Secondary Anetoderma Overlying Schwannoma

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Sir,

Anetoderma is a clinical entity characterized by localized atrophy of the skin owing to destruction of dermal elastic fibres. Anetoderma may be classified as primary or secondary. Primary anetoderma develops on clinically normal skin without any other preceding dermatoses. Secondary anetoderma occurs in association with another disease. Here, we describe a patient with anetoderma occurring in association with schwannoma.

CASE REPORT

A 46-year-old woman presented with a tumour on her left upper arm of 3 years’ duration. There was no history of trauma at the site. Physical examination revealed a 1.5 x 1.5 cm, wrinkled, pink, elliptical, redundant lesion with a firm underlying subcutaneous mass (Fig. 1). The patient complained of pain and tenderness. A biopsy specimen showed well-demarcated nodules composed of spindle cells with alternating Antoni A and B areas, consistent with schwannoma. The collagen bundles were pale and attenuated in the oedematous overlying dermis, but appeared normal in the dermis opposite the lower tumour margin. Verhoeff-van Gieson staining showed a marked reduction in the quantity of elastic fibres in the upper dermis above the schwannoma, but not in the dermis below the tumour (Fig. 2A and B). The patient was therefore diagnosed with anetoderma secondary to schwannoma. The tumour was completely excised and the patient healed well without recurrence.

DISCUSSION

Secondary anetoderma is a rare disorder, most commonly observed in women aged 20–40 years. Histopathological examination of the dermis reveals atrophic and oedematous changes associated with diminished fragmented collagen and absent elastic tissue. Secondary anetoderma is usually associated with inflammatory cutaneous diseases, including lupus erythematosus, acne, and granuloma annulare; cutaneous infections, such as herpes zoster and syphilis; and use of medica-
tions such as penicillamine (1). Secondary anetoderma can also be a complication of cutaneous neoplasms, such as pilomatricoma (2), xanthogranuloma (3), lymphoma (4) and plasmacytoma (5). The underlying tumour may be visible through soft and atrophic anetodermic skin. The association with schwannoma is very rare, with only one previous case report to date (6).

There are various hypotheses regarding the pathogenesis of anetoderma. Elastolysis has been regarded as enzymatically mediated, probably by elastases released from macrophages in inflammatory diseases (7). Tumour cells may release catabolic enzymes when subjected to mechanical stress or irritation, and these enzymes may damage elastic fibres (2). Continuous mechanical stimulation of a cutaneous tumour would thus predispose to secondary anetoderma, as observed in our patient, in whom we observed a difference in elastolysis between the upper and lower dermis. Tumour cells in the upper marginal zone are prone to experience more mechanical stress and release more catabolic enzymes than those in the lower dermis. It could be postulated that secondary anetoderma is more likely to occur in association with cutaneous tumours located on sites exposed to continuous pressure and/or mechanical irritation, tumours with hard indurations, such as pilomatricomas, and tumours located in the superficial dermis.

REFERENCES