2 Deeths MJ, Chapman JT, Dellavalle RP *et al.* Treatment of patch and plaque stage mycosis fungoides with imiquimod 5% cream. *J Am Acad Dermatol* 2005; **52**:275–80.
3 Didona B, Benucci R, Amerio P *et al.* Primary cutaneous CD30+ T-cell lymphoma responsive to topical imiquimod (Aldara). *Br J Dermatol* 2004; **150**:1198–201.
4 Willemze R, Jaffe ES, Burg G *et al.* WHO–EORTC classification for cutaneous lymphomas. *Blood* 2005; **105**:3768–85.
5 Schon M, Bong AB, Drewniok C *et al.* Tumor-selective induction of apoptosis and the small-molecule immune response modifier imiquimod. *J Natl Cancer Inst* 2003; **95**:1138–49.
6 Urosevic M, Maier T, Benninghoff B *et al.* Mechanisms underlying imiquimod-induced regression of basal cell carcinoma *in vivo*. *Arch Dermatol* 2003; **139**:1325–32.
7 Schon MP, Wienrich BG, Drewniok C *et al.* Death receptor-independent apoptosis in malignant melanoma induced by the small-molecule immune response modifier imiquimod. *J Invest Dermatol* 2004; **122**:1266–76.
8 Coors EA, Schuler G, von den Driesch P. Topical imiquimod as treatment for different kinds of cutaneous lymphoma. *Eur J Dermatol* 2006; **16**:391–3.

Conflicts of interest: none declared.

Hydroxycarbamide: a treatment for lichen sclerosus?

DOI: 10.1111/j.1365-2133.2007.07991.x

SIR, A 67-year-old woman was diagnosed with lichen sclerosus (LS) 10 years ago in our dermatology department. She presented with vulval itching and soreness and had shiny, white, atrophic plaques with telangiectasia and purpura on the labia minora, as well as erythema and fissuring at the posterior fourchette. Over the last 10 years, she has experienced vulval soreness daily. She was treated initially with clobetasone butyrate 0·05% (Trimovate®; GlaxoSmithKline, Uxbridge, U.K.) cream twice daily but has needed to use clobetasol propionate 0·05% (Dermovate®; GlaxoSmithKline) ointment frequently to control her symptoms. In November 2005, she was diagnosed with polycythaemia rubra vera following investigations for malaise. She was started on hydroxycarbamide (hydroxyurea) 1 g daily. Within a month she noticed that her vulval soreness and pruritus had improved, and was asymptomatic by January 2006. She has not used any treatment for her LS over the last 9 months and attributes the improvement in her vulval symptoms to the hydroxycarbamide that she continues to take. She remains asymptomatic with only mild scarring over the labia minora. To our knowledge this is the first case report of LS improving following the initiation of hydroxycarbamide.

LS is a chronic inflammatory disease of unknown aetiology. There is evidence of an autoimmune basis to the condition with circulating IgG autoantibodies to the glycoprotein extracellular matrix protein 1 demonstrated in up to 75% of affected individuals.[1] However, the exact nature of the inflammatory changes that result, and of the signals provoking them, is uncertain. A recent immunohistochemical study has shown increased staining for interferon (IFN)-γ and IFN-γ receptor in LS in comparison with normal vulval skin.[2] Production of IFN-γ, among other cytokines, characterizes T-helper 1 (Th1) responses which promote cell-mediated immunity. Treatment of peripheral blood mononuclear cell cultures with hydroxycarbamide has a differential effect on cytokine production.[3] Th1 cell-mediated immunity is decreased as IFN-γ levels are lowered in a dose-dependent fashion in response to hydroxycarbamide.[3] It is possible that hydroxycarbamide may be beneficial in LS by reducing IFN-γ production.

Hydroxycarbamide is used in the treatment of chronic myelogenous leukaemia, essential thrombocytosis, sickle-cell disease and as an antiretroviral agent in patients with human immunodeficiency virus. Its antineoplastic mechanism of action is believed to be based on its inhibition of the enzyme ribonucleotide reductase. Although hydroxycarbamide is frequently prescribed for the treatment of patients with polycythaemia rubra vera, use is limited by its toxicity and potential leukaemogenicity.[4] These factors need to be considered if it is to be used for patients with LS. Although spontaneous resolution may have occurred coincidentally in this patient, we feel the close timing with initiation of hydroxycarbamide treatment was significant in the resolution of her disease.

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References

1 Chan I, Oyama N, Neill SM *et al.* Characterization of IgG autoantibodies to extracellular matrix protein 1 in lichen sclerosus. *Clin Exp Dermatol* 2004; **29**:499–504.
2 Farrell AM, Dean D, Millard PR *et al.* Cytokine alterations in lichen sclerosus: an immunohistochemical study. *Br J Dermatol* 2006; **155**: 931–40.
3 Weinberg A. *In vitro* hydroxyurea decreases Th1 cell-mediated immunity. *Clin Diagn Lab Immunol* 2001; **8**:702–5.
4 Silver RT. Treatment of polycythemia vera. *Semin Thromb Hemost* 2006; **32**:437–42.

Conflicts of interest: none declared.

Metastatic calcinosis cutis presenting as bilateral vulval cysts

DOI: 10.1111/j.1365-2133.2007.07981.x

SIR, Calcinosis cutis refers to cutaneous deposition of calcium salts in the skin. It may be classified as metastatic, dystrophic, idiopathic or iatrogenic type based on the aetiopatho-