To the Editor:
A 9-year-old girl was admitted with fever, fatigue, cough, eye and mouth pain, and scattered cutaneous pustules that rapidly progressed to painful erosions and bullae involving the conjunctivae, genitals, lips, and body. The patient was hospitalized 18 months prior to presentation with similar symptoms; she was diagnosed with Stevens-Johnson syndrome (SJS) secondary to *Mycoplasma pneumoniae* infection, which was confirmed by polymerase chain reaction (PCR) analysis. She also reported several mild self-limiting episodes of lip crusting and pustular rash prior to admission with no history of concomitant medication use. Physical examination revealed an ill-appearing febrile child with ulcerations involving the oral mucosa and genitalia, notable conjunctival erythema, and targetoid bullae and erosions scattered on her face and body (Figure 1). Herpes simplex virus PCR, respiratory virus PCR, blood culture, and antistreptococcal antibody titers were negative. Skin biopsy demonstrated a subepidermal blister with interface dermatitis (Figure 2). Three oropharyngeal *Mycoplasma* PCR samples were negative, though samples were suboptimal due to remarkable oral pain. *Mycoplasma* IgM and IgG titers were elevated, suggestive of an acute infection. A chest radiograph revealed middle lobe pneumonia. With these findings, a diagnosis of recurrent SJS secondary to *M pneumoniae* infection was made. She was treated with supportive care and intravenous immunoglobulin at a total dose of 4 g/kg over 3 days. Follow-up 2 weeks after discharge demonstrated interval resolution of bullae and erosions with hyperpigmentation.

The association of *M pneumoniae* infection with SJS is well-established. Typically, *Mycoplasma*-induced SJS affects children and young adults, with males more commonly affected. Cutaneous manifestations include erosions, erythematous plaques, target lesions, and bullae, while mucosal involvement includes ocular, oral, and urethral erosions. A mucosal-predominant variant has been reported.  

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**Figure 1.** Diffusely distributed targetoid bullae with erosions, desquamation, and postinflammatory hypopigmentation with substantial mucosal involvement of the face (A) and arm (B).

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The authors report no conflict of interest.

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Although cutaneous manifestations typically resolve with postinflammatory dyschromia, conjunctival involvement may lead to corneal scarring, adhesions, and decreased visual acuity.

Recurrent SJS due to *Mycoplasma* infection rarely has been reported in the literature, with few documented cases in children.\(^2\)\(^-\)\(^5\) The pathophysiology of *Mycoplasma*-associated SJS is not known; however, in respiratory epithelium, *M pneumoniae* infection is known to stimulate a cell-mediated immune response and cytokine production, leading to a cytotoxic response.\(^6\) Mitogenic stimulation of B lymphocytes and T lymphocytes as well as the production of circulating immune complexes also occurs and is associated with well-characterized autoimmune phenomena, including the production of cold agglutinins and development of neurologic complications (eg, Guillain-Barré syndrome).\(^6\) Protective immunity to *Mycoplasma* may not develop, leading to reinfec tion; thus reexposure to *Mycoplasma* without protective immunity may result in recurrence in patients who are immunologically predisposed to developing SJS.

Confirmation of *M pneumoniae* infection in the appropriate clinical setting is best accomplished by use of serologic tests and PCR. Diagnosis of *M pneumoniae* infection can be complicated by testing limitations, including the relatively poor sensitivity and specificity of serologic tests, especially early in infection and by limited availability of PCR. This limitation may account for the paucity of reports of recurrent *Mycoplasma*-associated SJS in the literature. Appropriate samples for the detection of *Mycoplasma* include nasopharyngeal or oropharyngeal swabs or aspirates and sputum; however, in cases of substantial mucosal involvement in SJS, as in our patient, obtaining an adequate specimen is difficult. Clinicians should recognize the possibility of recurrence of SJS to provide appropriate evaluation and therapeutic intervention. Therapeutic regimens for SJS include intravenous immunoglobulin, systemic corticosteroids, wound care, and prevention of skin infection; additionally, suppressive therapies such as oral corticosteroids or dapsone may be considered with recurrence.

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REFERENCES

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**Figure 2.** A subepidermal blister with associated interface dermatitis, including vacuolar degeneration of basal layer keratinocytes and dyskeratotic keratinocytes within the lower layers of the epidermis. Full-thickness epidermal necrosis with an orthokeratotic stratum corneum also was observed (H&E, original magnification ×40 [inset in bottom left corner, original magnification ×400]).