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Conflicts of interest: none declared.

Omeprazole-induced dermatomyositis

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SIR, The proton pump inhibitor omeprazole is a well-studied agent effective as monotherapy for the treatment of gastro-oesophageal reflux disease with or without oesophagitis.[1] To our knowledge, there have not been any cases of omeprazole-induced dermatomyositis reported to date. We present the first case of a dermatomyositis-like reaction induced by omeprazole.

An 81-year-old woman presented with a 1-month history of increasing symmetrical proximal muscle weakness characterized by difficulty getting up out of a chair and lifting her arms above her head. She also had a 1-month history of a progressive pruritic erythematous eruption which started as a vesicular rash on her dorsal hands and then spread to her face and most of her lower limbs. The day prior to presentation, she had become unwell with worsening lethargy, anorexia and fever (38 °C). Her medical history included dementia, umbilical hernia and severe reflux oesophagitis (grade 5: oesophagitis with deep ulceration). Three days prior to the onset of her muscle weakness and skin eruption she had commenced oral omeprazole 40 mg daily for treatment of her reflux oesophagitis. Her other medications, all of which she had been taking for at least 1 year, included meloxicam, prochlorperazine maleate and docusate sodium tablets. She had no previous known allergies to medications.

On examination, she had erythematous papules over the dorsal metacarpophalangeal and proximal interphalangeal joints of both hands consistent with Gottron's papules, a pathognomonic cutaneous feature of dermatomyositis.[2] There was a photosensitive erythematous papular eruption on the face and upper chest, including a heliotrope rash on the upper eyelids (Fig. 1). Her legs displayed erythematous papules associated with scattered petechiae. The clinical differential diagnoses included dermatomyositis, vasculitis and a drug eruption.

Laboratory tests showed an elevation in serum creatine kinase at 921 IU L^{-1} (normal < 145), C-reactive protein 45 mg L^{-1} (normal < 5) and erythrocyte sedimentation rate 24 mm in the first hour (normal < 20). Renal function was normal. A 3-mm skin punch biopsy was taken from both the lower right leg and the dorsum of the right hand. The hand biopsy showed oedema,

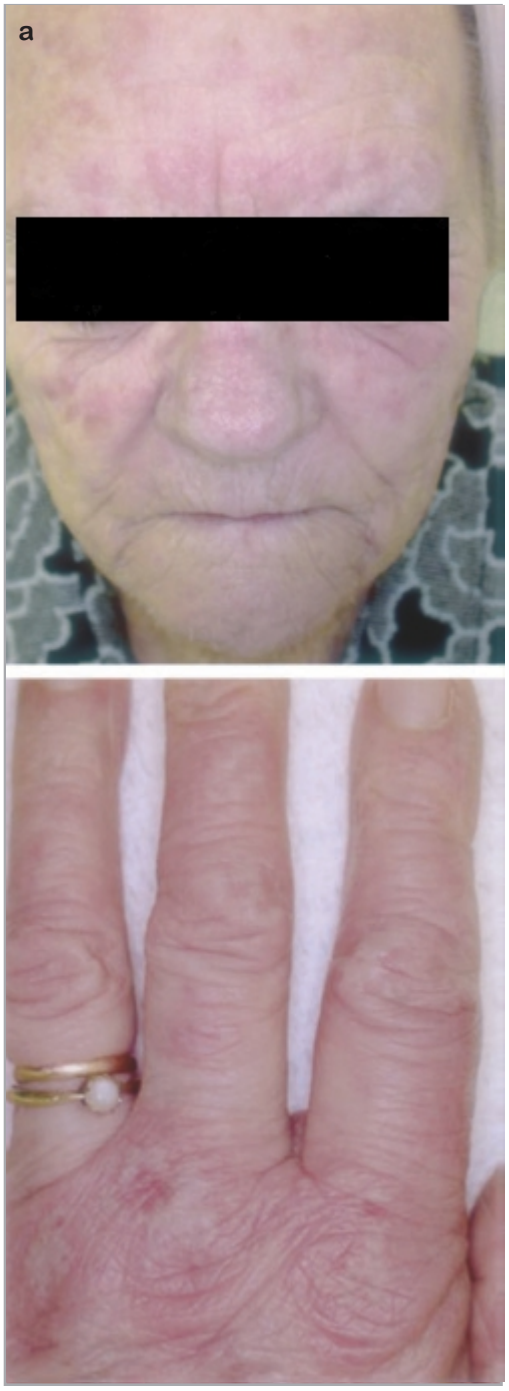

Fig 1. (a) Heliotrope rash of upper eyelids and erythematous papules on face. (b) Vesicular papules with surrounding erythema overlying the metacarpophalangeal and proximal interphalangeal joints.

focally necrotic epidermis, a band-like palisaded histiocytic reaction and a superimposed mixed acute on chronic inflammatory infiltrate with focal leucocytoclasis (Fig. 2). The leg biopsy was nonspecific. Direct immunofluorescence was negative. These findings were most consistent with a drug-induced reaction. Given that the eruption and muscle weakness began 3 days after commencement of omeprazole, this was considered the likely cause. Omeprazole was replaced with ranitidine hydrochloride 300 mg daily, and mometasone furoate ointment twice daily was prescribed for the rash. On review after 1 week, the patient had less muscle weakness and was able to lift both of her arms above her head. Her rash had also settled significantly with less erythema and oedema, particularly over her dorsal hands.

In summary, we have reported what we believe to be the first case of omeprazole-induced dermatomyositis. Drug-induced dermatomyositis-like reactions are rare. They have been described in association with chemotherapeutic agents (hydroxycarbamide, cyclophosphamide and etoposide),[3,4] D-penicillamine, nonsteroidal anti-inflammatory drugs (aspirin, diclofenac and phenylbutazone), anti-infectious agents (penicillins, sulphonamides and isoniazid) and lipid lowering drugs (nicotinic acid, fibrates and statins).[2] With the widespread use of omeprazole, it is likely that this dermatomyositis-like reaction may occur in more patients than we recognize. We believe that a drug history is important when assessing a patient with dermatomyositis.

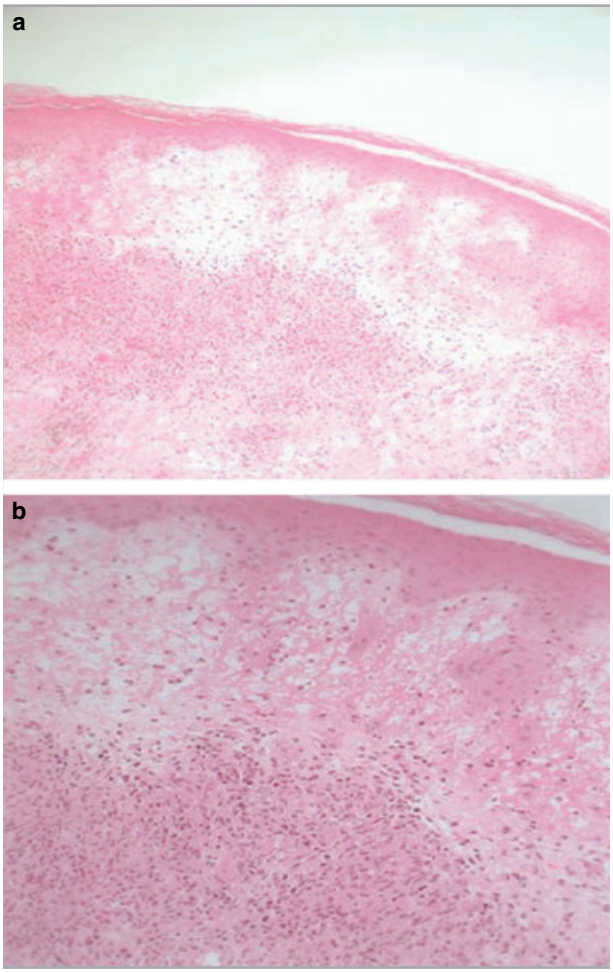

Fig 2. Histological findings. (a) Low-power photomicrograph showing necrosis and degenerative changes in the basal layer of the epidermis, marked oedema and necrosis of the upper dermis and a prominent histiocytic and lymphocytic inflammatory reaction in the immediately subjacent dermis. (b) Higher power photomicrograph showing detail of the lower epidermal and dermal necrosis, inflammation in these regions and the prominent histiocytic reaction in the underlying dermis. There was no definite small vessel vasculitis identified.

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Routine, rapid, noninvasive diagnosis of viral skin exanthems

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Sir, We read with great interest the case report by Clark *et al.*[1] reporting a case of human sealpox. We are, though, concerned that the histopathological approach to diagnosis, which involved taking a biopsy from the patient's hand, is an unnecessarily invasive, complicated, costly and time-consuming procedure. In cases of putative virus-induced vesicular-ulcerative lesions such as this, we would prefer using direct diagnostic negative-contrast electron microscopy.

Orthopoxvirus and parapoxvirus infections cause skin lesions/rashes with vesicular/pustular/ulcerated/crusting lesions. Poxvirus infections, like those caused by herpesviruses, contain high concentrations of virus, often exceeding 10^{10} particles mL^{-1}. These high concentrations allow rapid and virus family-specific detection of the causative virus by direct electron microscopy. Instead of taking an invasive biopsy under local anaesthesia, the diagnosis can be made straightforwardly and quickly by negative-contrast electron microscopy on material