

A Case of Papular-purpuric “Gloves and Socks” Syndrome Caused by *Mycoplasma pneumoniae*

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ARTICLE HISTORY

Received: April 7, 2019

Accepted: August 28, 2019

ABSTRACT

We present a case of “gloves and socks” syndrome associated with *Mycoplasma pneumoniae* infection in a 6-year-old child hospitalized for febrile syndrome associated with monomorphic purpuric papular eruption localized on the distal part of extremities, in a “gloves and socks” pattern. Clinical diagnosis was confirmed by positivity of specific IgM against *Mycoplasma pneumoniae*. Favorable outcome was obtained by administration of oral clarithromycin.

Keywords: purpuric papular eruption, “gloves and socks”, *Mycoplasma pneumoniae*

INTRODUCTION

The “gloves and socks” syndrome (GSS) is an acute, rare, highly contagious, purpuric and papular eruption of the hands and feet, mostly described in children and adolescents,¹ caused by parvovirus B19, Coxsackievirus B6, cytomegalovirus (CMV), and Epstein-Barr virus (EBV).^{2,3} We present a case of GSS associated with *Mycoplasma pneumoniae* (*M. pneumoniae*) infection in a young child treated with oral clarithromycin.

CASE PRESENTATION

A 6-year-old boy hospitalized in the Pediatric Hospital for febrile syndrome had an eruption of the hands and feet. Dermatological examination revealed a monomorphic purpuric papular and erythematous eruption localized in the distal part of the extremities, in a “gloves and socks” pattern (Figure 1 A, B).

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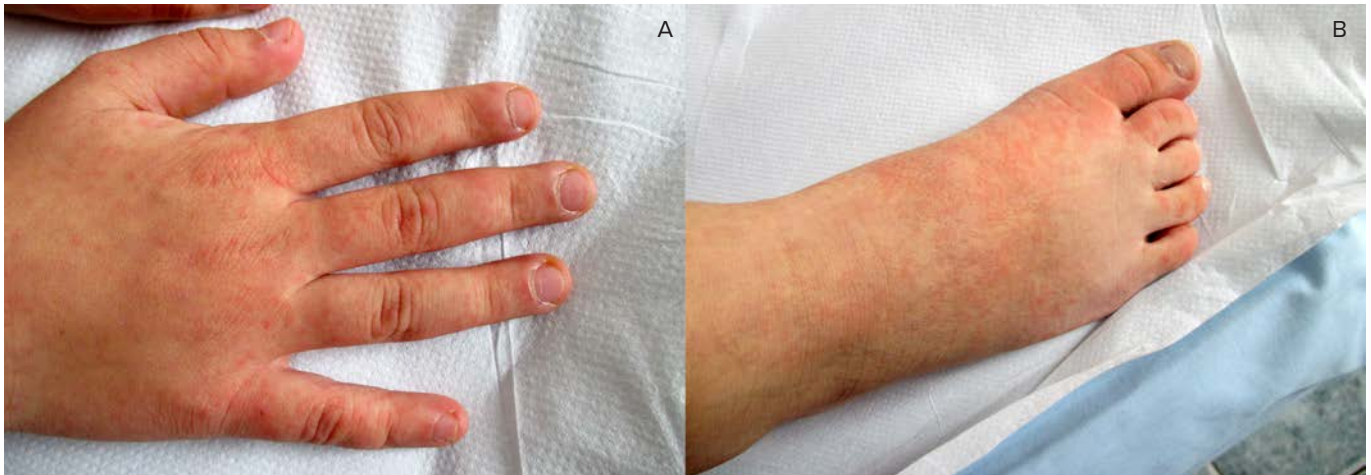


FIGURE 1. Purpuric papular eruption localized on the dorsal face of the hands (A) and feet (B).

The child complained of slight pruritus, anorexia, and asthenia. Clinical examination excluded systemic involvement or lymphadenopathy.

A clinical suspicion of GSS was raised, and laboratory investigations were performed. A skin biopsy was not taken due to the young age of the child and lack of compliance from family members.

Abnormal laboratory values included: leukocytosis (leukocyte count 10,330/ μ L), neutrophils 5,350/ μ L, negative serological levels for IgM and IgG against parvovirus B19, EBV, and CMV. The specific IgM against *M. pneumoniae* was positive (25.03 U/mL), coupled with IgG negativity. A 7-day course of oral clarithromycin was recommended, which was followed by a favorable patient outcome. In most cases of GSS, the disease cures itself; symptomatic treatment is rarely necessary; the fever and arthralgia are usually treated with nonsteroidal anti-inflammatory drugs. Informed consent for the publication of the images was obtained from the boy's mother.

DISCUSSIONS

We hypothesize a causal association between acute *M. pneumoniae* infection and GSS in this case. *M. pneumoniae* causes atypical pneumonia, tracheobronchitis, pharyngitis, and asthma, especially in children and elderly.⁴ *M. pneumoniae* can cause extrapulmonary manifestations, affecting the skin, cardiovascular, hematologic, musculoskeletal, and nervous systems due to systemic bacterial dissemination or secondary to autoimmune reactions.⁵ Skin manifestations, such as exanthemas, erythema nodosum, urticaria, Stevens-Johnson syndrome, and *M. pneumoniae*-associated mucositis have been reported in almost 25% of all *M. pneumoniae* infections.⁶

Asymptomatic carriage of *M. pneumoniae*, without any type of disease, varies accordingly to the study and period of time; for example between 2009 and 2010 the interval was 3% to 58%, probably related to cyclic epidemics of *M. pneumoniae* infections.⁷ Asymptomatic children were found to be carriers of various pathogens in their nose and throat: *M. pneumoniae*, *S. pneumoniae*, *Staphylococcus aureus*, *Moraxella catarrhalis*, *Haemophilus influenzae*, influenza A/B viruses, human metapneumovirus, respiratory syncytial virus, parainfluenzavirus, rhinovirus, coronavirus, bocavirus, and adenovirus.^{8,9}

The presence of *M. pneumoniae* in the upper respiratory tract does not confirm the diagnosis of a *M. pneumoniae* infection. According to the latest guidelines, only PCR and serological tests are of great value in proving the bacterial infection as a causative agent of the disease.¹⁰ Detection of specific IgM starts one week after the initial infection and persists for two weeks before IgG detection. Positive results may be encountered in asymptomatic carriers, and the infection rate may be overestimated. The diagnosis of *M. pneumoniae* requires a fourfold increase in antibody titer, but in daily practice this needs time, thus delaying initiation of treatment. Clinicians should make their decision based on clinical grounds, positive PCR, and serology test results.

According to the majority of reports found in the literature, GSS affects children and is caused mainly by parvovirus B19 infection.

CONCLUSIONS

The present case of GSS, diagnosed clinically, could have been induced by *M. pneumoniae* and represents an unusual case developed in a young child; we identified only one other case of bullous papular purpuric "gloves and socks"

syndrome resulting from *M. pneumoniae* infection. The diagnosis is based on clinical grounds, and laboratory confirmation is rarely done in daily practice in immunocompetent patients by detection of specific immunoglobulin M antibodies and excluding other etiological factors.

CONFLICT OF INTEREST

Nothing to declare.

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