30 years: interestingly, this age group had the highest incidence rate ratio of all age groups analysed.

It would appear from the limited data presented that there may be evidence of comorbid disease, associated with metabolic syndrome, detected at an early age in young people with psoriasis. Clearly more work needs to be done in this area. In the interim this provides an opportunity to reinforce healthy lifestyle choices in children in general but particularly those with psoriasis and raises the question of whether we should be monitoring for associated features of metabolic syndrome in this age group.

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References

1 Gisondi P, Tessari G, Conti A *et al.* Prevalence of metabolic syndrome in patients with psoriasis: a hospital-based case–control study. *Br J Dermatol* 2007; **157**:68–73.
2 Naldi L, Mercuri SR. Epidemiology of comorbidities in psoriasis. *Dermatol Ther* 2010; **23**:114–18.
3 Benoit S, Hamm H. Childhood psoriasis. *Clin Dermatol* 2007; **25**:555–62.
4 Augustin M, Galeske G, Radtke MA *et al.* Epidemiology and comorbidity of psoriasis in children. *Br J Dermatol* 2010; **162**:633–6.
5 Kämpfe SM, Augustin M, Schäfer I *et al.* Prevalence and health care situation of juvenile psoriasis in Germany. *Exp Dermatol* 2012; **21**:e21 (Abstract).
6 Zhu KJ, He SM, Zhang C *et al.* Relationship of the body mass index and childhood psoriasis in a Chinese Han population: a hospital-based study. *J Dermatol* 2012; **39**:181–3.
7 Boccardi D, Menni S, La Vecchia C *et al.* Overweight and childhood psoriasis. *Br J Dermatol* 2009; **161**:484–6.
8 Wu Y, Lin Y, Liu H-J *et al.* Childhood psoriasis: a study of 137 cases from central China. *World J Pediatr* 2010; **6**:260–4.
9 Brauchli YB, Jick SS, Meier CR. Psoriasis and the risk of incident diabetes mellitus: a population-based study. *Br J Dermatol* 2008; **159**:1331–7.

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Localized depigmentation on genital melanosis: a clue for the understanding of vitiligo

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Madam, The term 'reticulate genital pigmentation associated with localized vitiligo' was recently suggested for describing heterogeneous genital hyperpigmentation associated with depigmented macules.[1] We report three cases of acquired heterogeneous hyperpigmentation associated with achromic lesions strictly limited to the genital area. A 23-year-old man had reticulated heterogeneous pigmentation of the penis which had been present since the age of 12 years. The lesion was unique and had shown minimal enlargement during the first 4 years. Six years after the onset of the hyperpigmentation, he noticed the appearance of depigmented macules that were stable for more than 4 years (Fig. 1a). An 18-year-old man presented with testicular hyperpigmentation that progressively increased in size for 6 years. He had developed depigmented macules strictly located at the site of the hyperpigmented lesions 3 years previously (Fig. 1b). A 54-year-old man had a heterogeneous hyperpigmentation of the penis which had slowly enlarged over 3 years. Depigmented macules located on the previously hyperpigmented area had developed in the last 8 months and a new hyperpigmented lesion was noted from 6 months previously on the base of the penis (Fig. 1c, d). None of the patients reported a history consistent with an inflammatory process or had systemic symptoms. In all cases, the histological examination showed a moderate hyperplasia of the epidermis and hyperpigmentation of the basal layers without melanocytic proliferation (Fig. 2a). Serial examination of the biopsies revealed a CD8+ T-cell infiltrate located around melanocytes (Fig. 2b).

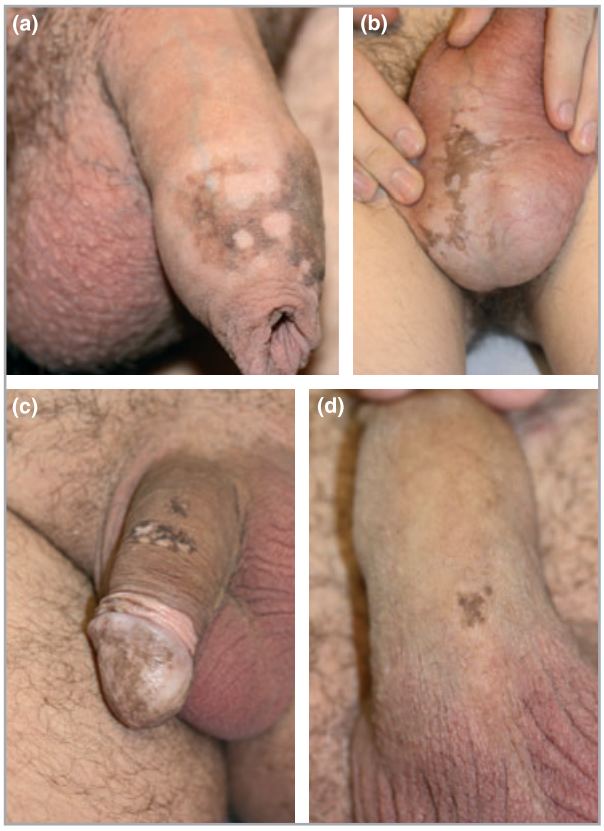

Fig 1. (a) Case 1. Reticulated heterogeneous pigmentation of the penis associated with achromic macules. (b) Case 2. Hyperpigmented lesions with irregular borders and achromic macules of the scrotum. (c, d) Case 3. (c) Reticulated hyperpigmentation with achromic macules. (d) Recent hyperpigmented lesion of the ventral side of the penis. Note the onset of the depigmentation strictly localized around and within the hyperpigmented macules.

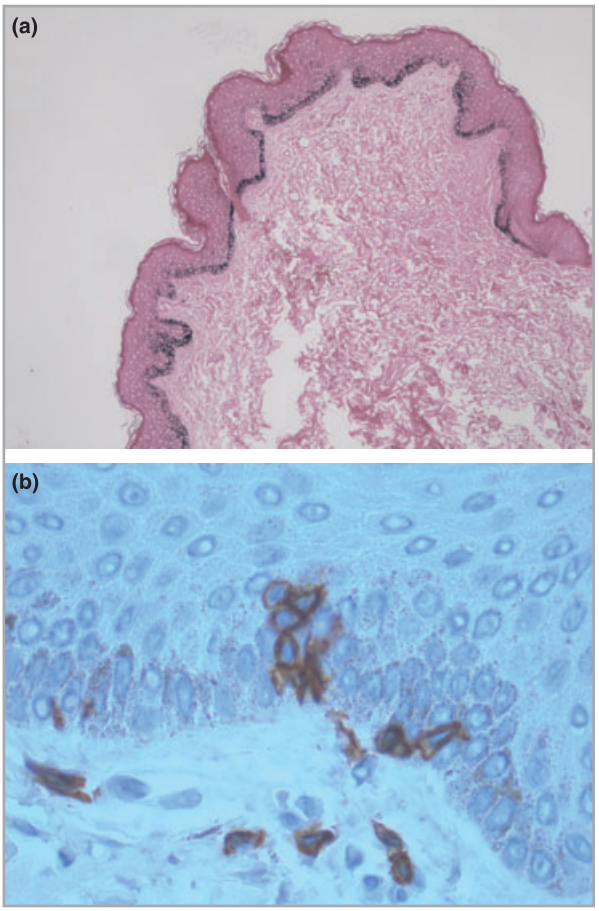

Fig 2. (a) Haematoxylin and eosin staining of a hyperpigmented lesion (×100) showing hyperpigmentation of the basal layers and moderate hyperplasia of the epidermis without melanocytic proliferation. (b) Anti-CD8 immunostaining of a hyperpigmented macule (×1000). Note the lymphocytic infiltrate in the epidermis surrounding melanocytes.

Melanotic genital macules have been described using several different terminologies. The term 'genital melanosis' is used to describe pigmented macules with an increased epidermal pigmentation while 'genital lentiginosis' is preferred when the melanocyte number is increased. However, a review of published cases showed a clinical and histological continuum between these two descriptions, supporting a single entity. Similarly, hypopigmented lesions such as those reported here and those published as reticulate genital pigmentation associated with localized vitiligo, can be found in the careful analysis of previously published cases of genital melanosis.[2] A lymphocytic infiltrate surrounding melanocytes was described in the largest histological series of genital melanosis.[3] A T-cell infiltrate closely located around melanocytes was also observed in the histological examinations that we performed. As in vitiligo, this infiltrate is probably responsible for the depigmentation.[4,5] Such a depigmentation is a very interesting phenomenon. None of the patients affected had a personal or familial history of vitiligo. The depigmentation always occurred after the melanosis had developed and was restricted to the previously hyperpigmented areas. Despite long-term follow-up, no depigmented macule has ever been observed apart from the genital melanosis lesions. This strongly suggests that the immune reaction leading to the disappearance of melanocytes was triggered by a local stimulus. Rather than a single entity, localized depigmentation on genital melanosis should be considered as an immune reaction against melanocytic lesions such as those observed in halo naevus, Meyerson naevus and regression in melanoma.[6,7] Quantitative or qualitative defects in melanocytic antigen presentation in genetically predisposed patients might explain the restriction of the depigmentation to previously hyperpigmented lesions. Then, depending on the genetic background, but probably on other factors that need to be addressed, the immune response might spread from the initial site or not. A parallel could be drawn with halo naevus, in which some patients develop generalized vitiligo and some only have depigmentation around the naevus.[8] The study of these pathophysiological mechanisms should provide a better understanding of the immune response against melanocytes.

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References

1 Romero-Mate A, Minano-Medrano R, Najera-Botello L *et al.* Reticulate genital pigmentation associated with localized vitiligo. *Arch Dermatol* 2010; **146**:574–5.
2 Barnhill RL, Albert LS, Shama SK *et al.* Genital lentiginosis: a clinical and histopathologic study. *J Am Acad Dermatol* 1990; **22**:453–60.
3 Breathnach AS, Balus L, Amantea A. Penile lentiginosis. An ultrastructural study. *Pigment Cell Res* 1992; **5**:404–13.
4 Mantovani S, Garbelli S, Palermo B *et al.* Molecular and functional bases of self-antigen recognition in long-term persistent melanocyte-specific CD8+ T cells in one vitiligo patient. *J Invest Dermatol* 2003; **121**:308–14.
5 Harris JE, Harris TH, Weninger W *et al.* A mouse model of vitiligo with focused epidermal depigmentation requires IFN-gamma for autoreactive CD8(+) T-cell accumulation in the skin. *J Invest Dermatol* 2012; **132**:1869–76.
6 Van Geel NA, Mollet IG, De Schepper S *et al.* First histopathological and immunophenotypic analysis of early dynamic events in a patient with segmental vitiligo associated with halo nevi. *Pigment Cell Melanoma Res* 2010; **23**:375–84.
7 Speeckaert R, Van Geel N, Vermaelen KV *et al.* Immune reactions in benign and malignant melanocytic lesions: lessons for immunotherapy. *Pigment Cell Melanoma Res* 2011; **24**:334–44.
8 Van Geel N, Vandenhaute S, Speeckaert R *et al.* Prognostic value and clinical significance of halo naevi regarding vitiligo. *Br J Dermatol* 2011; **164**:743–9.

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