

Mycoplasma Pneumoniae-associated Subcorneal Pustular Dermatosis: Not as Rare as We Think?

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Introduction

Subcorneal pustular dermatosis (SPD) or Sneddon-Wilkinson disease is a rare condition included in the spectrum of neutrophilic dermatoses. Seldom it can be triggered by infections caused by Mycoplasma pneumoniae (MP) [1]. Herein we present the case of a young woman with SPD associated with an otherwise asymptomatic MP infection. Topical corticosteroids resolved lesions with no relapses after one-year follow-up.

Case Presentation

A 40-year-old woman presented with a two-week history of asymptomatic skin lesions located on the cervical, axillar, inframammary and inguinal folds, with no improvement despite treatment with topical clotrimazole and oral fluconazole. She felt otherwise well and reported no other symptoms. She was diagnosed with irritative eczema and treated with 0.5 mg/kg daily oral prednisone with clinical worsening a few days later.

Clinical examination revealed well-demarcated non-infiltrated erythematous plaques with multiple fragile pustules measuring less than 1 mm over them, located on the aforementioned folds (Figure 1), some of them with annular and polycyclic borders. Physical examination was otherwise unremarkable. Oral prednisone was immediately switched to mometasone furoate 0.1% cream twice a day. Skin samples were culture-negative for bacterial and fungal pathogens and blood tests showed positive MP IgM and negative IgG. Skin biopsy (Figure 2) showed a subcorneal pustule filled with neutrophils, neutrophilic exocytosis below the pustule without other prominent epidermal changes, and dermal perivascular and interstitial infiltrate composed by neutrophils and lymphocytes. Direct immunofluorescence was negative, and no fungal structures were observed with PAS stain. These histological features were consistent with that of subcorneal pustular dermatosis. A few weeks later, blood tests demonstrated MP IgG seroconversion. After 15 days with topical mometasone the skin lesions had completely healed. With a diagnosis of MP-associated SPD, the patient was followed up for 1 year, with no recurrence.



Figure 1. Clinical examination: large well-demarcated erythematous plaques with annular and polycyclic borders involving axillar and inframammary folds, with pustules measuring less than 1 mm.

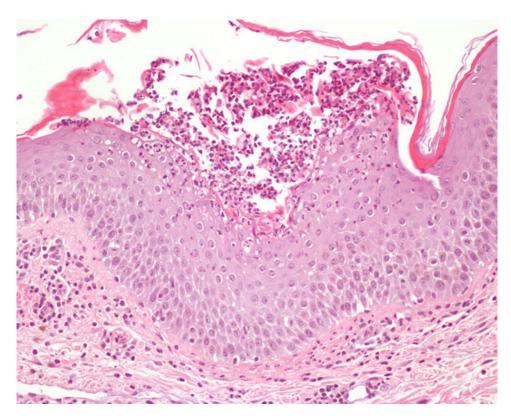


Figure 2. H&E ×20. Skin biopsy suggestive of subcorneal pustular dermatosis. A subcorneal pustule with neutrophilic exocytosis in the epidermis below it is observed, with no other remarkable epidermal changes; neutrophilic and lymphocytic dermal perivascular and interstitial infiltrate.

Conclusions

MP-associated SPD has been previously reported by Winnock et al in a 43-year-old male, Papini et al in an 8-year-old male, Bohelay et al in a 19-year-old male and Lombart et al in a 36-year-old woman [2-5]. All the patients were young like our case, and MP infection clinical manifestations varied: some patients had a mild cough while others suffered pneumonia that required inpatient treatment. A few days later, the patients developed the skin manifestations

compatible with SPD. Winnock and Papini patients' were treated with oral dapsone with resolution of the skin lesions in 1-3 months, and Bohelay and Lombart patients' were treated with topical corticosteroids with improvement in 5-15 days, similarly to our case [2-5]. None of the patients suffered a relapse during follow-up [2-5]. These findings suggest that MP-SPD may require a different approach than "classical" SPD, for these patients have self-limiting and non-relapsing disease. Thus, we propose topical corticosteroids as a first-line therapy; oral dapsone could be reserved for unresponsive or serious cases. A scheduled follow-up visit may be unnecessary. As stated by Bohelay and Lombart MP is probably an underestimated trigger for SPD, given the asymptomatic course of the infection in many cases like our patient [4,5].

Even without symptoms of infection, laboratory testing for MP should be performed in young patients with SPD, as it may change the clinical approach to the patient, thus avoiding unnecessary systemic treatment and follow-up.

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