STAPHYLOCOCCUS AUREUS is a common pathogen observed in head and neck infections. Over the past 20 years, methicillin-resistant S aureus (MRSA) has become an important source of such infections. A study from the VA Medical Center in Dallas, Tex (1988), reported methicillin resistance in 61% of S aureus isolates. Patients with chronic or recurrent infections such as otitis media or sinusitis are at a higher risk of contracting MRSA as the result of repeated antimicrobial therapy. Thus, the practicing otolaryngologist should be acutely aware of this pathologic entity.

Early reports of MRSA focused primarily on nosocomial acquisition; however, the incidence of community-acquired MRSA (CA MRSA) infections has been increasing in recent years. These infections have been more commonly described in adult patients. Intravenous drug abusers, nursing home residents, and chronically ill patients have been identified as “high-risk” populations. Researchers have now noted an increase in CA MRSA in the pediatric population. At the University of Chicago Children’s Hospital, Chicago, Ill, the prevalence of CA MRSA infections in children without identified predisposing risk factors increased from 10 per 100 000 admissions in 1988 through 1990 to 259 per 100 000 admissions in 1993 to 1995. More recent data by the same group in a follow-up, prospective assessment performed in 1998-1999 show this incidence remaining high.

Resistance to penicillins is presumed to be independent of the development of macrolide resistance. Nosocomial strains of MRSA tend to be uniformly resistant to macrolides, such as clindamycin and erythromycin, making vancomycin the drug of choice. However, a 1999 study observed a high correlation between CA MRSA infections and clindamycin susceptibility. Isolates that were susceptible to clindamycin were usually...
susceptible to erythromycin as well. Other studies reported similar patterns of susceptibility in hospitalized children and recommended treatment of CA MRSA with clindamycin because of high percentages of susceptible organisms.12,16 In contrast to vancomycin, which is a large molecule and does not penetrate areas of poor blood supply,17 clindamycin is thought to be concentrated in phagocytes,18 which subsequently transport the drug to the site of infection.

A recent study alluded to the changing susceptibility of CA MRSA and found that while 93% of CA MRSA cases were susceptible to clindamycin, only 64% were susceptible to erythromycin.19 This showed increasing resistance to erythromycin compared with earlier studies. The present study was prompted by an increase in the number of pediatric CA MRSA cases seen at our institutions and the emergence of MRSA isolates that were clindamycin sensitive but erythromycin resistant.

METHODS

The Otolaryngology Department at the University of Texas Health Science Center, Houston, identified 7 pediatric patients (age <18 years) with CA MRSA head and neck infections at 2 tertiary level teaching institutions between June 21, 2001, and October 4, 2001. All cases were discovered spontaneously through requested consultations; no specific or addi-
resistant pattern was observed in 6 (86%) of the 7 isolates (inducible MLS\textsubscript{B} resistance).

All infections resolved with the prescribed antibiotic treatments presented in Table 1. Of the 7 patients, 5 were initially treated with intravenous antibiotics, which included clindamycin, vancomycin, and rifampin, and 6 were discharged with oral antibiotic treatments. One patient who was not discharged with an oral antibiotic regimen had been treated with a definitive, 2-week course of intravenous antibiotics. Three patient’s oral regimen included trimethoprim-sulfamethoxazole and rifampin; the other 3 were treated with oral clindamycin. Two cases of erythromycin-resistant, clindamycin-sensitive MRSA treated with oral clindamycin alone resolved without complication.

The incidence of CA MRSA infections is increasing. Gottlieb et al\textsuperscript{19} documented 15 head and neck cases of CA MRSA in 1992, accumulated over a period of 3 years. Our 7 patients were identified in the course of 4 months. In keeping with previous reports,\textsuperscript{5,7,10,12} most cases were soft-tissue infections. However, one of the patients presented with acute mastoiditis, which underscores the broad spectrum of infections this pathogen can produce.

Most MRSA strains identified in our study were clindamycin sensitive and erythromycin resistant (6 [86%] of the 7 cultures). Previous authors have reported most strains to be clindamycin and erythromycin sensitive and advocated treating CA MRSA infections with clindamycin.\textsuperscript{10,12,17,18} The present study found only 1 (14%) of the 7 cultures to be susceptible to both clindamycin and erythromycin.

Macrolide-lincosamycin resistance in clinical isolates has been recognized for several decades.\textsuperscript{22} Methylation of an adenosine residue of bacterial 23S ribosomal RNA is one of the most common mechanisms of acquired resistance to macrolides (ie, erythromycin), lincosamides (ie, clindamycin), and streptogramin-B and confers cross-resistance to all MLS\textsubscript{B} antibiotics (the MLS\textsubscript{B} phenotype). The methylated RNA has a lower affinity for MLS\textsubscript{B} antibiotics than unmethylated RNA, and thus the antibiotics are unable to efficiently inhibit protein translation. This resistance is plasmid mediated, and the resistance is encoded on transposons.

Expression of MLS\textsubscript{B} resistance in staphylococci is either constitutive or inducible. When it is constitutive, bacteria exhibit resistance to all MLS\textsubscript{B} antibiotics. When resistance is inducible, staphylococci are resistant to 14- and 15-membered macrolides, such as erythromycin, but retain susceptibility to 16-membered macrolides and lincosamides, such as clindamycin. This dissociated resistance arises from differences in inducing capacities of MLS\textsubscript{B} antibiotics. Erythromycin is a more effective inducer of MLS\textsubscript{B} resistance than is clindamycin.\textsuperscript{21} The phenotype of “erythromycin-resistant, clindamycin sensitive” is indicative of inducible MLS\textsubscript{B} resistance.

Panagea et al\textsuperscript{24} studied the rate at which staphylococci developed resistance to clindamycin using strains resistant to erythromycin and sensitive to clindamycin. Resistance developed rapidly in vitro, leading them to rec-
REFERENCES


