More severe local reactions include extensive or protracted ulceration, purulent discharge, and suppurative lymphadenitis with draining sinuses. Other uncommon complications include keloid formation, lupus vulgaris, erythema multiforme, hypersensitivity reactions, lichen scrofulosorum, tuberculids, systemic infections and development of basal cell carcinoma in the BCG scar.[2]

Pilomatricoma, or benign calcifying epithelioma of Malherbe, is the commonest hair-follicle tumour and is composed of cells resembling those of the hair matrix, which undergo 'mummification' and may calcify. It typically occurs on the head, neck or upper limb. The tumour may show a wide variety of signs,[3] and may be subcutaneous, ulcerating, perforating, pigmented (with haemosiderin), keratotic, vascular, anetodermic, bullous, or telangiectatic and resembling basal cell carcinoma.[4] To our knowledge, pilomatricoma has not been reported to arise in scars or following vaccination, but a history of preceding trauma was reported in two cases.[4,5] Tumours found in vaccination scars could be induced by trauma, persistent inflammation/wound healing, scarring, and/or the inoculated attenuated agent itself, but the underlying pathogenesis of such lesions is not understood. Although coincidental occurrence in our case cannot be excluded, the history supports a true association. We suggest that pilomatricoma be included in the differential diagnoses of chronic ulcerating lesions arising at BCG vaccination sites.

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References

1 Dostrovsky A, Sagher F. Dermatological complications of BCG vaccination. *Br J Dermatol* 1963; **75**: 181–92.
2 Gawkrodger DJ. Mycobacterial infections. In: *Textbook of Dermatology* (Champion RH, Burton JL, Burns DA, Breathnach SM, eds), 6th edn, Vol. 2. Oxford: Blackwell Scientific Publications, 1998: 1205.
3 Julian CG. A clinical review of 209 pilomatricomas. *J Am Acad Dermatol* 1998; **39**: 191–5.
4 Inui S, Kanda R, Hata S. Pilomatricoma with a bullous appearance. *J Dermatol* 1997; **24**: 57–9.
5 Sevin K, Can Z, Yilmaz S *et al.* Pilomatrixoma of the earlobe. *Dermatol Surg* 1995; **21**: 245–6.

Acne induced by 'Sus' and 'Deca'

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A 22-year-old man presented with a 4-month history of an acute onset of a severe pustular eruption on his chest and back. He had been commenced on minocycline 100 mg daily by his general practitioner (GP). Two weeks prior to attending the dermatology department, this had been increased to 100 mg twice daily. The patient reported that he had been improving on this treatment. He had had a similar episode 1 year previously, which had taken 6 months to settle. There had been no other skin problems prior to this and he had not had acne as a teenager.

On examination, we found a muscular man, with numerous scars on his back and chest that in some places were hypertrophic. There were comedones and active pustular and nodular lesions on the back and chest and a crusted area overlying the sternum (Fig. 1). His face was clear.

The patient had admitted to his GP that he had been using anabolic steroid injections for 4 weeks to help increase muscle mass as part of his body-building programme, and that the eruption had started during this time. He admitted to us that the episode 1 year prior to presentation had also been preceded by the use of anabolic steroids. On both occasions, he had purchased substances known to him as 'Sus' and 'Deca', which he had been administering intramuscularly on a weekly basis. He had stopped using these after 4 weeks on both occasions.

A diagnosis of anabolic–androgenic steroid (AAS)-induced acne was made and he was advised to continue with the minocycline 100 mg twice daily for a further 3 months and to use Vioform HC hydrocortisone ointment to the crusted area for 2 weeks. He made good progress, and after 6 months of minocycline therapy had very few active lesions. He declined the option of oral isotretinoin.

The use of self-administered AAS by recreational body-builders is well recognized in the UK. A feature of their use is polypharmacy with large doses of both human and veterinary preparations.[1,2] 'Sus' and 'Deca' are terms commonly used for Sustanon, a preparation of four testosterone esters and nandrolone decanoate.

The side-effects of AAS that may cause an individual to seek the advice of a dermatologist include acne, male pattern hair loss, hirsutism and drug eruptions. O'Sullivan *et al.* found in one study that 43% of participants admitted to problems with acne while taking AAS.[3] This is one of the most frequent adverse effects of these drugs but for many users is an acceptable one. AAS increase skin surface lipids and the density of *Propionibacteria acnes*, leading to the increased likelihood of acne.[4]

The management of AAS-induced acne can be difficult, as patients are often unwilling to admit using AAS. Indeed, some individuals may not be aware that the agents they are using contain AAS. It is important to elicit a history of AAS use in such individuals and advise them to stop these drugs. Knowledge of the colloquial names of these drugs may help to obtain an accurate history of use. Standard

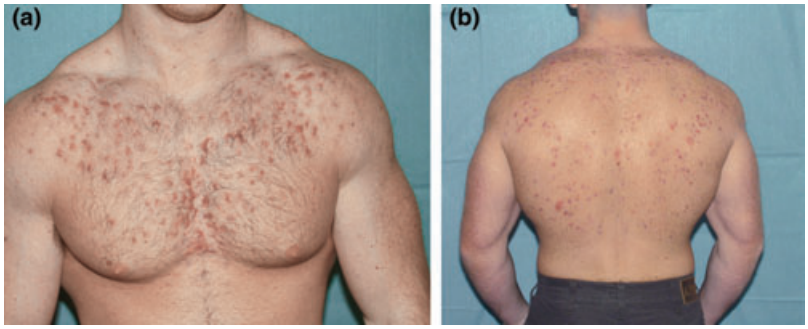

Figure 1 Anabolic steroid-induced acne on the (a) chest and (b) back in a body-builder.

treatment determined by the severity of the acne should then be instituted.[5]

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References

1 Evans NA. Gym and tonic: a profile of 100 male steroid users. *Br J Sports Med* 1997; **31**: 54–8.
2 Perry HM, Wright D, Littlepage BN. Dying to be big: a review of anabolic steroid use. *Br J Sports Med* 1992; **26**: 259–61.
3 O'Sullivan AJ, Kennedy MC, Casey JH *et al.* Anabolic–androgenic steroids. medical assessment of present, past and potential users. *Med J Aust* 2000; **173**: 323–7.
4 Kiraly CL, Alen M, Korvola J, Horsmanheimo M. The effect of testosterone and anabolic steroids on the skin surface lipids and the population of *Propionibacteria acnes* in young postpubertal men. *Acta Derm Venereol* 1988; **68**: 21–6.
5 Simpson NB, Cunliffe WJ. Disorders of the Sebaceous Glands. In: *Rook's Textbook of Dermatology* (Burns T, Breathnach S, Cox N, Griffiths, C, eds), 7th edn, Vol. 3. Oxford: Blackwell Scientific Publications, 2004; 43.1–43.75.

Lentiginous hyperpigmentation confined to resolved psoriatic plaques and treated with a Q-switched ruby laser

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Hyperpigmented lesions confined to resolved psoriatic plaques have been rarely reported and have been described previously as lentigines, naevus spilus hyperpigmentation and speckled pigmentation.[1–3] We present a similar case causing cosmetic concern, which was treated using a Q-switched ruby laser (QSRL).

A 55-year-old white man (skin type II) with a 28-year history of chronic plaque psoriasis noted freckling on his skin 6 months after broad-band UVB phototherapy in combination with dithranol. His psoriasis had cleared after 34 treatments (cumulative dose 6.73 J/cm^2) with no adverse reactions. Four years later the freckling was still persisting without fading, and this, along with reoccurrence of psoriasis, caused him to re-present. He had used a sunbed daily for a month after phototherapy but denied other excess sun exposure. His medication included methotrexate, started shortly after phototherapy for psoriatic arthropathy. Topical treatments comprised calcipotriol, emollients and steroids. On examination, he had light- and dark-brown macules around the periphery of the cleared psoriasis plaques (Fig. 1a). Scattered plaques of psoriasis were also evident. There was no clinical evidence of photodamage on sun-exposed sites. A skin biopsy of a pigmented lesion showed small areas of epidermal basal-layer hyperpigmentation.

In view of the patient's wish to treat this pigmentation, three test areas were treated with two lasers. A Q-switched frequency double (KTP) Nd:YAG laser (QSFDNYL) was tested in two areas (532-nm, 4-mm spot diameter) using either 1 J/cm^2 or 1.5 J/cm^2. A QSRL was tested in another area (694-nm, 5-mm spot diameter, 3.8 J/cm^2). At the 10-week review, the patient reported pain and blistering at the QSFDNYL sites, and no

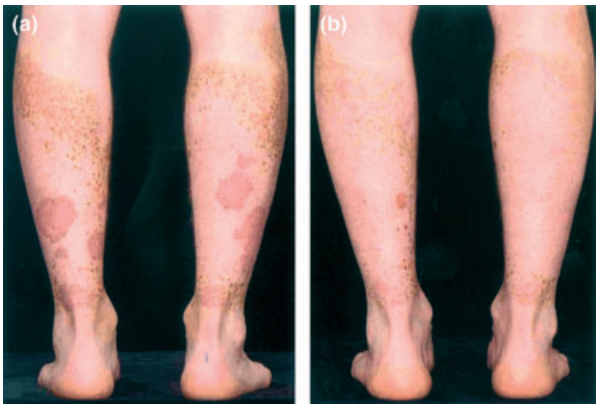

Figure 1 (a) Before laser treatment; (b) after four treatments with the Q-switched ruby laser.