CORRESPONDENCE

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Diagnostic Uncertainty in *Mycoplasma pneumoniae*-Induced Rash and Mucositis

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To the Editor,

We read with interest the case report by Kanat et al. (1) entitled "Uncommon Presentation of *Mycoplasma pneumoniae*: Rash and Mucositis Manifestation," recently published in Infectious Diseases and Clinical Microbiology. The authors present a young adult with community-acquired pneumonia who subsequently developed mucosal involvement and limited cutaneous lesions, interpreted as *Mycoplasma pneumoniae*-induced rash and mucositis (MIRM). We congratulate the authors for drawing attention to this uncommon and clinically important extrapulmonary manifestation of *M. pneumoniae* infection.

However, we believe that several diagnostic aspects of this case require further discussion. First, the diagnosis of acute *M. pneumoniae* infection appears to have been based solely on single-sample serology, with both immunoglobulin M (IgM) and immunoglobulin G (IgG) positivity. In adults, isolated serologic positivity may be difficult to interpret because antibody responses may be delayed, persist after infection, or yield false-positive and cross-reactive results. Previous studies have emphasized that paired serology demonstrating seroconversion or a significant rise in antibody titers, together with molecular testing, provides stronger diagnostic evidence for acute *M. pneumoniae* infection (2,3). In this context, confirmation by respiratory polymerase chain reaction, seroconversion, or a fourfold rise in antibody titers would have strengthened the microbiological basis of the diagnosis. The report states that a multiplex respiratory tract pathogen panel was negative; however, it is not clearly specified whether this panel included *M. pneumoniae*. This point is important, as a negative molecular test, including *M. pneumoniae*, would weaken the microbiological support for the diagnosis.

Second, the temporal relationship between moxifloxacin exposure and the development of mucocutaneous lesions should be considered more carefully. The patient developed conjunctivitis, oral mucositis, lip erosions, and targetoid or vesiculobullous skin lesions on the seventh day of moxifloxacin therapy. This timing is compatible with not only MIRM but also fluoroquinolone-associated drug reactions. Although such reactions are uncommon, fluoroquinolones, including moxifloxacin, have been reported as potential triggers of severe cutaneous adverse reactions (4,5). Therefore, although MIRM is a plausible diagnosis, a drug-induced mucocutaneous reaction cannot be definitively excluded, particularly because moxifloxacin was continued after hospital admission.

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Third, the distinction between MIRM and drug-induced mucocutaneous eruptions may be challenging on clinical grounds alone. MIRM is generally characterized by prominent mucositis, relatively sparse cutaneous involvement, younger age, and a favorable prognosis compared with classic Stevens-Johnson syndrome/toxic epidermal necrolysis (6,7). However, these features are not entirely specific. In the reported case, no dermatologic biopsy, histopathologic evaluation, or direct immunofluorescence findings were provided. Although these investigations are not mandatory in every case, they may be particularly useful when the diagnosis is uncertain and when a recently introduced drug represents a competing explanation.

Finally, the negative herpes simplex virus result requires clarification regarding the type of sample tested. If herpes simplex virus (HSV) polymerase chain reaction was not performed directly from mucosal or ocular lesions, exclusion of herpes-associated disease may remain incomplete. Moreover, although purulent conjunctival secretion was reported, no conjunctival culture or molecular testing was provided; therefore, concomitant bacterial or viral conjunctivitis could not be confidently excluded.

Overall, the clinical presentation is compatible with MIRM, particularly given the predominance of mucosal involvement, limited skin disease, young age, and favorable outcome. Nevertheless, the available findings support a probable rather than definitive diagnosis. Therefore, this case may be more cautiously interpreted as a MIRM-like mucocutaneous eruption associated with suspected acute *M. pneumoniae* infection, while acknowledging the temporal association with moxifloxacin exposure as an important diagnostic limitation.

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The Authors Reply

Dear Editor,

We thank the authors for their thoughtful comments on our case report entitled "Uncommon Presentation of *Mycoplasma pneumoniae*: Rash and Mucositis Manifestation" (1). We appreciate the opportunity to clarify several diagnostic aspects of the case.

First, we agree that the microbiological diagnosis of acute *M. pneumoniae* infection would have been strengthened by paired serologic testing demonstrating seroconversion or a significant rise in antibody titers, or by a positive molecular assay. Previous studies have emphasized the limitations of single-sample serology for the diagnosis of acute *M. pneumoniae* infection, particularly in adults, because antibody responses may be delayed, persist after infection, or yield false-positive and cross-reactive results (2,3).

In our patient, the diagnosis was based on compatible clinical and radiological findings of pneumonia together with positive *M. pneumoniae* IgM and IgG serology. We would like to clarify that the multiplex respiratory pathogen panel used in our center was the Respiratory Tract Reverse Transcription Quantitative Polymerase Chain Reaction (RT-qPCR) MX-24 Panel, which included *M. pneumoniae*. All targets in this panel, including *M. pneumoniae*, were negative. However, upper respiratory tract sampling was technically difficult because the patient had severe oral mucositis and could not open his mouth adequately. Therefore, we interpreted the negative molecular result cautiously. We acknowledge that, in the absence of paired serologic testing or a positive respiratory PCR result, the microbiological evidence supports a probable rather than a definite acute *M. pneumoniae* infection.

Second, we agree that the temporal relationship between moxifloxacin exposure and the development of mucocutaneous lesions represents an important diagnostic limitation. The patient developed mucosal and cutaneous findings on the seventh day of moxifloxacin therapy; therefore, a drug-related mucocutaneous reaction could not be completely excluded. Fluoroquinolones, including moxifloxacin, have rarely been associated with severe mucocutaneous adverse reactions (4,5). However, at the time of admission, pneumonia findings were still present, and moxifloxacin was continued because the patient required ongoing treatment for pneumonia and because there was no clear evidence of progressive severe drug hypersensitivity at that time. The patient had no eosinophilia, liver enzyme elevation, renal dysfunction, or systemic deterioration. In addition, the lesions improved after systemic corticosteroid therapy despite continuation of moxifloxacin, and complete resolution was observed during follow-up. These findings suggest that a progressive moxifloxacin-induced severe cutaneous adverse reaction was less likely, although not impossible.

Third, dermatology consultation was obtained because of diagnostic uncertainty. The differential diagnosis included Stevens-Johnson syndrome, generalized fixed drug eruption, pemphigus vulgaris, bullous pemphigoid, and Sweet syndrome. A conventional skin biopsy was performed. However, a biopsy for direct immunofluorescence could not be obtained because of timing-related limitations and the initiation of systemic corticosteroid therapy.

Histopathologic examination revealed widespread full-thickness epidermal necrosis with a silhouette-like appearance and subepidermal separation. In the small areas without separation, numerous dyskeratotic cells and basal vacuolization were ob-

served. Fibrin, lymphocytes, and neutrophils were present beneath the separated area. The superficial dermis showed mild perivascular mononuclear inflammatory infiltration and mild erythrocyte extravasation. Only very rare eosinophils were observed, and no significant pathological finding was detected in the deep dermis. Periodic acid–Schiff (PAS) staining did not reveal fungal organisms, and the basement membrane could not be clearly identified in the separated areas.

Although these histopathologic findings are not pathognomonic for *Mycoplasma pneumoniae*-induced rash and mucositis (MIRM) and do not definitively exclude a drug-related reaction, they were not suggestive of drug reaction with eosinophilia and systemic symptoms (DRESS), pemphigus vulgaris, bullous pemphigoid, Sweet syndrome, or an infectious fungal process. MIRM is generally characterized by prominent mucositis, limited cutaneous involvement, younger age, and a more favorable clinical course compared with classic Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) (6,7). When interpreted together with the clinical findings—prominent oral and ocular mucositis, limited skin involvement, negative Nikolsky sign, absence of eosinophilia or systemic organ involvement, and a favorable clinical course—the overall picture was considered more compatible with a MIRM-like mucocutaneous eruption than with classic SJS/TEN or other vesiculobullous and neutrophilic dermatoses.

Fourth, we would like to clarify the evaluation for herpes simplex virus (HSV) infection. HSV PCR was performed using a plasma sample. HSV-1 IgG was positive and HSV-1 IgM was negative, while HSV-2 IgG and IgM were negative. These findings were interpreted as evidence of prior HSV-1 exposure rather than acute HSV infection. We agree that HSV PCR performed on mucosal or ocular lesions would have provided stronger evidence for excluding localized HSV-associated disease. Therefore, the absence of lesion-based HSV PCR should be acknowledged as a limitation.

Regarding conjunctival involvement, the patient had bilateral conjunctival hyperemia and purulent secretion and was evaluated by the Department of Ophthalmology. Topical treatment was initiated after ocular involvement had already developed. No conjunctival culture or ocular molecular testing was performed. We agree that this limits the ability to completely exclude concomitant infectious conjunctivitis. However, the simultaneous presence of pneumonia, prominent oral and ocular mucositis, limited vesiculobullous/targetoid skin lesions, and a favorable response to systemic corticosteroid treatment supported the diagnosis of a MIRM-like mucocutaneous involvement.

In conclusion, we acknowledge that this case should be interpreted cautiously. Rather than a definite diagnosis, the findings are best described as a probable MIRM-like mucocutaneous eruption associated with suspected acute *M. pneumoniae* infection. We also acknowledge the temporal association with moxifloxacin exposure, the lack of paired serology or positive respiratory PCR, the absence of direct immunofluorescence, and the absence of lesion-based HSV PCR or conjunctival microbiological testing as important limitations. Nevertheless, we consider that the clinical constellation remains compatible with a MIRM-like mucocutaneous eruption and that the case is valuable in reminding clinicians to consider *M. pneumoniae*-associated mucocutaneous disease in patients presenting with pneumonia, prominent mucositis, and limited cutaneous involvement.

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