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Clinical Letter

Dear Editor,

Ethionamide-induced acneiform drug eruption in a patient with multiresistant tuberculosis

A 21-year-old woman of Nepalese descent presented with acute onset of a worsening, pruritic, papulopustular eruption beginning on the forehead and extending to the mandible, neck, arms and upper trunk. The eruption began 6 weeks following commencement of ethionamide for multidrug-resistant tuberculosis. Her only other medications at the time of the eruption were Moxifloxacin 400 mg daily (commenced 9 months prior) and Clofazamine 100 mg daily (commenced 9 months prior). The patient was otherwise well, and her only medical history was being mild acne since adolescence. She did not have any known allergies. Her tuberculosis was diagnosed after she presented with left-sided neck swelling. A biopsy of a cervical lymph node revealed necrotising granulomatous inflammation. There was no evidence of pulmonary disease clinically or radiologically. She subsequently developed peripheral neuropathy, which was thought to be related to her ethionamide treatment.

On examination, she had a fairly monomorphic papulopustular eruption affecting the face and extending to the neck, upper chest, breasts, shoulders and upper arms (Fig. 1a,b). Scattered comedones were present in the same areas.

A punch biopsy from the mandibular area demonstrated acute folliculitis and perifolliculitis. There was a central inflamed cystically dilated follicle and perifollicular inflammation with a moderate neutrophilic infiltrate (Fig. 2a,b). No necrotizing or well-formed granulomata were present.

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Figure 1 (a and b) Severe facial papulopustular eruption extending to the upper chest. (c and d) Papulopustular eruption settled following cessation of ethionamide, leaving scattered comedones and postinflammatory hyperpigmentation.

Figure 2 (a and b) Inflamed cystically dilated follicle and perifollicular inflammation with a moderate neutrophilic infiltrate. There is a surrounding superficial perivascular lymphohistiocytic infiltrate.
No organisms were identified on PASD or H&E. Tissue culture grew coagulase-negative staphylococcal species, and mycobacterial cultures were negative. Biochemistry showed ongoing transaminasitis thought to be a remnant side effect to previous antituberculosis medications. Her white cell count and eosinophils were both within normal ranges.

A diagnosis of acneiform drug reaction secondary to ethionamide was made. Ethionamide was ceased about 5 weeks after the onset of the acneiform eruption. We considered commencing her on oral doxycycline, but due to her ongoing transaminasitis and poor tolerance of many previous antibiotic therapies, she was not commenced on any specific therapy for her eruption. The eruption settled spontaneously, and on review about a month after ceasing ethionamide, the papulopustular lesions had almost all resolved, albeit with marked postinflammatory hyperpigmentation (Fig. 1c,d). Some scattered comedones remained, which was in keeping with the mild background acne that she had since adolescence.

The sudden onset of the eruption a few weeks after commencing ethionamide, the extensive involvement and the fairly dramatic improvement upon cessation of the drug are consistent with the eruption being induced by ethionamide. Although the patient did suffer from underlying acne since adolescence, this had been quite mild and had essentially been limited to the face. Based on the Naranjo algorithm, an adverse drug reaction is probable (Naranjo score 6).3

Ethionamide is a second-line agent used in the treatment of multidrug-resistant tuberculosis. It is a prodrug, which when activated, inhibits synthesis of mycolic acid, a key component of the mycobacterial cell wall. Ethionamide is a structural analogue of isoniazid, another antituberculous drug that has been associated with acneiform drug eruptions.4 Ethionamide has been associated with cutaneous adverse drug reactions including drug rash with eosinophilia and systemic symptoms (DRESS) and Stevens-Johnson syndrome (SJS).4 Although ‘acne’ is listed as a possible adverse effect of ethionamide in the product prescribing information,2 we were unable to find any case reports or studies that describe this adverse effect. To our knowledge, this is the first description of the clinical characteristics of an acneiform eruption secondary to ethionamide. Based on our case report, ethionamide may cause a predominantly papulopustular monomorphic acneiform eruption on the face, upper trunk and arms, which settles fairly quickly upon cessation of the medication, without any treatment.

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Clinical Letter

Management of genital hidradenitis suppurativa and lymphoedema with the restoration of erectile function

Hidradenitis suppurativa (HS) involving the anogenital and inguinal regions can cause considerable morbidity. Genital lymphoedema is an under-recognised complication of long-standing severe HS. Despite the advent of immunomodulatory therapy, surgical management is still required in some situations. We present a case of anogenital HS with associated genital lymphoedema and discuss the multidisciplinary management of this disease.

A 40-year-old man developed scrotal and penile lymphoedema after a 20-year history of treatment-resistant, anogenital and inguinal, Hurley Stage III HS (Fig. 1a,b). His disease had caused impotence, notwithstanding weekly adalimumab 40 mg with intermittent intraleosinal steroid injections, doxycycline and a combination of clindamycin and rifampicin for 2 years. Given extensive induration and lymphoedema, he proceeded to surgery without interruption to adalimumab.

Circumcision was performed to enable the visualisation of the buried and obscured glans and urethral meatus, necessary to facilitate urinary catheterisation. Excision of the multidisciplinary management of this disease.

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The patient involved in this report has given informed consent for their relevant clinical information and photographs to be used in its production.