selective media, pour plates, and anaerobic or carbon dioxide incubation of plates. Such procedures increase the proportion of positive cultures that are often missed with simpler, standard office methods. The question arises whether less sensitive bacteriologic methods result in missed significant clinical disease. We suggest that an appropriate standard would be the methods of Denny et al, which established the value of penicillin therapy in eradication of GABHS from the tonsillopharynx as a primary prevention technique for acute rheumatic fever. In those studies, the culture methods were more similar to those routinely used in our office practice.

The GABHS carriers in our study population had higher colony counts than often reported; however, the relation between colony count and carrier status has been previously challenged. Although the children in group 2 (children with sore throat and apparent viral URI) appeared to have viral URIs, and typical symptoms and signs of GABHS infection were absent, we cannot exclude the possibility that some of these children had concurrent bona fide GABHS infection as well as viral infections.

There continues to be recognized disagreement with regard to the significance of the carrier state. Some investigators have regarded the presence of GABHS in the nasopharynx as a source of danger and suggest eradication is a desirable goal, while others have argued that given the vast number of school children who are regular carriers, it seems inappropriate to proceed on a course of attempting to eradicate GABHS since there is a marginal relationship to clinically significant illness and sequelae on a community-wide basis. The widely held notion that GABHS carriers are harmful to themselves and to others is not accurate. Kuttner and Krumwiede described a number of GABHS outbreaks caused by carriers at the Irvington House on the Hudson River, NY, prior to the advent of antibiotics. They showed in their institutional study setting that the carrier could be an important vector in these infections and that the frequency of spread and resulting disease was often characteristic of a specific strain of GABHS. Thus, between epidemics, although 10% of children were found to be carriers, often none showed a great tendency to contagiousness. However, during certain other periods of observation, major and minor outbreaks occurred usually by strains of GABHS that were new to the institution; several of these outbreaks were introduced by carriers. In a family setting, James et al showed that children with clinical illness infect other members of the family approximately 25%
of the time, whereas children who are carriers infect other members of the family about 9% of the time. Asymptomatic spread of GABHS from a streptococcal carrier within a family has been described to occur. The same GABHS strains that are responsible for invasive, toxic shock and necrotizing fasciitis infections may be prevalent among carriers and patients with symptomatic pharyngitis in a community, transmission of these strains from the carrier reservoir to the latter groups of patients likely occurs.

Since we did not M and T type our strains in this study, we are unable to comment on strain variation presented in our carrier population. In the acute GABHS–infected patients who received antibiotic treatment, acquisition of a new strain vs persistence of the original infecting strain may occur. When serotyping of strains is done, our group and others have previously shown that after antibiotic treatment, about two thirds of the isolates will be the same serotype and one third will be new acquisitions.

We elected not to measure antistreptolysin O and anti-DNase B titers in our study population. In different study populations, Kaplan et al found that streptococcal antibody rises were observed in only 5% of asymptomatic individuals with positive cultures for GABHS, and Moffett et al found that about 5% of individuals who are asymptomatic and who have no GABHS isolated on throat swabs show significant antistreptolysin O and/or anti-DNase B rises. Extrapolation from those studies to ours should be done cautiously since the study settings were different, but their results are suggestive that we might have found measurable antibody rises in about 5% of our asymptomatic population of carriers and in our culture-negative cohort. Antistreptolysin O and anti-DNase B antibody rises occur in approximately 60% of individuals following apparent acute GABHS infection, but that does not mean the resident carriers. Antibiotic treatment of GABHS throat infections suppresses the antibody response significantly, making the validity of the 60% estimate questionable. Some of the patients in the antibiotic-treated population of our study could have been carriers. However, based on the carrier rates in asymptomatic patients and patients with viral sore throats, it is unlikely that the number exceeded 5%.

It has been suggested that the superiority of treatment with cephalosporins (and perhaps macrolides) in comparison with penicillin in bacteriologic outcome following acute GABHS tonsillopharyngitis is due to enrollment of carriers in comparative studies and due to more effective eradication of the carrier state with the broader spectrum agents. Our results challenge this notion. The carrier rate in our practice is 2.5% to 6.9% and to 25% higher eradication rate after cephalosporin and azithromycin treatment as compared with penicillin. Thus, our results show that the GABHS carrier state follows penicillin treatment more often than following cephalosporin or macrolide treatment, but other, much larger factor(s) must contribute to differences in eradication rates among these therapies.

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**Announcement**

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