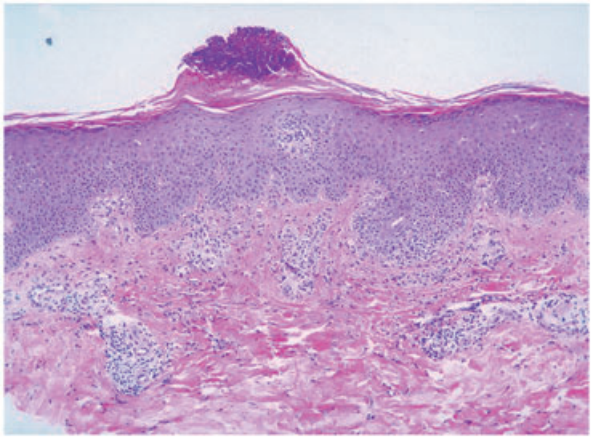

Figure 2 Histopathological examination of a 4-mm punch biopsy specimen revealed a slight psoriasiform hyperplasia with parakeratosis and a intracorneal microabscess of neutrophils (haematoxylin and eosin; original magnification × 100).

but when the lesions became extensive, the patient required psoralen ultraviolet A therapy, which led within 2 months to eventual blanching, except for isolated small plaques, which have been treated topically.

Since the diagnosis of psoriasis, the patient has received nine additional infusions of infliximab, every 4–6 weeks, in order to control perianal CD. New skin lesions of psoriasis, involving mainly the flexural areas, have continued to appear, but have been adequately controlled with topical treatment.

Infliximab treatment is generally well tolerated and has been associated with few adverse effects. Cutaneous reactions attributed to infliximab have been described, including a psoriasiform eruption with a histological lichenoid pattern,[1] a case of pustular psoriasis[2] and a case of plaque-type psoriasis,[3] in three patients who received infliximab treatment for CD, chronic ulcerative colitis and rheumatoid arthritis, respectively. An additional patient developed psoriasis vulgaris and exacerbations of guttate psoriasis following treatment of her rheumatoid arthritis with etanercept, another anti-TNF-α agent.[3]

The requirement for increased frequency of infliximab infusions to maintain its therapeutic effect may be related to the development of anti-infliximab antibodies or decreased serum levels of infliximab, but we were not able to confirm this hypothesis, as these tests are not available to us. The clinical efficacy of infliximab on our patient's CD was maintained, even though the degree of perianal involvement may have been compounded by the development of inverse psoriasis.

Our patient had not been treated with any drug known to be associated with the development or aggravation of psoriasis, such as beta-blockers, lithium, antimalarials drugs, angiotensin-converting enzyme inhibitors, tetracyclines or nonsteroidal anti-inflammatory agents, except celecoxib, which had ceased to be administered months before the rash first appeared.

The mechanisms underlying this unusual and apparently paradoxical adverse effect are debatable, but it is of interest that treatment with TNF-α has previously been found to be effective in at least some patients with psoriasis.[4] There may be heterogeneity in pathogenesis, predominant inflammatory pathways and therapeutic response among different subtypes of psoriasis. The potential genetic heterogeneity of inverse-flexural psoriasis compared with guttate psoriasis and psoriasis vulgaris may eventually be confirmed, as with palmoplantar pustulosis,[5] which has been reported to occur following infliximab therapy in 3% of patients with spondyloarthropathy.[6]

L. Peramiquel, L. Puig, J. Dalmau, E. Ricart,* E. Roé and A. Alomar
*Departments of Dermatology and *Gastroenterology, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain*
Correspondence: Dr L. Peramiquel
E-mail: lperamiquel@hsp.santpau.es
Conflict of interest: none declared.
Accepted for publication 4 April 2005

References

1 Verea MM, Del Pozo J, Yebra-Pimentel MT, Porta A, Fonseca E. Psoriasiform eruption induced by infliximab. *Ann Pharmacother* 2004; **38**: 54–7.
2 Thurber M, Feasel A, Stroehlein J, Hymes SR. Pustular psoriasis induced by infliximab. *J Drugs Dermatol* 2004; **3**: 439–40.
3 Dereure O, Guillot B, Jorgensen C, Cohen JD, Combes B, Guilhou JJ. Psoriatic lesions induced by antitumor necrosis factor-alpha treatment: two cases. *Br J Dermatol* 2004; **150**: 506–25.
4 Creaven PJ, Stoll HL Jr. Response to tumor necrosis factor in two cases of psoriasis. *J Am Acad Dermatol* 1991; **24**: 735–7.
5 Asumalahti K, Ameen M, Suomela S *et al.* Genetic analysis of PSORS1 distinguishes guttate psoriasis and palmoplantar pustulosis. *J Invest Dermatol* 2003; **120**: 627–32.
6 Baeten D, Kruithof E, Van den Bosch F *et al.* Systematic safety follow up in a cohort of 107 patients with spondyloarthropathy treated with infliximab: a new perspective on the role of host defence in the pathogenesis of the disease? *Ann Rheum Dis* 2003; **62**: 829–34.

Anaphylaxis after dexpanthenol exposure by multivitamin tablets

doi: 10.1111/j.1365-2230.2005.01881.x

A 30-year-old woman was admitted to the emergency department with oedema of the face including the eyelids and tongue, and dyspnoea, dizziness and faintness. Symptoms had started 20 minutes after breakfast. An emer-

gency physician was called immediately, and treatment was started. A few weeks prior to admission, the patient had observed itching of the lips and face, a coated tongue and swelling of the eyelids. The symptoms worsened over time, always starting shortly after breakfast. She had no history of atopic dermatitis or pollinosis. As a mother of five children, the patient emphasized the necessity of food supplementation by multivitamin tablets, which she took more or less regularly after breakfast with orange juice. The common ingredients of these tablets (Nutrilite® Doublex (I and II) and Nutrilite® vitamin B complex, both Amway, USA) are, per tablet: vitamin B1 (1.4 mg), B2 (1.6 mg), B6 (2 mg), B12 (1 μg), folic acid (167 μg), and dexpanthenol (3.33 mg), with additional substances being cellulose, lactose, calcium stearate, siliconoxid and glycerine.

Several weeks after recovery from the anaphylactic reaction, scratch testing with the patient's tablets was performed; all three tablets gave positive reactions (Fig. 1). The scratch and prick tests are both used to analyse early skin reactions. While the prick test is used for standardized test solutions, the scratch test is reserved for the analysis of solid and native substances as in our case. With a standardized lancet (Mediprick®; Seropax, Germany) the skin was scratched on an area of 5 mm. A minimal amount (tip of the spatula) of the pulverized tablet was put on the skin scratch and dissolved with a drop of 0.9% sodium chloride solution. Serum total IgE was low at 9.0 KU/L. Specific IgE values for casein (CAP®; Pharmacia, Freiburg, Germany) showed no increase. Prick skin tests of food extracts (Allergopharma®, Reinbek, Germany) and scratch test of common preservatives and orange juice were negative.

Fifteen minutes after scratch testing, the patient felt a tightness in her throat and she developed a facial oedema and breathlessness. Treatment was started with intravenous prednisolone 250 mg, clemastinhydrogenfumarate 2 mg and a fenoterol inhaler.

Under emergency conditions, a further skin scratch test was performed to identify the causative ingredient in the tablets. Vitamin B1 (10 mg), B2 (10 mg), B6 (40 mg), B12 (1 mg), and folic acid (5 mg) (all per tablet) showed no reactions compared with the positive control (histamine hydrochloride 10 mg/mL). Testing of dexpanthenol by friction test (dexpanthenol 5% in purified vaseline; Hermal®, Hamburg, Germany) led to pruritus and erythema in the tested skin area and to pruritus on the lips and a coated tongue. Consequently, no further tests were performed.

Confirming the skin test results, the patient remembered that dexpanthenol-containing sun cream had caused pruritus and local urticaria previously.

To our knowledge, this is the first report of a severe systemic reaction induced by dexpanthenol. Owing to the frequent use of dexpanthenol in a variety of cosmetic and topical medical products allergic contact dermatitis to dexpanthenol is often described.[1,2] Contact urticaria caused by a dexpanthenol-containing hair conditioner has been described in a single case report.[3] In this report no systemic symptoms had occurred.

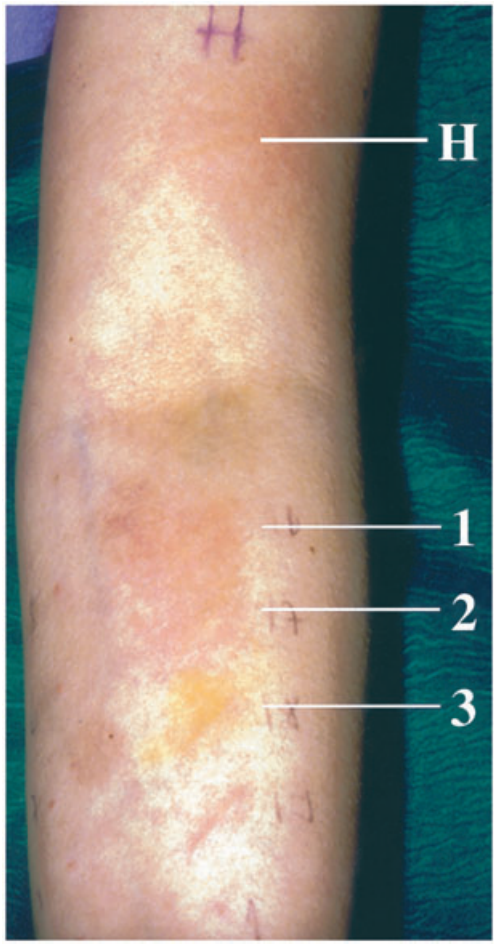

Figure 1 Scratch test of Nutrilite® doublex I (1), Nutrilite® doublex II (2), and Nutrilite® Vitamin B complex (3), compared with a positive control (H, histamine hydrochloride). A negative control (0.9% sodium chloride solution) was applied on the right arm and did not show any reaction. Scratch test of the same substances with a control person was negative. The photograph was taken approximately 45 min after scratch testing, when the patient had recovered from the systemic allergic reaction.

Dexpanthenol is the stable alcohol of pantothenic acid ($HOCH_2C(CH_3)_2CHOHCONH(CH_2)_2CH_2OH$), a water-soluble vitamin (B5) that is an essential constituent of coenzyme A (CoA). It is synthetically produced from 2,4-dihydroxy-3,3-dimethylbutyric acid and β-alanine.[4] Inside the cell, dexpanthenol is readily oxidized to pantothenic acid, which has an anti-inflammatory effect. It is applied to improve wound healing by topical administration and is used as a pro-vitamin in the cosmetic and pharmaceutical industry. The allergologically relevant component of dexpanthenol remains unclear, but the involvment of β-alanine in the pathomechanism has been discussed.[1]

As pantothenic acid is found in a wide variety of foods,[4] a deficiency is rare, and oral substitution not necessary. At the present time, nutrients such as pantothenic acid have been approved for food fortification.[5] In the light of this case report, the risk–benefit ratio of food fortification with dexpanthenol should be reassessed.

H. Röckmann, S. Goerdt and C. Bayerl
Department of Dermatology, Venereology, and Allergology, University Medical Center Mannheim, Ruprecht-Karl University of Heidelberg, Mannheim, Germany
E-mail: h.roeckmann@dkfz.de
Conflict of interest: none declared.
Accepted for publication 11 March 2005

References

1 Stables GI, Wilkinson SM. Allergic contact dermatitis due to panthenol. *Contact Dermatitis* 1998; **38**: 236–7.
2 Hemmer W, Braun R, Wolf-Abdolvahab S, Focke M, Gotz M, Jarisch R. Maintenance of hand eczema by oral pantothenic acid in a patient sensitized to dexpanthenol. *Contact Dermatitis* 1997; **37**: 51.
3 Schalock PC, Storrs FJ, Morrison L. Contact urticaria from panthenol in hair conditioner. *Contact Dermatitis* 2000; **43**: 223.
4 Lewis RJ. *Sir Hawley's Condensed Chemical Dictionary*, 12th edn. New York: Van Nostrand Reinhold, 1993.
5 Meltzer HM, Aro A, Anderson NL, Koch B, Alexander J. Risk analysis applied to food fortification. *Public Health Nut* 2003; **6**: 281–91.

Solitary anogenital xanthogranuloma

doi: 10.1111/j.1365-2230.2005.01885.x

Xanthogranuloma (XG) is an uncommon benign disorder characterized by solitary or multiple yellow-red papulonodules in the skin, and occasionally other organs. We report two cases of solitary xanthogranulomas affecting anogenital skin.

Patient 1. A 50-year-old man presented with a 6-month history of an asymptomatic papule on his glans penis. The lesion had not changed in size and there was no history of bleeding or discharge. On examination, there was a 5 × 7-mm orangey-red, domed papule located dorsomedially on the coronal rim (Fig. 1a). Skin biopsy showed a circumscribed dermal nodule composed of plump spindle cells, macrophages, foam cells, scattered lymphocytes with plasma cells and scattered eosinophils and prominent Touton giant cells, consistent with xanthogranuloma (Fig. 2a).

Patient 2. A 10-month-old baby boy presented with a 5-month history of a slowly enlarging papule on the perineum. On examination, there was a 5-mm erythematous, smooth domed papule (Fig. 1b). A skin biopsy showed

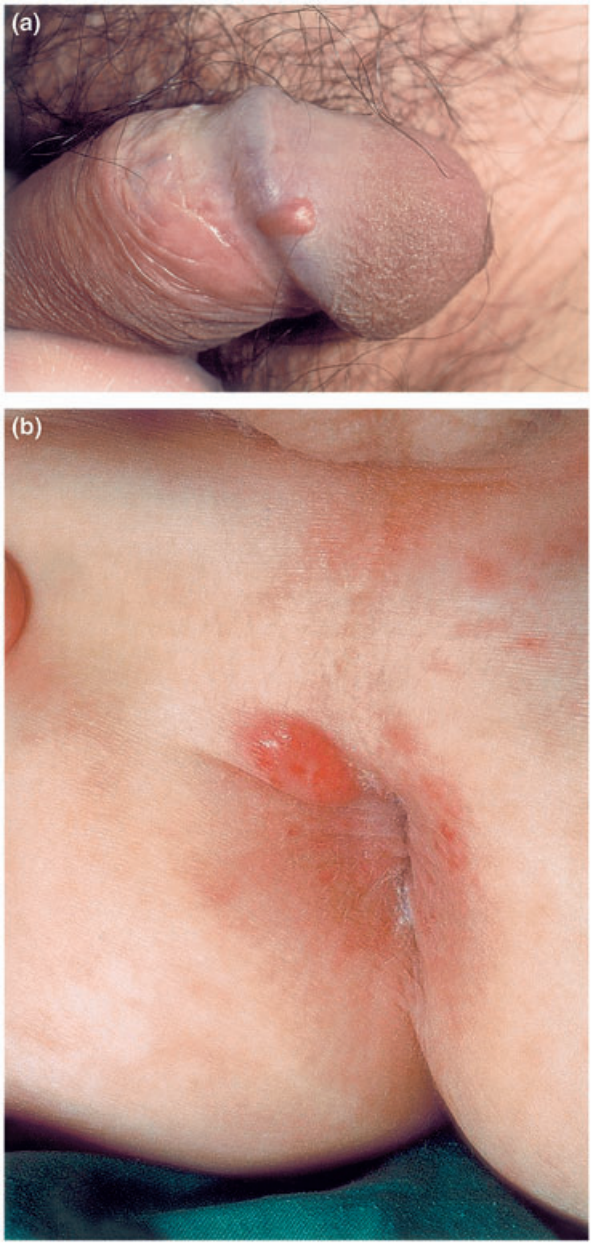

Figure 1 Xanthogranuloma: (a) glans penis, right lateral coronal rim, patient 1; (b) perineum, patient 2. Reproduced with kind permission from Elsevier Science.[5]

a diffuse dermal infiltrate of vacuolated to spindle-shaped macrophages, lymphocytes and multinucleate giant cells compatible with an early stage of juvenile xanthogranuloma (Fig. 2b).

XG is a benign disorder and typically presents with multiple lesions, involving the head trunk and limbs. It is predominantly a disease of infancy or early childhood, hence the term 'juvenile xanthogranuloma' introduced by Helwig and Hackney in 1954.[1] In a series of nine cases of

