Management of the mycosis fungoides and Sézary syndrome spectrum is a staged approach. The early stage of mycosis fungoides requires skin-directed therapies, such as corticosteroids, phototherapy (psoralen and ultraviolet radiation), topical chemotherapy and topical or systemic bexarotene (an agonist of the retinoid X receptor involved in the regulation of cell differentiation and proliferation) (Prince et al, 2009).

The patient required a combination of bexarotene, α-interferon and extracorporeal photopheresis. In photopheresis therapy, blood is extracted, treated with photoactivable drugs (psoralens), exposed to ultraviolet light and then returned to the patient (Zic, 2012). Photochemically damaged T cells appear then to induce cytotoxic effects on T cell proliferation.

Systemic chemotherapy, including single agent chlorambucil and multiagent regimens such as CHOP (cyclophosphamide, doxorubicin, vincristine and prednisolone), has short-lived responses. Immunomodulatory agents, proteasome inhibitors and monoclonal antibodies are under investigation. Several histone deacetylase inhibitors have gained approval (Whittaker et al, 2010), expanding treatment options and combination therapies. **BJHM**

Table 1. Differential diagnoses of erythroderma

Eczema
Psoriasis
Seborrheic dermatitis
Pityriasis rubra pilaris
Lichen planus
Drug induced (e.g. lithium, carbamazepine, antimalarials, gold, phenytoin, allopurinol)
Contact dermatitis (i.e. rubber, solvents, detergents)
Stasis dermatitis (gravitational eczema)
Bullous diseases (e.g. pemphigous, pemphigoid)
Human immunodeficiency virus (HIV) infection
Graft *vs* host disease
Connective tissue disease
Internal malignancy as a non-metastatic manifestation
Haematological malignancies (lymphoma, Hodgkin's disease, leukaemia)
Cutaneous T-cell lymphoma (Sézary syndrome)
Idiopathic (may account for as many as 30% of cases)

Agar NS, Wedgeworth E, Crichton S et al (2010) Survival outcomes and prognostic factors in mycosis fungoides/Sézary syndrome: validation of the revised International Society for Cutaneous Lymphomas/European Organisation for Research and Treatment of Cancer staging proposal. *J Clin Oncol* **28**(31): 4730–9

Prince HM, Whittaker S, Hoppe RT (2009) How I treat mycosis fungoides and Sézary syndrome. *Blood* **114**(20): 4337–53

Vonderheid EC, Bernengo MG, Burg G et al (2002) Update on erythrodermic cutaneous T-cell lymphoma: report of the International Society for Cutaneous Lymphomas. *J Am Acad Dermatol* **46**(1): 95–106

Whittaker SJ, Marsden JR, Spittle M et al (2003) Joint British Association of Dermatologists and UK Cutaneous Lymphoma Group guidelines for the management of primary cutaneous T-cell lymphomas. *Br J Dermatol* **149**(6): 1095–107

Whittaker SJ, Demierre MF, Kim EJ et al (2010) Final results from a multicenter, international, pivotal study of romidepsin in refractory cutaneous T-cell lymphoma. *J Clin Oncol* **28**(29): 4485–91

Willemze R, Jaffe ES, Burg G et al (2005) WHO-EORTC classification for cutaneous lymphomas. *Blood* **105**(10): 3768–85

Zic JA (2012) Photopheresis in the treatment of cutaneous T-cell lymphoma: current status. *Curr Opin Oncol* **24** (Suppl 1): S1–10

LEARNING POINTS

- Sézary syndrome can be a diagnostic challenge even for dermatologists.
- The presence of peripheral blood CD4+ lymphocytosis and characteristic Sézary cells helps to confirm the diagnosis.

Life-threatening constipation

An 80-year-old woman presented to the emergency department with sudden onset abdominal pain and vomiting. Her abdomen was soft with umbilical tenderness.

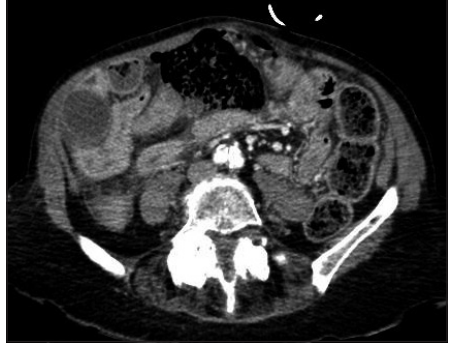
Laboratory tests revealed a leukocytosis of 16.7×10^9/litre, levels of amylase 189 IU/litre and lactate 7.4 mmol/litre, while other investigations were within normal range.

Dr Sumeet Hindocha *is F2 Doctor in the Department of Geriatrics, John Radcliffe Hospital, Oxford OX3 9DU and*
Dr Bob Soin *is Consultant Surgeon in the Department of General Surgery, Wexham Park Hospital, Slough*

Correspondence to: Dr S Hindocha (sumeet.hindocha@hwph-tr.nhs.uk)

Contrast computed tomography showed extensive faecal loading but no cause of her symptoms (*Figure 1*). Laparotomy revealed a 20 mm perforation of the transverse colon with free faeces in the peritoneal cavity.

Figure 1. Computed tomography scan showing extensive faecal loading.



Faecalomas cause ischaemic necrosis of the colon wall, leading to stercoral perforation. This is often plugged by omentum, causing signs of peritonitis to be absent. Chronic constipation and non-steroidal anti-inflammatory drug use are risk factors (Hollingworth and Alexander-Williams, 1991).

This case highlights the need for a high index of suspicion in cases of acute abdomen with a history of chronic constipation or non-steroidal anti-inflammatory drug use. Clinical suspicion of perforation is needed in light of unusual laboratory investigations, even in the absence of clinical and radiological evidence of perforation. **BJHM**

Hollingworth J, Alexander-Williams J (1991) Non-steroidal anti-inflammatory drugs and stercoral perforation of the colon. *Ann R Coll Surg Engl* **73**(6): 337–40