The interesting point about this case was that the activity of DH was closely associated with the inflammatory state of chronic tonsillitis. Several lines of evidence support this conclusion: First, the eruption was clinically reproducible by surgical manipulation of the tonsils. Secondly, lymphocytes harbouring in the patient’s tonsil expressed predominantly IgA. This finding is noticeable because the majority of lymphocytes in normal tonsils express IgG in much the same way as chronic tonsillitis without complications (9, 10). Under certain conditions, such as IgA nephropathy, which is possibly induced by tonsillitis, IgA expressing lymphocytes are predominant (10, 11). Thirdly, granular IgA deposits in the dermal papillae disappeared after tonsillectomy.

The current explanation for DH is that lymphocytes recruited in conjunction with GSE produce IgA autoantibodies that also react with dermo-epidermal junction (12). In the present case, the inflammatory reaction in the tonsils might be responsible for producing IgA class autoantibodies that react with the dermo-epidermal junction.

REFERENCES

Multiple Becker’s Naevi: A Rare Presentation

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Sir,

Becker’s naevus (pigmented hairy epidermal naevus), a variety of epidermal naevus (1) is present in about 0.5% of young men (2). It is about 5 times more frequent in males than in females. Characteristically, it is a unilateral single lesion of the shoulder, upper arm, anterior chest or scapular region in males, appearing during adolescence. It may affect other sites, e.g. the lower limb (3, 4). It may rarely also be multiple and bilateral (5). We report here a rare case of multiple Becker’s naevi with 7 distinct lesions present on different sites including the lower limbs.

CASE REPORT

A 28-year-old male consulted us for multiple, asymptomatic light to dark-brown macules that had been present for 15 years over both lower extremities, abdomen and front of chest, extending on to the left upper limb. At first, the patient noticed multiple asymptomatic light-brown macules over the lower left limb, which coalesced to form a larger patch and the pigmentation also grew darker. He noticed increased hair growth over the lesion and gradually over the years he developed 6 similar lesions on the chest, abdomen and right lower limb. There was no history to suggest any neurological or any other systemic illness.

On cutaneous examination, 7 light-brown to dark-brown macules of sizes varying from 10 × 15 cm to much larger areas covering almost half of the lower limb with a “splash on” appearance at the periphery and central hypertrichosis in most of them. The lesions were situated on the chest, abdomen, back and right groin, extending on to the medial aspect of the right thigh, both knees and anterior aspect of both legs, left arm and forearm (Fig. 1). The lesions on the chest and anterior abdominal wall showed a sharp midline margin. Thorough physical examination did not reveal any neurological or musculoskeletal defect. Routine laboratory investigations including a haemogram, liver and renal function tests, urinalysis and examination of stool were all within normal limits. X-ray of the chest, spine and lower extremities did not reveal any pathological findings. Histopathological examination of the lesion from the chest showed mild acanthosis with increased pigment (melanin) in the basal cell layer, especially at the
bases of the rete ridges (Fig. 2). Smooth muscle bundles were seen, though these were considered within normal limits.

DISCUSSION

Becker’s naevus has classically been described as acquired, localized, unilateral hypermelanosis around the shoulders of adolescent males, but since the original description of the entity, the definition has undergone numerous changes. An extensive search of the literature has revealed fewer than 10 patients described with multiple lesions. Our patient had 7 distinct lesions, which we believe is an unusually high number and perhaps the highest reported so far. The sharp midline cut-off in the truncal lesions, as in other cases, has been suggested to be due to the development of the lesion at the time of organogenesis but manifesting later in life.

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