problem in patients taking this drug. Daily supplementation with vitamin B12 and folate, which are the cofactors of the enzymatic reactions involved in homocysteine metabolism, can lower plasma levels of homocysteine,[5] and therefore could reduce, if not totally counteract, the side-effects of isotretinoin.

A. Ghanizadeh and M. R. Namazi*
*Research Center for Psychiatry and Behavioural Sciences and *Medicinal and Natural Products Chemistry Research Center and Dermatology Department, Shiraz University of Medical Sciences, Shiraz, Iran*
E-mail: ghanizad@sina.tums.ac.ir
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An increasingly recognized cause of dysphagia in patients with severe atopic dermatitis

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Eosinophilic oesophagitis (EO) is an increasingly recognized cause of a variety of oesophageal symptoms, caused by a dense infiltration of eosinophils. It is a rare condition that predominantly affects people with an atopic background, and has often been referred to as 'asthma of the oesophagus'.[1] Isolated cases of EO in the absence of a history of atopy exist, as well as cases in familial clustering, suggesting that the pathogenesis is multifactorial.[2] We report a case of a patient with atopic dermatitis (AD) who developed oesophageal symptoms due to EO.

A 32-year-old man presented with long-standing dysphagia and food impaction. There was no associated weight loss or haematemesis. He denied experiencing heartburn. He had a history of AD since childhood, and his oesophageal symptoms coincided with a marked deterioration of his skin condition. He had a total eosinophil count of 0.6×10^9/L (normal range $0.04–0.4 \times 10^9$/L) and total IgE count of 1203 ng/mL. A 3-month course of proton pump inhibitor (PPI) prescribed by his general practitioner had no effect on his symptoms, which became progressively worse.

He underwent gastroscopy, which did not show any evidence of ulcerations, strictures or malignancy. The mucosa appeared normal macroscopically. On histological examination of biopsies taken from the oesophagus, strips of hyperplastic epithelium infiltrated by a significant number of eosinophils, which were also present within the stroma, were seen (Fig. 1). These clinicopathological findings, together with the lack of response to the PPI, led to the diagnosis of the rare but well-described condition of atopic eosinophilic oesophagitis disease. The patient was started on montelukast, which later had to be substituted by azathioprine to achieve sustained symptomatic control.

EO was first described in 1993 and is becoming an increasingly recognized condition, particularly in patients with atopy, with dysphagia and food impaction being the commonest presenting symptoms, with or without regurgitation.[1,3] The diagnosis is based on clinicopathological correlation in an individual with an atopic background who either fails to respond to a 6-week course of PPIs or has a normal oesophageal pH study, thereby excluding gastro-oesophageal reflux disease (GORD). Histological specimens from the oesophagus should contain at least 15 eosinophils per high-power field.[1,4] Other conditions in which oesophageal eosinophilic infiltration occurs include parasitic infections, oesophageal carcinoma, drug hypersensitivity, connective tissue disease, Crohn disease and

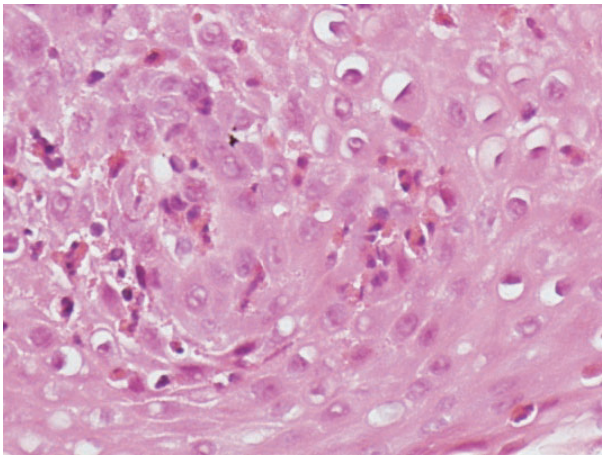

Figure 1 Oesophageal squamous epithelium with large eosinophilic infiltrate.

GORD. The differentiation is based on the history, clinical presentation and histological findings. EO is therefore often a diagnosis of exclusion.[2,3]

The disease is more common in men, and presents mostly in the third to fourth decades of life; however, children can also be affected, and classically present with symptoms of regurgitation and failure to thrive.[2,3] The main changes that occur in the oesophagus are visible at the submucosal level, and may include thickening of the muscularis propria layer and myenteric plexus dysfunction (leading to peristalsis) secondary to the eosinophilic infiltration and inflammation. This may explain the presence of symptoms in a macroscopically normal-looking oesophagus. It is believed that food and inhaled allergens play an important role, with significant symptomatic improvement after avoidance reported in the literature.[1]

There is increasing evidence to suggest that EO is primarily a T-helper 2 inflammatory process with certain interleukins (ILs) playing an important role (mainly IL-3, IL-5 and IL-13). IL-5 seems to be the most important of these cytokines, as it is a powerful eosinophilic chemoattractant. This is further supported by the documented efficacy of the humanized monoclonal antibody against IL-5 (mepolizumab) in the treatment of EO.[5] Various other treatments have been used including systemic steroids and antileucotriene antagonists;[2,4] however, further studies are needed. AD is a very common inflammatory condition, and dermatologists should be aware of this rare but increasingly recognized associated condition.

F. Al-Niaimi and J. Yell
Department of Dermatology, Salford Royal Hospital, Stott Lane, Salford, Manchester, M6 8HD, UK
E-mail: firas55@hotmail.com
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Nonerythrodermic Sézary syndrome

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A 39-year-old man presented to his local dermatologist with a 9-month history of an intensely itchy rash over the arms and legs. His medical history included hayfever and treated depression. He was not taking any regular medications, and there was no family history of atopy. Scaly papular lesions were present on the forearms, legs, palms and soles, and a diagnosis of atopic dermatitis was made. The patient's skin improved transiently with emollients and potent topical corticosteroids, but the severe pruritus remained. Intramuscular triamcinolone and 33 sessions of narrowband ultraviolet B phototherapy were ineffective; 6 months after the initial presentation the patient remained intensely pruritic. Histological examination of a skin biopsy taken from the dorsum of the right hand was consistent with a diagnosis of cutaneous T-cell lymphoma (CTCL). A blood film showed atypical lymphocytes. The patient was sent to St John's Institute of Dermatology for further investigation and treatment.

On physical examination, hyperkeratotic lesions were seen on the hands, feet and nose with vesiculation (Fig. 1). There was no erythema nor patches, plaques or tumours characteristic of mycosis fungoides. There was generalized shotty palpable lymphadenopathy (< 15 mm).

Laboratory investigations revealed peripheral lymphocytosis of 7.4×10^9/L (normal range $1.5–4 \times 10^9$/L), high lactate dehydrogenase level of 872 IU/L (240–480 IU/L) and a Sézary count of 46%. Lymphocyte subset analysis gave a total lymphocyte count of 8.58×10^9/L ($1.0–3.9 \times 10^9$/L), CD4 count 8.74×10^9/L ($0.4–1.8 \times 10^9$/L) and CD8 count 1.22×10^9/L (0.2–0.9). A blood film confirmed numerous Sézary cells.

A diagnosis of CTCL was confirmed by a repeat skin biopsy taken from the dorsum of the right hand. T-cell receptor (TCR) β and γ gene rearrangement studies confirmed the presence of a clonal population.

On histopathological examination of a bone marrow trephine biopsy, low level (< 5%) bone marrow involvement with CTCL was seen. PCR confirmed the presence of an identical clonal TCR gene rearrangement to that in the skin.

A staging computed tomography (CT) scan showed small-volume cervical, axillary and inguinal lymphadenopathy; positron emission tomography-CT showed high fluorodeoxyglucose uptake in the right groin node. On histological examination of a right inguinal biopsy, an effaced lymph node was seen, with extensive infiltration by abnormal CD4+ T cells (EORTC stage N_3). Molecular genetic studies confirmed the presence of an identical clonal proliferation to the blood, bone marrow and skin.

Thus, the skin biopsy, blood film, haemocytology, bone marrow and lymph node analysis were all consistent with a diagnosis of stage T1B1N3M0 (IVA) CTCL. However, this patient did not satisfy all the clinical criteria for the

