

Pustular Bacterid Associated with Ankylosing Spondylitis

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Sir,

Pustular bacterid (PB) is a pustular disorder of the hands and/or feet. It was first described by Andrews and co-workers (1, 2). There is controversy as to whether PB is a separate disease or is a variant of psoriasis pustulosa palmaris et plantaris (PPP). We describe here a case of PB in a patient with ankylosing spondylitis (AS). This is the first report of PB associated with AS and supports the theory that PB is a distinct disease.

CASE REPORT

A 42-year-old Chinese woman presented on 16 August 2005 with a one-week history of pustules on both hands, a fever of approximately 38°C, sore throat, cough and expectoration with yellow-coloured sputum. She had a 13-year history of painful orthopaedic symptoms with morning stiffness in the sacro-iliac area, which improved with exercise. AS had been diagnosed according to characteristic clinical, X-ray and computed tomography

(CT) findings in the Department of Rheumatology of the same hospital approximately one month previously. She had been prescribed oral sulfasalazine 250 mg t.i.d., leflunomide 10 mg q.d. and celecoxib 200 mg b.i.d. Her pain had relieved significantly within one week and she was discharged after 11 days, continuing the 3 medications until admitted to our department. She had no personal or family history of psoriasis.

Physical examination revealed multiple isolated 2–8 mm diameter pustules on both hands, especially the palms (Fig. 1). The feet and other parts of body were spared. No fissured or geographic tongue was observed.

Laboratory tests, including blood, urine and stool routine tests, liver and kidney function tests, fast blood glucose and electrolytes were normal. Serum IgG, IgA, IgM and complement levels were normal. Serum anti-streptolysin antibodies, C-reactive protein, rheumatoid factors, antinuclear antibodies, anti-dsDNA antibodies, anti-extractable nuclear antigen antibodies, anti-cardiolipin antibodies and anti-neutrophil cytoplasmic



Fig. 1. (A, B) Multiple isolated 2–8 mm diameter pustules on both hands, especially the palms.



Fig. 2. (A, B) Most of the pustules crusted after a single dose of 500 mg azithromycin.

antibodies were negative. HLA-B27 were negative. The erythrocyte sedimentation rate was 21 mm/h. Serum antibodies against *Mycobacterium tuberculosis*, *Mycoplasma pulmonis*, human immunodeficiency virus, herpes simplex virus, rubella virus, toxoplasma virus, cytomegalovirus, respiratory syncytial virus, Epstein-Barr virus, coxsackie virus, adenovirus and hepatitis A, B, C and E viruses were negative. Serum hepatitis B surface antigen was negative. Repeated cultures from contents of pustules, sputum and blood for bacteria and fungi were negative. Throat smears revealed many Gram-positive cocci. Throat swab cultures revealed growth of group A β -haemolytic *Streptococcus*.

PB and acute upper respiratory tract infection were clinically diagnosed. Intravenous azithromycin was administered as a single daily dose of 10 mg/kg for 3 days. The pustules resolved dramatically overnight (Fig. 2). The fever, sore throat, cough and expectoration were also relieved. The patient refused a biopsy and requested a discharge. The pustules crusted over completely after 3 days' azithromycin therapy and the patient was then discharged. No relapse of pustules occurred during a 2-year follow-up.

DISCUSSION

To date, the aetiology of PB is not clear. Many authors have found that PB is triggered by upper respiratory tract infections (3). PB has been reported to be associated with sternocostoclavicular hyperostosis or Tietze's syndrome (4, 5). Occasionally, PB can be generalized (6). In the present case, it seems that the PB might be related to AS, as the patient had a long history of AS,

which could affect her immune status, resulting in susceptibility to infections.

Many authors still question whether PB is a distinct disease. It is difficult to differentiate PB histopathologically from psoriasis PPP. Bacharach-Buhles suggested that "true" PB had the following features: coincidence with an infection, no personal or family history of psoriasis, isolated sterile pustules, no lesions of psoriasis vulgaris, good response to antibiotics and a short course (3). The present case fulfilled the above features of PB and had clinical manifestations fairly distinct from those of psoriasis PPP. This case provides further evidence that PB might be a distinct entity.

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