

1. Scully C, Carrozzo M, Gandolfo S, Puiatti P, Monteil R. Update on mucous membrane pemphigoid: a heterogeneous immune-mediated subepithelial blistering entity. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999; 88: 56-68.
2. Calabresi V, Carrozzo M, Cozzani E, Arduino P, Bertolusso G, Tirone F, Parodi A, Zambruno G, Di Zenzo G. Oral pemphigoid auto-antibodies preferentially target BP180 ectodomain. *Clin Immunol* 2007; 122: 207-13.
3. Poskitt L, Wojnarowska F. Treatment of cicatricial pemphigoid with tetracycline and nicotinamide. *Clin Exp Dermatol* 1995; 20: 258-9.
4. Poskitt L, Wojnarowska F. Minimizing cicatricial pemphigoid orodysnia with minocycline. *Br J Dermatol* 1995; 132: 784-9.
5. Megahed M, Schmiedeberg S, Becker J, Ruzicka T. Treatment of cicatricial pemphigoid with mycophenolate mofetil as a steroid-sparing agent. *J Am Acad Dermatol* 2001; 45: 256-9.
6. Ingen-Housz-Oro S, Prost-Squarcioni C, Pascal F, Doan S, Brette MD, Bachelez H, Dubertret L. Cicatricial pemphigoid: treatment with mycophenolate mofetil. *Ann Dermatol Venereol* 2005; 132: 13-6.

Inflammation of actinic keratoses with capecitabine therapy for colon cancer

Capecitabine is an orally administered systemic prodrug of 5-fluorouracil (5-FU), currently used as an anti-neoplastic agent. Tumor-specific conversion to the active drug improves tolerability and intra-tumor drug concentrations. It is currently approved for colorectal and breast cancer, and has been used either alone or in combination with other chemotherapy regimens [1].

Actinic keratoses are focal areas of abnormal keratinocyte proliferation and differentiation, presenting as keratotic lesions on chronically light-exposed skin. They are usually considered a premalignant lesion, with a low risk of progression to invasive squamous cell carcinoma. There is vast experience with topical use of 5-FU on actinic keratoses, with randomised controlled trials confirming efficacy [2, 3].

We describe a 67-year-old Caucasian male, with a 3-year history of rectal adenocarcinoma, with local recurrences treated with surgery and radiotherapy. Chemotherapy was started with capecitabine, 2,150 mg (1,250 mg/m²) twice daily for 2 weeks, followed by a 1 week rest period. Four days after starting capecitabine, the patient developed areas of inflammation on the scalp and face, and was sent for dermatological assessment. On examination, all inflammatory aspects were restricted to areas of previous actinic keratoses, with erythema and localised pain (figure 1). The chemotherapy schedule was maintained, and after 8 weeks no remaining inflammatory activity was present, with complete clearing of the actinic keratoses.

After 6 weeks of therapy he also developed erythema and skin darkening on the palms and soles, with mild discomfort. This frequent side-effect of anti-neoplastic chemotherapies is called hand-foot syndrome or palmo-plantar erythrodysesthesia, and 5-FU and derivatives are the most often implicated agents [4].

Inflammation of actinic keratoses with the systemic use of capecitabine has very rarely been reported in the literature [5, 6]. It could be considered an almost expected side-effect, due to the efficacy of topically applied 5-FU on actinic keratoses, and probably has been under-reported. It is important to recognise this reaction and advise patients with actinic keratoses of the potential inflammatory response, assuring them that it will be limited and may even be beneficial. ■

Conflict of interest. None of the authors has any affiliation or financial involvement in any organization or entity with a direct financial interest in the subject matter or materials discussed in the manuscript.

Dermatology Department, Hospital dos Capuchos, Alameda Santo António dos Capuchos, 1150 – 314 Lisbon, Portugal
vvsferreira@netcabo.pt

Vasco VIEIRA
SERRÃO
Ana BARATA FEIO



Figure 1. A) and B) Inflammation restricted to areas of actinic keratoses; C) and D) Complete improvement, with resolution of the actinic keratoses, 8 weeks after capecitabine therapy.

1. Hwang JJ, Marshall JL. Capecitabine: fulfilling the promise of oral chemotherapy. *Expert Opin Pharmacother* 2002; 3: 733-43.
2. Stockfleth E, Kerl H; Guideline Subcommittee of the European Dermatology Forum. Guidelines for the management of actinic keratoses. *Eur J Dermatol* 2006; 16: 599-606.
3. de Berker D, McGregor JM, Hughes BR; British Association of Dermatologists Therapy Guidelines and Audit Subcommittee. Guidelines for the management of actinic keratoses. *Br J Dermatol* 2007; 156: 222-30.
4. Janusch M, Fischer M, Marsch WC, et al. The hand-foot syndrome - a frequent secondary manifestation in antineoplastic chemotherapy. *Eur J Dermatol* 2006; 16: 494-9.
5. Lewis KG, Lewis MD, Robinson-Bostom L, Pan TD. Inflammation of actinic keratoses during capecitabine therapy. *Arch Dermatol* 2004; 140: 367-8.
6. Higa GM, Kovach RF, Abraham J. Actinic keratosis and capecitabine therapy. *J Oncol Pharm Pract* 2005; 11: 151-3.

Progressive nodular histiocytosis – A five-year follow up

Progressive nodular histiocytosis (PNH) was first described as progressive nodular histiocytoma by Taunton *et al.* [1]. PNH is an extremely rare skin disorder which belongs to the group of non-Langerhans cell histiocytosis. Clinically, the disease is characterized by the progressive appearance of widespread xanthomatous papules and nodules without showing spontaneous remission. The histological appearance of the disease is related to juvenile xanthogranuloma (JXG). Herein, we report a 10-year-old girl who was first diagnosed as JXG but, for 5 years, had had progressively growing cutaneous lesions during follow-up.