accessed 14 Jul 05) over the next 60 years, we were able to calculate how the number of cases would change with time and what the shift in age profile of patients presenting to dermatologists might look like.

In making these estimates we assumed that the risk of skin cancer remained unchanged over this period. Skin cancer incidence has been increasing for a number of decades for factors more to do with lifestyle than demography and so while this assumption is unlikely to be true, we defend it for two reasons. Firstly, predicting the future incidence of skin cancer on the basis of changes in lifestyle (i.e. sun exposure) is a fragile exercise;[2] and secondly, any impact of an ageing population on skin cancer incidence could well be masked by the uncertainty associated with incorporating a model based on sun exposure and risk of disease. For similar reasons, we also neglect the impact that immunosuppressive drugs will continue to have on the incidence of skin cancer.

The relative numbers of patients presenting with NMSC in Great Britain over the next 60 years are shown in Fig. 1. It can be seen that by 2030 the number of cases presenting to dermatologists could be 50% higher than it is presently. Numbers will continue to rise until around the middle of this century and thereafter we might see them start to fall as a consequence of the falling birth rate in the last few decades of the 20th century.

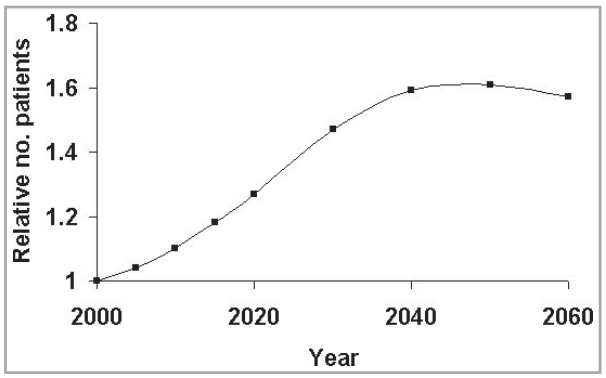

Fig 1. The relative numbers of patients presenting with non-melanoma skin cancer.

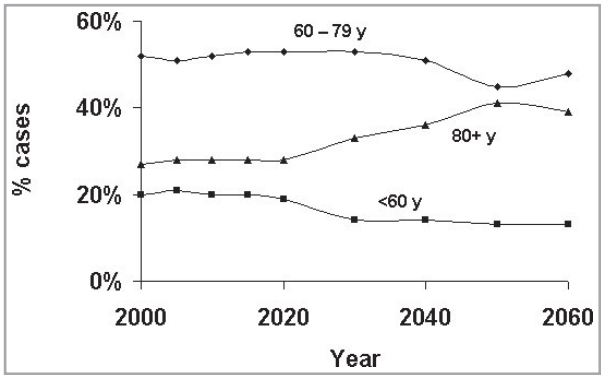

Fig 2. Future projection of skin cancer workload.

Figure 2 shows the impact of the ageing population on the age profile of patients presenting with NMSC. The profiles will remain fairly steady until around 2020 and thereafter there will a fairly rapid rise in the proportion of very elderly (80 years and over) patients attending for skin cancer management, accompanied by a corresponding decrease in the proportion of young and middle-aged (< 60 years) patients. In absolute terms, of course, the overall workload will increase, as illustrated in Fig. 1.

Regional Medical Physics Department, B.L. Diffey
Newcastle General Hospital, J.A.A. Langtry*
Newcastle upon Tyne NE4 6BE, U.K.
**Department of Dermatology,*
Sunderland Royal Hospital, Sunderland SR4 7TP, U.K.
E-mail: b.l.diffey@ncl.ac.uk

References

1 Cancer Research UK. London, 2005 (http://www.cancerresearchuk.org/aboutcancer/specificcancers/non_melanoma_skincancer?version=1 accessed 14 Jul 05).
2 Diffey BL. The future incidence of cutaneous melanoma within the U.K. *Br J Dermatol* 2004; **151**:868–72.

Conflicts of interest: none declared.

Ingestion of topical steroid triggering pustular psoriasis?

DOI: 10.1111/j.1365-2133.2005.06818.x

Sir, A 7-year-old girl was admitted as an emergency under the care of the paediatricians, with painful erythroderma, systemic upset and pyrexia of up to 40 °C. She had a history of special educational needs and hyperactive behaviour, and had previously been treated for psoriasis by her general practitioner (GP), although she had not needed to use any topical therapy for 6 months. There was a strong family history of psoriasis, with both her mother and her maternal grandmother affected. Seven days prior to admission she had ingested more than half of a 100-g tube of clobetasone butyrate (Eumovate®; GlaxoSmithKline, Uxbridge, U.K) belonging to her mother. Forty-eight hours after this she began to develop redness and scaling initially on the face and subsequently on the lower limbs. When the GP was consulted the following day pustules were noted, and oral flucloxacillin suspension 125 mg four times daily was commenced along with topical Aqueous Cream. Despite these measures the rash became more widespread, resulting in admission.

On examination, the patient was pyrexial and erythrodermic. The skin was tender to palpation, with numerous visible

small pustules and areas of desquamation (Fig. 1). Thick scale was present in the scalp. The most likely diagnosis was generalized pustular psoriasis (GPP), von Zumbusch type. The differential diagnoses included acute generalized exanthematous pustulosis (AGEP), seborrhoeic dermatitis, severe skin sepsis and pustular miliaria. Initial investigations revealed neutrophilia (neutrophils $9{\cdot}21 \times 10^9$ L^{-1}) and raised C-reactive protein (93 mg L^{-1}), with no significant growth on skin swabs and blood cultures. A punch biopsy taken from the right thigh for histopathological assessment showed subcorneal pustules with associated mild spongiosis and epidermal hyperplasia. This was reported as consistent with a diagnosis of GPP, but was not able to rule out AGEP.

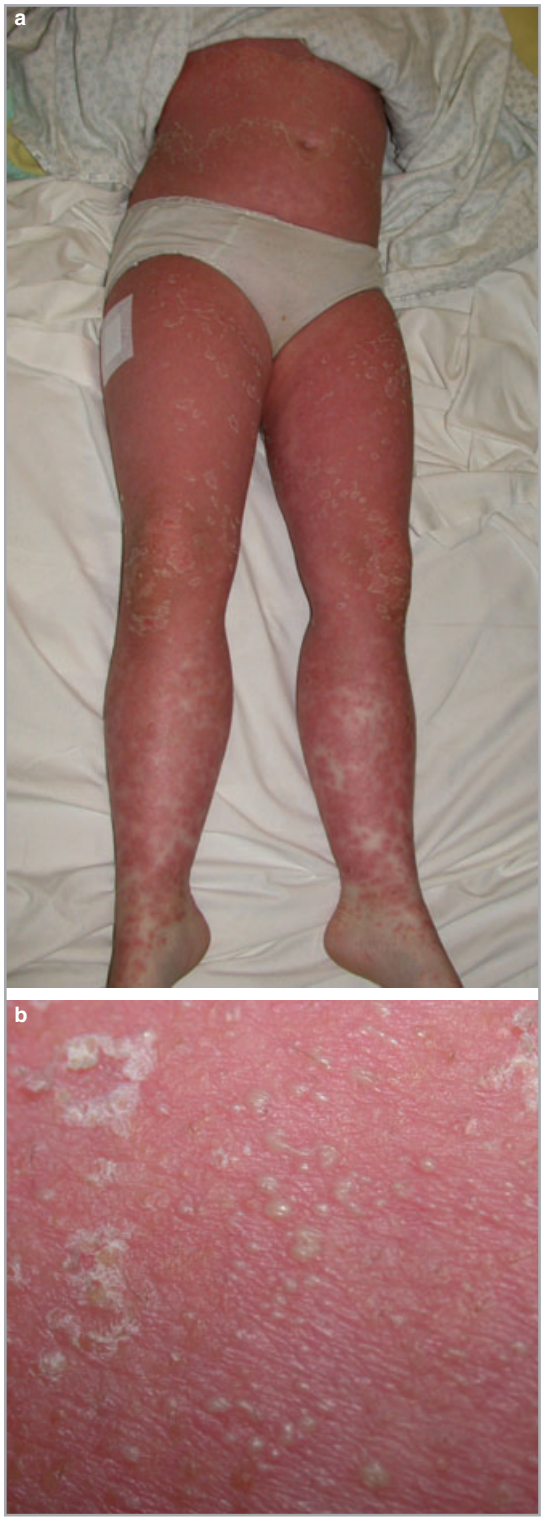

Fig 1. (a) Generalized erythroderma; (b) small pustules on an erythematous background, with areas of desquamation.

Supportive measures including intravenous fluids were given, and bland emollients were applied to the skin. Over the next 4 days the pustular element of the rash settled, as did the fever and systemic upset. Significant areas of hair loss also appeared at this time. However, as the erythroderma persisted ciclosporin 50 mg twice daily was commenced, producing a dramatic improvement within a week.

GPP is a rare disorder in children, with fewer than 200 individual cases reported in the literature.[1] It is most common in the first year of life,[2] but can occur at any age in childhood. There have even been cases of congenital GPP.[3] In contrast to psoriasis in general and to adult GPP, there is a male preponderance with an approximate male/female ratio of 3 : 2·1 There may be a personal history of psoriasis vulgaris in up to 59% of cases, with as many as one in four having a positive family history.[2] It tends to be more benign than in adults, with no reported fatalities.[2] Common triggers include infection, ultraviolet B radiation and vaccination as well as topical and oral corticosteroid use.[4–6] Systemic therapies used with success in childhood GPP include retinoids, methotrexate and ciclosporin.[6–8]

AGEP was the most likely alternative diagnosis. This is a generalized pustular eruption usually triggered by drugs such as beta-lactam and macrolide antibiotics.[6] There is one case report of this condition induced by administration of a dexamethasone injection, with subsequent patch testing producing a pustular response.[9] However, the preservatives and even local anaesthetic in the particular agent used could also have been responsible. AGEP may have appeared in our patient therefore secondary to exposure either to the steroid or to excipients present in the cream, and the subsequent flare of psoriasis may have been as a result of koebnerization of her pre-existing disease. We felt that the strong personal and family history of psoriasis, coupled with the potential corticosteroid trigger from the ingested clobetasone butyrate, favoured GPP as the diagnosis from the outset. The rapid appearance of hair loss, far too soon to be a telogen effluvium, is seen in GPP and is a further indication to support the diagnosis. While histology was inconclusive in differentiating between psoriasis and AGEP, as is often the case, the latter typically settles rapidly on withdrawal of the causative agent, something that clearly did not happen here.

Did the ingestion of topical steroid trigger acute GPP in this case? Oral steroids and particularly their withdrawal are well-known aetiological factors in this disorder.[4–6] The steroid content of a 100-g tube of Eumovate is equivalent to 50 mg of prednisolone (verbal communication, Medicines Information Unit, Pharmacy, Solihull Hospital). The amount our patient

ingested therefore equates to 25–30 mg of prednisolone, a reasonable adult dose and, for this child, 1 mg kg^{-1}. There are no available data concerning the likely absorption of corticosteroid from a topical preparation such as Eumovate in a child of this age; however, it would be a coincidence if this event were not involved in the development of GPP in this case. While unlikely to be a common occurrence in everyday clinical practice it does appear that ingestion of topical steroid preparations may be a potential trigger for acute GPP.

*Departments of Dermatology and *Paediatrics, Heartlands and Solihull NHS Trust (Teaching), Solihull Hospital, Lode Lane, Solihull, West Midlands B91 2JL, U.K.*
S.D. Orpin
J. Hafiji
H.M. Goodyear*
A. Salim
E-mail: s.orpin@nhs.net

References

1 Cassandra M, Conte E, Cortez B. Childhood pustular psoriasis elicited by the streptococcal antigen: a case report and review of the literature. *Pediatr Dermatol* 2003; **20**:506–10.
2 Zelickson B, Muller S. Generalised pustular psoriasis in childhood. *J Am Acad Dermatol* 1991; **24**:186–94.
3 Beylot C, Puissant A, Bioulac P *et al.* Particular clinical features of psoriasis in infants and children. *Acta Derm Venereol (Stockh)* 1979; **59** (Suppl. 87):95–7.
4 Ryan T, Baker H. The prognosis of generalized pustular psoriasis. *Br J Dermatol* 1971; **85**:407–11.
5 Ryan T, Baker H. Systemic corticosteroids and folic acid antagonists in the treatment of generalized pustular psoriasis. Evaluation and prognosis based on the study of 104 cases. *Br J Dermatol* 1969; **81**:134–45.
6 Mengesha Y, Bennett M. Pustular skin disorders: diagnosis and treatment. *Am J Clin Dermatol* 2002; **3**:389–400.
7 Burden A. Management of psoriasis in childhood. *Clin Exp Dermatol* 1999; **24**:341–5.
8 Leman J, Burden A. Psoriasis in children: a guide to its diagnosis and management. *Paediatr Drugs* 2001; **3**:673–80.
9 Demitsu T, Kosuge A, Yamada T *et al.* Acute generalised exanthematous pustulosis induced by dexamethasone injection. *Dermatology* 1996; **193**:56–8.

Conflicts of interest: none declared.

A case of Bazex–Dupré–Christol syndrome associated with multiple genital trichoepitheliomas

DOI: 10.1111/j.1365-2133.2005.06819.x

Sir, Bazex–Dupré–Christol syndrome was first described by Bazex *et al.* in 1964.[1] It is characterized by a triad of congenital hypotrichosis, follicular atrophoderma (affecting the dorsa of the hands and feet, the face, and extensor surfaces of the elbows or knees) and the development of basocellular neoplasms (including basal cell naevi and basal cell carcinomas) from the second decade onwards.[2–4] Other reported features include associated hair shaft abnormalities (pili torti and trichorrhexis nodosa) admixed with hypotrichosis, prominent milia affecting the face, hypohidrosis, pinched nose with hypoplastic nasal alae and prominent columella, atopic diathesis with comedones, keratosis pilaris, joint hypermobility, lingua plicata and hyperpigmentation of the forehead.[5,6]

A 3-year-old girl presented at the age of 2 years with increasing numbers of multiple brown asymptomatic papules over the genital area and medial aspect of the thighs (Fig. 1a). She had hypotrichia since birth (Fig. 1b), and had prominent facial milia and follicular atrophoderma of the cheeks (Fig. 1c) consistent with a diagnosis of Bazex–Dupré–Christol syndrome. Multiple members of her family showed similar features (the patient's grandfather was originally reported by Gould and Barker in 1978[3]) (Fig. 2). Her mother had features of facial milia, follicular atrophoderma of the cheeks and dorsa of the hands, hypotrichia (since birth), hypohidrosis and axillary hidradenitis suppurativa. The patient's newborn brother was born with hypotrichia, mild erythema and ichthyosis and has subsequently developed multiple facial milia, suggesting that he also has Bazex–Dupré–Christol syndrome. A biopsy from the labial area in our patient revealed multiple dermal nodules consisting of well-defined arrangements of rounded nests of basaloid cells in a loose lobular (or 'organoid') pattern (Fig. 1d). A loose myxoid stroma without retraction artefact was present surrounding the interconnecting strands of basaloid cells (Fig. 1e). Well-formed papillary mesenchymal bodies, mitotic figures and necrosis were not seen. A diagnosis of multiple benign trichoepitheliomas occurring in Bazex–Dupré–Christol syndrome was made.

Bazex–Dupré–Christol syndrome is thought to be transmitted by an X-linked dominant pattern of inheritance.[7] The genetic defect has been reported to localize to Xq24-q27.[8] Neither trichoepitheliomas nor hidradenitis suppurativa have been reported in association with Bazex–Dupré–Christol syndrome. Most of the reported clinical features of Bazex–Dupré–Christol syndrome demonstrate abnormalities in the development of follicular structures, suggesting that the defective gene codes for a protein intimately involved in follicular differentiation and development.

Trichoepitheliomas are benign tumours which arise from cells derived from the hair follicle.[9] Lesions may occur singly as a papule or nodule (up to 2 cm in diameter) or as multiple 2–8 mm diameter skin-coloured papules on the face of children or young adults centred around the nasolabial folds and preauricular regions. Trichoepitheliomas rarely affect the vulval region.[10] The major histological and clinical differential diagnosis of trichoepithelioma includes basal cell carcinoma and trichoblastoma.[9,11] There is considerable controversy regarding the histological definition of these follicular tumours and differentiation between these entities can be difficult, although various features are reported to be helpful in differentiating trichoepitheliomas from basal cell carcinomas.[11,12] Furthermore, it remains unclear whether basal cell carcinomas may arise from trichoepitheliomas.[9]