The role of *Mycoplasma pneumoniae* in dermatological diseases

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ABSTRACT

Mycoplasma pneumoniae is an atypical bacterium causing respiratory tract infections mainly in the pediatric population. As a superantigen, it dysregulates the immune system and promotes immunological reactions. Dermatological symptoms occur in approximately one-fourth of the patients infected with this bacterium. This review describes skin diseases occurring during *Mycoplasma pneumoniae* infection. Differences in the course of these diseases compared to their presentation associated with other etiological factors, are also discussed. Among the cutaneous manifestations of *Mycoplasma pneumoniae* infection, unspecific rashes and urticaria are the most common. This bacterium is also a frequent cause of erythema multiforme, Stevens-Johnson syndrome, *Mycoplasma*-induced rash and mucositis, and erythema nodosum. Less frequently toxic epidermal necrolysis, leukocytoclastic vasculitis, IgA vasculitis, subcorneal pustular dermatosis, Gianotti-Crosti syndrome, and Sweet syndrome are described. Familiarity with *Mycoplasma*-induced entities is important and can be useful in dermatological practice in determining the etiology and implementing appropriate treatment.

Key words: *Mycoplasma pneumoniae*, urticaria, erythema multiforme, Stevens-Johnson syndrome, *Mycoplasma*-induced rash with mucositis, erythema nodosum, leukocytoclastic vasculitis, IgA vasculitis, subcorneal pustular dermatosis, Gianotti-Crosti syndrome, Sweet syndrome

INTRODUCTION

Mycoplasma pneumoniae (*MP*) is an atypical bacterium that primarily causes respiratory tract infections. It is responsible for approximately 40% of cases of community-acquired pneumonia in children over the age of 5 [1]. Exposure to *MP* in the majority of cases leads to asymptomatic carriage, while symptomatic infections most commonly manifest as tracheobronchitis appearing 2–3 weeks post-incubation. Low-grade fever, headache, and dry cough are typical presenting symptoms and may persist for over 2 weeks. Infections may also present as primary atypical pneumonia. The clinical course is generally mild [2]. Antigen tests, designed for rapid diagnosis, exhibit low sensitivity and specificity, while species-specific polymerase chain reaction (PCR) tests have excellent sensitivity but low specificity. The most popular method is serological testing for IgG and IgM antibodies. A rise in antibody titers should be demonstrated early in the infection and 3–4 weeks later. *MP* is susceptible to macrolides, tetracyclines and fluoroquinolones, all equally effective in treatment. Preventing *MP* infections is difficult because patients remain infectious for a long time even during antibiotic therapy. The immune response following infection is weak, and an effective vaccine has yet to be developed [2, 3]. As a superantigen, *MP* causes dysregulation of the immune system, which predisposes to immune reactions, often with dermatological manifestations [2]. Skin symptoms occur in 10–25% of

patients infected with *MP* and include: non-specific rashes, urticaria, erythema multiforme, Stevens-Johnson syndrome, MIRM (*Mycoplasma*-induced rash and mucositis), erythema nodosum and less frequently: toxic epidermal necrolysis, leukocytoclastic vasculitis, IgA vasculitis, subcorneal pustular dermatosis, Gianotti-Crosti syndrome or Sweet syndrome. Skin diseases may occur during active or asymptomatic infection. In some cases, more than one cutaneous manifestation may be present [4].

NON-SPECIFIC RASHES

The most common dermatological manifestation of *MP* infection is a non-specific maculopapular exanthem, which typically resolves spontaneously and does not require treatment. It occurs in 8–33% of patients with the *MP* respiratory tract infection [5]. In the study by Meyer Sauteur *et al.* involving 152 children with community-acquired pneumonia (CAP), dermatological manifestations occurred in 22.7% of patients with *MP*-induced CAP [6]. The median age of this group was 8.7 years. The duration of the rash ranged from 2 to 22 days. In 4 cases, therapy with amoxicillin was initiated, while in 1 case, no treatment was administered. The exanthema usually appears in the early days of the illness, initially as a single erythematous macule, which over a few days may spread to the limbs, face (especially the cheeks), palms, and soles. Skin lesions are accompanied by pruritus. The involvement of mucous membranes may be observed [7, 8]. Differential diagnosis includes varicella exanthem or scarlet fever [9, 10]. In a study involving 81 patients with *MP* infection, Macfarlane described the appearance of skin lesions in 15 (18.5%) cases [11]. However, the author suggested, that in the majority of patients, skin lesions were associated with the treatment rather than *MP* infection itself. It is hypothesized that the interaction between the pathogen and the administered medication increases hypersensitivity to the specific drug and leads to the development of drug-induced eruptions.

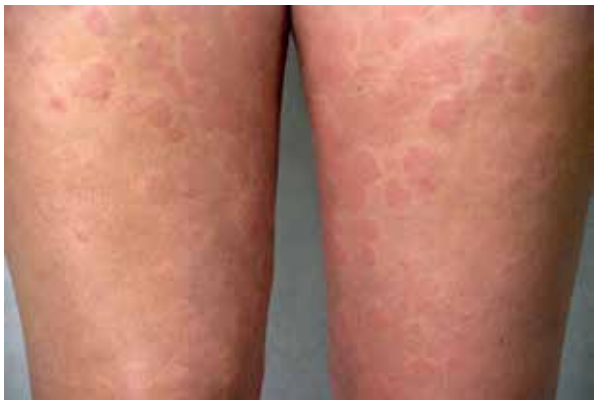

Figure 1. Acute urticaria

URTICARIA

Urticaria occurring due to *MP* infection is observed mainly in the pediatric population but can also appear in adults. *MP* is the cause of urticaria in 3.43% of cases [12]. In the study by Wu *et al.*, among 65 children with urticaria who were unresponsive to antihistamine treatment, 21 (32%) had serological symptoms of *MP* infection [13]. However, patients with *MP*-associated urticaria treated with antibiotics demonstrated faster improvement compared to the patients with urticaria unrelated to *MP*. The study by Yong *et al.* revealed that the risk of developing urticaria during *MP* infection did not significantly differ when compared to the cohort with *MP* infection excluded [14]. However, it was observed that in the 20–59 years age group, the incidence of urticaria was significantly higher in patients with *MP* infection. Positive serological tests may prompt the initiation of antibiotic therapy, potentially abbreviating the duration of urticaria (fig. 1).

ERYTHEMA MULTIFORME

MP is the second most common cause of erythema multiforme (EM) after *Herpes simplex virus* and was reported as the etiological cause in 7% of cases in children and 3.6% in adults with EM [15, 16]. Infections are responsible for 90% of EM cases. Other etiologies include medications and medical conditions such as malignancy and inflammatory bowel disease [17].

In one study, it was demonstrated that *MP* infection more frequently predisposed individuals to the major form of EM (14 out of 23 cases [61%]) compared to the minor form (4 out of 18 cases [22%]) and atypical lesions with central blistering were more frequently observed [18]. The authors suggested that patients with atypical eruptions should undergo diagnostic evaluation for *MP* infection, and if clinical history indicates infection, empirical antibiotic therapy should be initiated. In a retrospective study by Amode *et al.*, 33 patients with *MP*-induced erythema multiforme (*M. pneumoniae* EM) were compared with 100 patients with EM induced by other factors (non-*M. pneumoniae* EM) [19]. It was found that involvement of mucous membranes in more than 2 locations (particularly ocular, pharyngeal, and laryngeal changes) and longer average hospital stay (9.5 days compared to 5.1 days) were more common in the *M. pneumoniae* EM group. Complications involving the eyes (decrease in visual acuity, symblepharon, corneal scarring, dry eyes, ingrown eyelashes) and genital organs (phimosis, penile

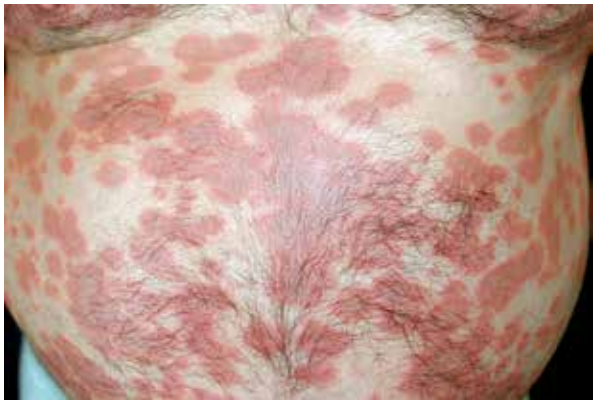

Figure 2. Erythema multiforme

and labial adhesions) were also more frequent in patients with *M. pneumoniae* EM (fig. 2).

STEVENS-JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS

MP is the predominant infectious etiology in Stevens-Johnson syndrome (SJS) and was reported in 22% of cases of SJS in children [18, 20]. In the study by Liew *et al*. from 180 patients with SJS, 3.33% had positive *MP* serologies [21]. SJS appears in 1–5% of *MP* infections [3, 4]. In *MP*-induced SJS, mucosal lesions involving the oral cavity, genitalia, and eyes are noted in 100%, 75%, and 66% of patients, respectively [4, 22]. Extensive ocular mucosal involvement has been demonstrated to be more prevalent in *MP*-associated SJS compared to drug-induced SJS [23] (fig. 3).

Currently, standardized treatment protocols for *MP*-induced SJS are lacking. Early initiation of systemic glucocorticosteroids concurrently with antibiotic therapy is proposed to potentially reduce hospitalization time and improve clinical outcomes in *MP*-related SJS cases [24]. Supportive treatment including nutritional optimization and pain management have been associated with enhanced patient satisfaction, albeit without concurrent reduction of hospital stay [25].

Although *MP* infection is a well-established trigger of SJS, toxic epidermal necrolysis (TEN) secondary to *MP* infection is rare. There are single reports where *MP* infection has been implicated as the causative agent for TEN [26, 27].

MIRM (MYCOPLASMA-INDUCED RASH AND MUCOSITIS)

MIRM (*Mycoplasma*-induced rash and mucositis) primarily affects children (mean age: 11.9 years), with a male predominance of 66% [28]. Among patients with MIRM, nearly half (47%) present with limited skin involvement, ranging from isolated to diffused

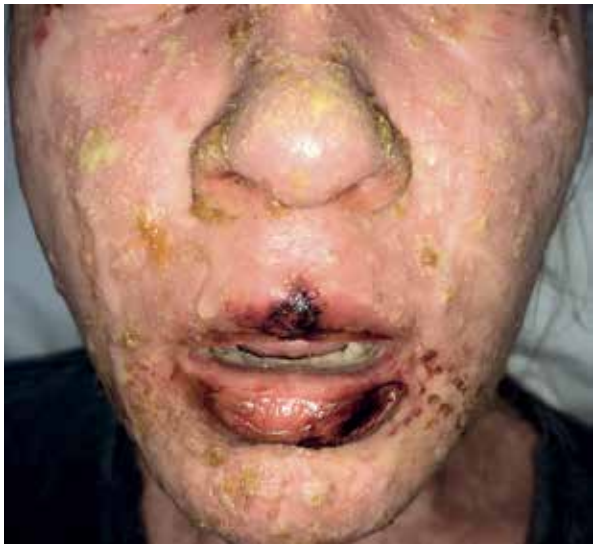

Figure 3. Stevens-Johnson syndrome progressing into toxic epidermal necrolysis (TEN)

lesions. Involvement of the oral mucosa, eyes, and genitourinary tract was reported in 94%, 82%, and 63% of cases, respectively [28] (fig. 4). Pulmonary symptoms preceded dermatological eruptions by 7.8 days on average [29].

Diagnostic criteria proposed for MIRM include: mucosal involvement in at least two locations; skin involvement in less than 10%; skin lesions presenting as few vesiculobullous or atypical scattered eruptions with or without targetoid lesions; laboratory confirmation of *MP* infection [29]. Diagnostic tests for active infection include: positive polymerase chain reaction (PCR) result from the throat swab and elevation of the immunoglobulin G (IgG) and M (IgM) level in two separately obtained samples [30]. Considering the extent of skin involvement, three subtypes of the disease are described: classic, MIRM sine rash, and severe MIRM. In the classic type, skin lesions commonly manifest as vesiculobullous eruptions (77%), scattered targetoid lesions (48%), papules (14%),

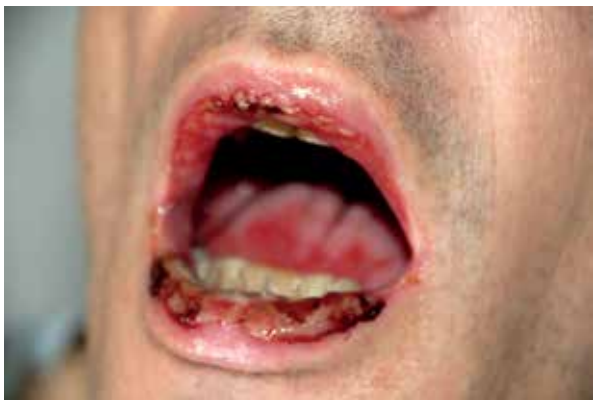

Figure 4. Mycoplasma-induced rash and mucositis (MIRM), oral involvement

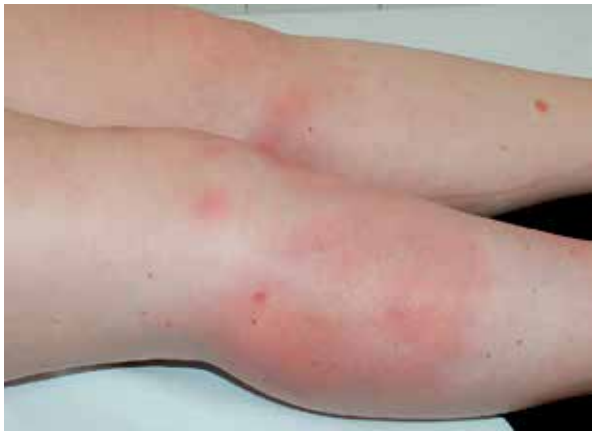

Figure 5. Erythema nodosum

macules (12%), or morbilliform eruptions (9%). The MIRM sine rash type lacks cutaneous involvement except for isolated vesicles. In severe MIRM skin lesions are extensive, predominantly presenting as blisters and atypical erythematous targetoid lesions [31].

Some cases of recurrent MIRM have been reported. Liakos *et al.* investigated differences in the course of isolated versus repeated MIRM [32]. Recurrences following the initial episode demonstrated less severe cutaneous and mucosal symptoms, often involving only one mucosal site, a rarer need for hospitalization, and a shorter hospital stay.

All patients with MIRM require mucosal care and correction of fluid and nutritional deficiencies. Lesions in specific locations should be consulted by an adequate specialist (e.g. ophthalmologist, otolaryngologist, gastroenterologist, or urologist). Analgesic therapy is necessary. Currently, there are no guidelines regarding routine antibiotic therapy for patients with MIRM. However, individuals diagnosed with MIRM often exhibit symptoms of atypical pneumonia, warranting antibiotic administration in such cases. Antibiotic options include: azithromycin, erythromycin (in all age groups), clarithromycin, and doxycycline (in children above 12 years of age). In patients with severe mucosal involvement, intravenous immunoglobulin (IVIG) therapy may be beneficial [31].

ERYTHEMA NODOSUM

Erythema nodosum is the most common type of subcutaneous tissue inflammation in childhood. *MP* is the cause of erythema nodosum in 8.6% of cases in the pediatric population [33], as the study by Kakourou *et al.* identified *MP* infection in three out of 35 children with erythema nodosum [33]. Whilst, in the adult population, *MP* is reported to be the cause of erythema nodosum in 0.8–3% of cases. Cribier *et al.* described 129 patients with erythema nodosum, with *MP* infection diagnosed in one patient (0.8%) [34], and Varas *et al.* identified *MP* infection as the fourth most common cause of erythema nodosum (following streptococcal infection – 32%, sarcoidosis – 11%, primary tuberculosis – 7%) among 91 patients, with *MP* etiology confirmed in 3% of cases [35]. It is noted that erythema nodosum often accompanies asymptomatic *MP* infection [36]. The most of cases described in the literature have been successfully treated with clarithromycin without recurrence. Antibodies against *MP* should be measured in children with erythema nodosum, even in the absence of respiratory system involvement [37] (fig. 5).

LEUKOCYTOCLASTIC VASCULITIS AND IgA VASCULITIS

The occurrence of leukocytoclastic vasculitis (LV) during *MP* infection is exceedingly rare [38–42]. It may be accompanied by encephalitis, retinal vasculitis, or nephritis [38, 39, 41]. In the study by Betti *et al.*, systemic symptoms were present in two-thirds of cases, while in one-third, the disease was limited to the skin [43]. LV associated with *MP* infection is observed more frequently in women than in men, unlike LV caused by other triggering factors. The most common type of skin vasculitis in children is IgA-associated vasculitis (IgAV) and *MP* was reported as the cause of IgAV in 9.1% of this population [44]. IgAV occurs in about 1% of pediatric patients with *MP* infection [44]. In the study by Shang *et al.*, among 10,519 children infected with *MP*, IgAV occurred in 131 (1.2%) cases [44]. Similarly, in the study by Timitilli *et al.*, IgAV was diagnosed in one among 92 *MP*-infected children (1.09%) [45]. Patients with *MP*-induced IgAV demonstrated significantly higher rates of renal involvement, abdominal pain, and joint inflammation. It was noted that vasculitis in *MP*-infected children lasted an average of 2.129 days longer than IgAV caused by other factors [44] (fig. 6).

SUBCORNEAL PUSTULAR DERMATOSIS (SNEDDON-WILKINSON DISEASE)

Subcorneal pustular dermatosis (SCPD) is a very rare dermatological manifestation of *MP* infection. Eight cases of SCPD associated with this pathogen have been described. Inflammatory changes in the lungs visible on the chest X-ray were observed in 7 out of 8 patients (87.5%). Seven (87.5%) patients had extensive pustular exanthema, mainly on the trunk, while mucous membrane involvement occurred in 3 (37.5%) patients. Patients underwent various therapeutic interventions, including: penicillin, ampicillin, erythromycin, prednisone,

dapsone, or cephalosporin. The majority of patients experienced remission within 2 weeks, without relapses [46]. The rapid and sustained remission observed in these cases distinguishes *MP*-induced SCPD from SCPD caused by other factors [46–54].

GIANOTTI-CROSTI SYNDROME

Gianotti-Crosti syndrome is a dermatosis characterized by pink-brown papular eruptions on the face, limbs, and buttocks with a symmetrical distribution [55]. The condition is more commonly associated with the viral infections, but *MP* infection can also be a causative factor [56, 57]. Two cases of Gianotti-Crosti syndrome induced by *MP* have been described. In addition to the characteristic skin lesions, in both patients symptomatic respiratory tract infections were also observed [56, 58].

ACUTE FEBRILE NEUTROPHILIC DERMATOSIS (SWEET SYNDROME)

It is speculated that *MP* infection may be one of the potential triggers for acute febrile neutrophilic dermatosis, although this etiology is not fully confirmed. Hsieh, Yalcindag, and Coghlin described the case of a 6-year-old patient with Sweet syndrome following *MP* infection [59]. Improvement was observed after 1 week of prednisone therapy.

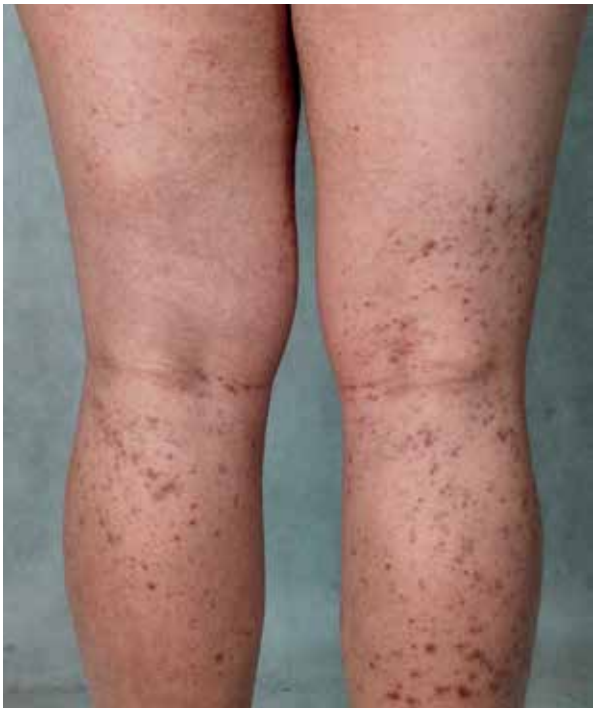

Figure 6. IgA vasculitis

Table 1. Cutaneous manifestations of Mycoplasma pneumoniae (MP)

Cutaneous manifestations	Incidence of MP as a causative factor	Differences between MP and other causative factors
Non-specific rashes	Adults 23% Children 8–33%	Single erythematous macules, which over a few days may spread to the limbs, face (especially the cheeks), palms, and soles; pruritus; involvement of mucous membranes may be observed
Urticaria	Adults 3.43% Children (with urticaria unresponsive to antihistamine treatment) 32%	Resistance to antihistamine therapy
Erythema multiforme	Adults 3.6% Children 7%	More frequent involvement of mucous membranes in more than 2 locations; longer hospital stay; higher rate of complications involving eyes and genital organs
Stevens-Johnson syndrome	Adults 3.33% Children 22%	More prevalent extensive ocular mucosal involvement
MIRM (Mycoplasma-induced rash and mucositis)	Adults 100% Children 100%	Extensive mucosal involvement (oral, eyes and genitourinary tract), variable erythema on the skin with the involvement < 10% of BSA
Erythema nodosum	Adults 0.8–3% Children 8.6%	Patients are successfully treated with clarithromycin without recurrence
Leukocytoclastic vasculitis	Lack of data	More often occurring in women
IgA vasculitis	Children 9.1%	Higher rates of renal involvement, abdominal pain, and joint inflammation
Subcorneal pustular dermatosis	Lack of data	Rapid and sustained remission
Gianotti-Crosti syndrome	Single cases	The characteristic skin lesions coexist with symptomatic respiratory tract infection
Acute febrile neutrophilic dermatosis	Single cases	Fast improvement with prednisone therapy

CONCLUSIONS

MP infection can result in various dermatological symptoms, particularly in the pediatric population. A nonspecific maculopapular rash is the most common dermatological form associated with *MP* respiratory tract infection, often resolving spontaneously. Urticaria, erythema multiforme, Stevens-Johnson syndrome, or M*ycoplasma*-induced rash with mucositis (MIRM) may also occur. Differences in the course of these diseases are observed in the individuals infected with *MP* compared to other etiological factors. Erythema nodosum, leukocytoclastic vasculitis, and IgA vasculitis are rare but confirmed manifestations of *MP* infection. Several cases of subcorneal pustular dermatosis, Gianotti-Crosti syndrome, and acute febrile neutrophilic dermatosis associated with *MP* infection have been described. A summary of differences in dermatological diseases caused by *MP* and other causative factors is presented in table 1.

The variety of symptoms is linked to the complexity of diseases caused by *MP*. In addition to its direct action at the site of infection through local inflammatory cytokines, *MP*, as a superantigen, can indirectly lead to the activation of multiple lymphocyte clones. Such *MP* action leads to dysregulation of the immune system and immunological manifestations of infection.

Awareness of the diversity of dermatological manifestations in *MP* infection is crucial for early diagnosis. It should be emphasized that fast recognition of *MP* infection, especially in children, has an impact on the treatment and prevention of complications.

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ETHICAL APPROVAL

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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