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Stevens-Johnson Syndrome in a Boy With Macrolide-Resistant *Mycoplasma pneumoniae* Pneumonia

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KEY WORDS

Mycoplasma pneumoniae, Stevens-Johnson syndrome, drug resistance, pneumonia

ABBREVIATIONS

SJS—Stevens-Johnson syndrome

Ig—immunoglobulin

PCR—polymerase chain reaction

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abstract

Mycoplasma pneumoniae is a highly specialized parasitic bacterium that is a significant cause of community-acquired pneumonia in children. Although most such respiratory infections are mild, a minor percentage of patients require hospitalization and, occasionally, intensive treatment for respiratory failure. A variety of extrapulmonary sequelae of *M pneumoniae* infections have been described, including Stevens-Johnson syndrome. Macrolide resistance in *M pneumoniae* has developed rapidly in Asia, particularly in China, over the past decade and is now appearing in the United States. Emerging resistance to macrolides creates a therapeutic conundrum, particularly for pediatricians caring for young children in whom absolute or relative contraindications exist for the use of tetracyclines or fluoroquinolones, the 2 other main classes of drugs shown to be efficacious for *M pneumoniae*. We describe here the case of a child with a prolonged febrile illness associated with Stevens-Johnson–like mucocutaneous involvement who was found to have a respiratory infection with macrolide-resistant *M pneumoniae*. *Pediatrics* 2011;127:e000

A marked increase in resistance to macrolides has occurred with *Mycoplasma pneumoniae* in Asia over the past decade, particularly in China. Recent reports have documented that up to 90% of *M pneumoniae* isolates in China are now highly resistant to macrolides; resistance was recently documented in the United States and may be found in up to 27% of cases.^{1–3} Macrolide resistance in this common respiratory pathogen should be of interest to pediatricians because of the lack of alternative antibiotics available to treat infections in young children. Extrapulmonary manifestations of *M pneumoniae* infection have been described, including arthritis, encephalitis, and Stevens-Johnson syndrome (SJS).^{4–6} Here we present a case of SJS in a child after a respiratory infection with macrolide-resistant *M pneumoniae*.

CASE REPORT

A previously healthy 10-year-old boy developed 2 to 3 weeks of malaise and upper respiratory infection symptoms, then worsening cough and fever to 39°C, followed ~2 days later by worsening sore throat, mouth ulcers, and anorexia. He then developed a progressive bullous rash that began on his hands and feet and became generalized. He was admitted briefly to a local hospital and then transferred to the University of South Alabama in Mobile, where he was diagnosed with SJS and pneumonia on the basis of his mucocutaneous findings and an ill-defined left lower-lobe infiltrate on his admission chest radiograph. He continued to have cough without respiratory distress or supplementary oxygen requirement. *M pneumoniae* infection was strongly considered in view of the clinical history and presentation, and he was treated with intravenous azithromycin and ceftriaxone along with 3 doses of intravenous immunoglobulin (Ig) (1 g/kg) and morphine for severe pain. When the patient's skin and mu-



FIGURE 1

Mucocutaneous lesions caused by *M pneumoniae* infection. A, Mucus membrane involvement; B, extensive deep bullae on the palms, showing some healing and scattered fresh lesions; C, larger bullae scattered across the buttocks, occasionally becoming confluent; D, close-up of tense bullae on an extremity.

cus membrane disease continued to worsen, he was transferred on the third hospital day to Children's Hospital in Birmingham, Alabama, ~10 days after the onset of fever.

Past Medical/Family/Social History

The patient had been generally healthy. There was no family history of frequent infections. Two weeks earlier, the patient's younger brother had had "bronchitis" with fever to 39°C.

Physical Examination

After transfer to Children's Hospital in Birmingham, his examination revealed a florid mucocutaneous dermatosis (Fig 1). There were multiple erosions over his tongue and hard palate and a large bulla covering the mucosal surface of his lower lip. The conjunctival membranes were injected, and there were small erosions on the pinna of the right ear. Numerous tender, tense bullae on an erythematous base were noted on his arms, legs, palms and soles. Over the buttocks they coalesced to form annular, targetoid, and polycyclic lesions. There were a few typical target lesions on his palms.

In addition, bullae were present on the shaft of his penis (Fig 2), and mild erythema and desquamation were noted around his urethral meatus.

Continued Hospital Course

Members of the ophthalmology department and the burn unit surgical nursing team were consulted. Skin biopsies were performed in the dermatology department on hospital days 3 and 6 and revealed a subepidermal blister with acute inflammation suggestive of linear IgA bullous disease. However, there was no deposition of IgA, IgG, or IgM at the basement membrane zone. Fever and new evolving skin lesions continued for 1 week after admission. By hospital day 7 the ophthalmology consultant believed that there was some evidence for mild anterior uveitis bilaterally, possibly related to blisters of the bulbar conjunctivae, and recommended topical tobramycin/corticosteroid drops. The patient required a nasogastric tube for nutrition and a morphine patient-controlled anesthesia pump for pain control.



FIGURE 2
Cutaneous ulcerations on the shaft of the patient's penis.

Laboratory Studies

The patient's initial chest radiograph after transfer to Children's Hospital in Birmingham revealed a tiny left effusion, whereas a second chest radiograph 6 days later revealed only some peribronchial cuffing. A complete blood count and differential revealed only a mild left shift. Serum IgG and IgM levels were moderately elevated; the former was likely attributable to his 3 intravenous Ig infusions in Mobile; follow-up measurements 18 months after discharge revealed serum IgM and IgG levels below normal for the patient's age (Table 1) (all Ig levels were measured in the Children's Hospital clinical laboratory by using a Vitros Fusion 5.1 analyzer [Randox Laboratories, Kearneysville, WV]). Results of tests for serum IgM, IgA, and IgG antibodies to *M pneumoniae* were all positive (IgG: 3.69 ISR [positive: >1.1 ISR]; IgA: 63 BU/mL [positive: >21 BU/mL]; IgM: 4715

U/mL [positive: >950 U/mL]), and although a throat culture tested negative for *M pneumoniae*, results of a real-time polymerase chain reaction (PCR) assay targeting the *repMp* gene were positive.⁷ A real-time PCR assay designed to detect macrolide resistance in *M pneumoniae*⁸ revealed that the organism was positive for the A2063G mutation in the 16S ribosomal RNA gene, which has been associated with resistance.

Continued Hospital Course and Discharge

On the seventh hospital day in Children's Hospital in Birmingham, he was started on oral prednisolone (2 mg/kg per day), dapsone (25 mg/day), and levofloxacin (400 mg daily); he subsequently improved and was discharged on the 12th day. Permission for publication of this case report was obtained from the University of Alabama at Birmingham institutional review board, the patient, and his parents.

DISCUSSION

Review of *M pneumoniae* Infections

M pneumoniae is a cell-wall-less highly specialized parasitic bacterium that infects the ciliated epithelium of

the human respiratory tract. Infection spreads primarily by airborne droplets, whereby the organisms adhere to the respiratory epithelium via a specialized adhesive tip and then spread slowly through the respiratory tract; the incubation period is often as long as 3 weeks. Symptoms may last for several weeks but usually ameliorate with antibiotic treatment.⁹

M pneumoniae-induced SJS with bullous skin lesions and mucocutaneous ulcerations has been known for decades and is the most common infection-associated cause of this condition.^{10–20} Some reports have documented isolation of the organism from skin lesions, which suggests that SJS represents true extrapulmonary spread of the active infection, at least in some instances.^{21,22} As with the respiratory infections, *M pneumoniae*-induced SJS can be recurrent in some people.²³ Because extrapulmonary spread of *M pneumoniae* has been reported in hypogammaglobulinemic patients,^{24,25} it is interesting that the patient's follow-up serum IgG and IgM levels were low (Table 1).

Macrolide Resistance in *M pneumoniae*

Historically, macrolides have been the treatment of choice for pediatric *M pneumoniae* infections because of the potential toxicities of fluoroquinolones and tetracyclines in young children. Macrolide resistance has been detected with increasing frequency in Japan and China as a result of selective pressure attributable to widespread use of macrolide antibiotics for treatment of respiratory infections.^{26–28} The appearance of resistance in France and Germany has also been documented within the past 3 years.^{29,30} According to recent studies, >80% of *M pneumoniae* infections in Shanghai and 90% in Beijing are currently caused by macrolide-resistant

TABLE 1 Serum Ig Levels

	IgM, U/mL	IgA, BU/mL	IgG, ISR
During illness	318 (H)	162	2082 (H)
2-mo follow-up	—	—	705
18-mo follow-up	40 (L)	74	581 (L)
Normal range	52–242	45–236	608–1572

H indicates high; L, low.

strains.^{2,26} Although the current prevalence of resistance in the United States is unknown, a Centers for Disease Control and Prevention–based investigation of a 2006–2007 outbreak of *M pneumoniae* respiratory illness associated with 3 cases of encephalitis found that 3 of 11 isolates (27%) were macrolide-resistant, and we have documented the occurrence of macrolide-resistant *M pneumoniae* in hospitalized children in Alabama.^{3,8}

Diagnosis

Clinically useful diagnostic tools for the detection of *M pneumoniae* infection are presently limited to serologic assays and nucleic acid–based amplification systems, which generally use PCR technology.⁴ Culture is relatively insensitive and can take up to 6 weeks. Positive serologic test results at least 1 week into the course of a significant illness provide supportive evidence, but because of the prevalence of IgG titers in the population, antibodies obtained at a single time point may not be indicative of acute infection. Furthermore, early in the course of infection, patients may not have made sufficient antibody to yield a positive result, older adults may not produce an IgM response, and IgM may persist in some people for years.^{6,31,32} Immunodeficient people may not be capable of mounting an antibody response at all.

PCR, particularly real-time PCR, has been the most important innovation in the rapid, accurate diagnosis of *M*

pneumoniae infections and in the determination of antibiotic resistance.^{3,8} The high levels of sensitivity and specificity are offset by the well-known propensity for false-positive results caused by contamination, issues that are under intense scrutiny as the development of DNA-based point-of-care diagnostic tests continues.⁴

Therapy

Treatment with antibiotics from the penicillin or cephalosporin families, the drugs most commonly used by physicians for the treatment of respiratory infections, has no effect on mycoplasmas because of the absence of a cell wall.³³ Antibiotic therapy with the bacteriostatic antibiotics from the macrolide or tetracycline classes improves respiratory symptoms, but the organisms may still be cultured from the respiratory tract even after treatment, sometimes for months.^{34–37} It is important to note that tetracyclines are contraindicated in children younger than 8 years because of permanent staining of the developing teeth. Ketolide antibiotics have some efficacy against *M pneumoniae*,³⁸ but no ketolides are currently available for use in children in the United States. Fluoroquinolones are effective clinically, but none of them are currently approved for pediatric use, and these drugs must be used with caution because of the possibility of toxicity or adverse effects. Clinical efficacy of other antibiotic classes has not been demonstrated.

Corticosteroids have been advocated for adjunctive use in severe *M pneumoniae* infections,^{39–42} their potential efficacy perhaps related to suppression of immunologic hypersensitivity^{6,43,44} and/or a direct inhibitory effect on the growth of the organism.⁴⁵ There is also supportive experimental evidence from animal models.⁴² However, the use of corticosteroids in *M pneumoniae*–associated SJS has not been well studied because the complication is infrequent. There have been scattered case reports suggesting that doses such as those that may be used for respiratory infection with *M pneumoniae* may be efficacious.²⁰ Current recommendations suggest that, if used in severe cases, intravenous solumedrol at 2 to 2.5 mg/kg per day should be started early in the course of the disease.¹⁰

CONCLUSIONS

M pneumoniae should be considered in patients with SJS, particularly those with an associated respiratory infection. As has been documented in patients with *M pneumoniae* arthritis, mild humoral deficiencies may be a risk factor for *M pneumoniae*–associated SJS, a subject that deserves further study. Emerging macrolide resistance in *M pneumoniae* is likely to present an increasingly significant therapeutic problem for clinicians in the treatment of complicated illness caused by *M pneumoniae* in young children for whom the use of tetracyclines and fluoroquinolones is not normally considered.

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