Secondary Anetoderma due to Pilomatricoma

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We report a case of secondary anetoderma due to pilomatricoma. A 15-year-old girl presented with a soft, 1.5×1.5cm in size, atrophic, pinkish and bulged-out lesion overlying the palpable subcutaneous mass on the left upper arm for several months. Histologic examination revealed the reduced number of dermal elastic fibers overlying a typical pilomatricoma.
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Anetoderma, which means looseness of the skin, is due to the loss of dermal elastic fibers and has been classified into primary and secondary forms[1]. The term secondary anetoderma implies that the lesion has occurred in skin in which another skin lesion was presented, in contrary to primary anetoderma which occurs in clinically normal skin[2].

Although inflammatory dermatoses have been reported to be the common causes of secondary anetoderma[3,4], anetodermic lesions have been reported in the sites of skin tumors, that is, pilomatricoma, nodular amyloidosis, lymphocytoma cutis and xanthomas in Western literature[2,5-11]. To the best of our knowledge, however, there is no reported case of secondary anetoderma due to pilomatricoma in Korea.

We herein present a case of anetoderma due to pilomatricoma that we have recently experienced.

REPORT OF A CASE

A 15-year-old girl was seen with a solitary lesion on the left upper arm, which appeared several months prior to her visit and had grown somewhat rapidly during that time. There was no particular record either on trauma or on family history. On skin examination, a soft, 1.5×1.5cm in size, atrophic, pinkish and bulged-out lesion on the extensor aspect of the left upper arm was noted (Fig. 1), and a firm subcutaneous mass palpated beneath the bulged-out lesion (Fig. 2).

Skin sections by punch biopsy showed histopathologic findings suggestive of a diagnosis of the pilomatricoma in the subcutis, but also showed increased vascularity, vasodilation with some thrombi and individually seperated collagen fibers in the dermis. The entire lesion was excised for treatment and a precise histopathologic diagnosis. Histopathologic examination of the excised lesion revealed mild acanthosis with rete ridge elongation in the epidermis and a well-circumscribed tumor mass in the subcutis (Fig. 3). The tumor mass was chiefly composed of shadow cell nests, and had mononuclear and giant cells in the periphery and stroma of the tumor (Fig. 4). On von-Kossa stain, focal areas of calcification were seen within the tumor. These findings were considered to be well consistent with those of pilomatricoma, but interestingly several small portions of shadow cell nests were scattered in the dermis and seperated from the main tumor mass in the subcutis (Fig. 3). The other dermal findings were consistent with the previous punch-biopsied findings. On Victoria blue stain, the dermal elastic fibers overlying the tumor mass were

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reduced in number as compared with the unaffected skin around the lesion from the patient (Fig. 5).

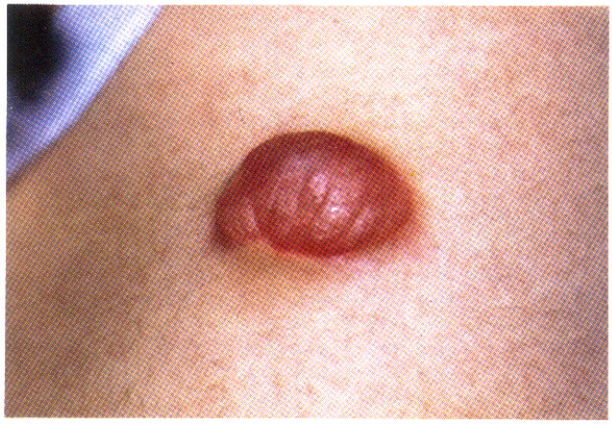

Fig. 1. An atrophic, pinkish, 1.5×1.5cm in size, bulged-out lesion on the extensor aspect of the left upper arm.

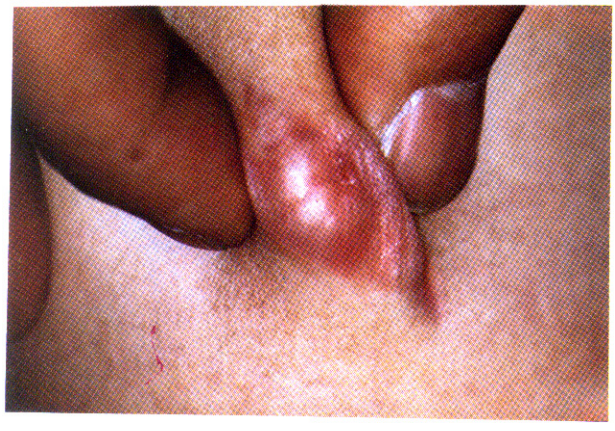

Fig. 2. A palpable subcutaneous tumor mass beneath the bulged-out lesion.

DISCUSSION

Although most reported cases of secondary anetoderma occurred due to inflammatory dermatoses, a few cases caused by skin tumor, pilomatricoma, have been reported in the literature since first described by Cordioviola[5] in 1943. In 1978, Moulin et al.[9] presented 5 cases that showed anetodermic change in the skin overlying pilomatricoma and subsequently reviewed the histopathologic features of the skin overlying additional 34 pilomatricomas. Evidence of dermal

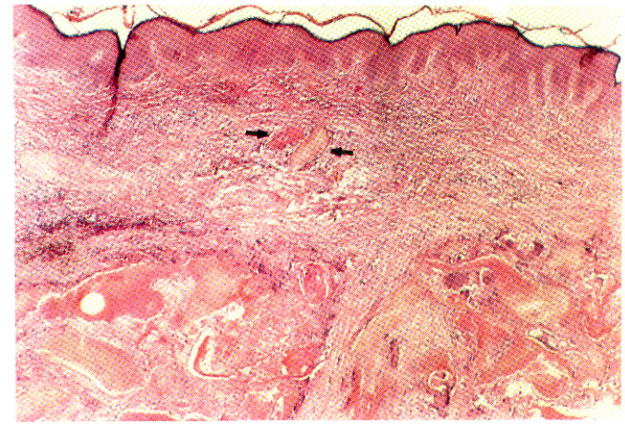

Fig. 3. Acanthosis with rete ridge elongation in the epidermis and a well-circumscribed tumor in the subcutis. Arrows indicate scattered small portions of shadow cell nests seperating from the main tumor mass in the dermis (H & E stain, ×40).

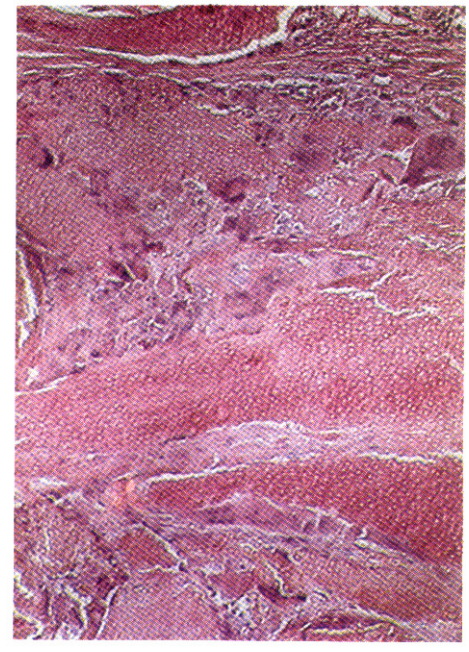

Fig. 4. Multiple shadow cell nests and mononuclear and giant cells in the periphery and stroma of the tumor (H & E stain, ×200).

atrophy was found in 20 of these patients. Jones and Tschen[11] recently reported 4 cases under the title of "anetodermic cutaneous changes overlying pilomatricomas" and reviewed the literature.

Anetoderma is generally known to appear as circumscribed lesions of soft, thinned, wrinkled skin that can be slightly depressed or bulged loosely[1,3]. Secondary anetoderma caused by

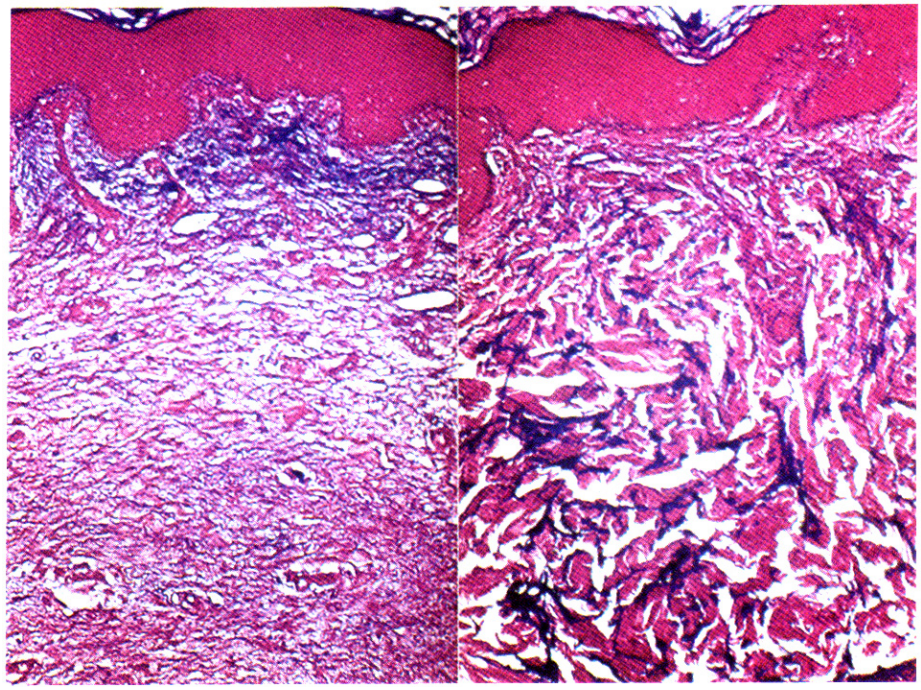

Fig. 5. Loss of elastic fibers in the dermis. left: lesion, right: control (Victoria blue stain, ×100).

pilomatricoma appeared as an atrophic, folded, pink, scarlike, striaelike or pseudoblistering lesion with an underlying firm tumor[5-11]. In the present case, it showed a soft, atrophic, pinkish and bulged-out lesion, whose clinical appearance was nearly identical to those reported previously.

The primary histopathologic finding in all forms of anetoderma is the destruction and loss of dermal elastic fibers. Examinations of anetodermic lesions overlying a pilomatricoma also revealed a considerably reduced number or complete absence of elastic fibers; in addition, the epidermis was normal, the overall thickness of the dermis was reduced, and there was increased vascularity and vasodilation in the dermis[11]. The histopathologic findings of our case were similar to these findings except in two respects; the presence of acanthosis with rete ridge elongation and the scattered shadow cell nests seperated from the main tumor mass in the dermis. Mehregan[12] supposed that in the biologic phenomenon of transepidermal elimination, the pathologic tissue acted as a mechanical irritant and caused hyperplasia of the epidermis which enclosed the pathologic material, and was eventually eliminated with keratinocytes. Some authors[13,14] reported several cases of perforating pilomatricoma showing transepidermal elimination with epidermal hyperplasia. Jones and Tschen[11] also suggested that cutaneous anetoderma might be the preceding step to the subsequent transepidermal elimination of pilomatricoma. Thus, the presence of epidermal hyperplasia in our case might support the transepidermal elimination of the tumor mass, although it is not certain that the presence of scattered dermal shadow cell nests is due to either the process of transepidermal elimination or the artifact produced by punch biopsy.

The mechanism of elastolysis in secondary anetoderma is unknown so far[1], but some authors proposed that tumor cells, inflammatory cells or both might produce elastase or unidentified catabolic enzymes that lead to the fragmentation of collagen and the destruction of elastic fibers[11,15,16]. A possible relevancy regarding this pathomechanism of secondary anetoderma in our patient might be explained by the fact the anetodermic zone of the overlying pilomatricoma was extended far beyond the periphery of the tumor[11].

It is not clear in what type of pilomatricoma the phenomenon of anetoderma and transepidermal elimination occur, but we suppose that more cases of pilomatricoma showing anetodermic cutaneous changes overlying the tumor or the phenomenon of transepidermal elimination could be found if careful clinical and histopathologic observations were performed.

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