recurrent clinical course and typical histological features, our case was finally diagnosed as pustular psoriasis, although the clinical feature was atypical.

Classification of pustular psoriasis is sometimes difficult (3). Our case could be distinguished from palmoplantar pustulosis and acrodermatitis continua. Her lesions resembled APP, which is a rare condition of recurrent erythema annulare-like lesions with psoriasiform histopathological features (1). Other names for APP are erythema circine recidivants (4), Lapière-type psoriasis (5), and erythema annulare centrifugum-type psoriasis (6). APP typically runs a cyclically recurrent course that may span decades (1). It may occur alone, in the complete absence of any stage of recognizable psoriasis and a family history of psoriasis vulgaris is usually absent. Lesions start as discrete areas of erythema that become raised and oedematous. Pustules begin peripherally on the crest of an advancing edge, become desiccated and leave a trailing fringe of scale as the lesion advances. Considering the clinical manifestation and course of the condition, our case is closer to APP than other types of pustular psoriasis. Although usually generalized, APP rarely occurs as a localized form. Zala & Hunziker (2) reported 2 cases of localized psoriasis of the erythema annulare centrifugum-type with pustulation; one on the scalp and forehead and the other on both thighs. However, to date, there has been no report of APP localized to the dorsa of the feet.

In the localized form of pustular psoriasis, the possible triggering factor of an irritating topical treatment may be of Koebnerization (7). Although in our case no triggering factors were identified, there might be a relationship to minor trauma or local irritants. The contribution of concomitant dermatophyte infection is unlikely, since organisms were not demonstrated on repeated KOH examination, fungal cultures, or PAS staining. Also the presence of coagulase-negative staphylococci grown in bacterial culture was not considered aetiological and it might be secondary to pustular formation and of little significance in our patient.

Localized pustular psoriasis is usually refractory to treatment. Treatments include the use of topical steroids, tar preparation and systemic therapy with etretinate and PUVA (8). However, aggressive topical and phototherapeutic treatment modalities can lead to worsening of the condition. There have been some anecdotal reports of positive experiences with dapsone (9). As in our patient, the benefits of such treatment are usually temporary and the lesions tend to recur with diminishing doses.

REFERENCES

1. Baker H, Ryan TJ. Generalized pustular psoriasis: A clinical and epidemiological study of 104 cases. Br J Dermatol 1968; 80: 771 – 793.
2. Zala L, Hunziker T. Lokalisierte forms der Psoriasis vom typ des erythema annulare centrifugum mit pustulation. Hautarzt 1984; 35: 53 – 55.
3. Zelickson BD, Muller SA. Generalized pustular psoriasis: A review of 63 cases. Arch Dermatol 1991; 127: 1339 – 1345.
4. Basex A, Dupré A, Christol B, Jalby J. Psoriasis à type d'érythema circiné récidivant de Bloch. Bull Soc Fr Dermatol Syphiligr 1967; 74: 689 – 695.
5. Clavert HT. Pustular psoriasis (Type Lapière) – (Annular centrifugal psoriasis). Br J Dermatol 1967; 79: 719 – 721.
6. Rajka G, Thune PO. On erythema annulare centrifugum-type psoriasis. Acta Derm Venereol (Stockh) 1979; 59 Suppl 85: 143 – 145.
7. Quan MB, Ruben BS. Pustular psoriasis limited to the penis. Int J Dermatol 1996; 35: 202 – 204.
8. Farber EM, Nall L. Pustular psoriasis. Cutis 1993; 51: 29 – 32.
9. Piamphongsant T, Nimsuwan P, Gritiyarangsan P. Treatment of generalized pustular psoriasis – clinical trials using different therapeutic modalities. Clin Exp Dermatol 1985; 10: 552 – 561.

Accepted September 7, 1998.

Young Min Park, Hoon Kang and Baik Kee Cho
Department of Dermatology, Our Lady of Mercy Hospital, The Catholic University of Korea, 665 Bupyung-dong, Bupyung-gu, Incheon 403-016, Korea. Email: mrhoo@nuri.net.

Cellular Neurothekeoma

Sir,
Cellular neurothekeoma (CNT) is a rare benign tumour of peripheral nerve sheath origin, first described in 1986 by Rosati et al. (1). The lesion usually occurs in young patients (20 – 30 years old) as a solitary nodule on the head. Its clinical aspect is aspecific resembling dermatofibroma, leiomyoma, annexal tumour or sebaceous cyst (2). The lesion arises as an asymptomatic red firm papule enlarging rapidly in the first 2 – 3 months and then more slowly until it reaches a diameter of about 3 cm. The treatment of choice is surgery; recurrences occur after incomplete excision. We report a case of a CNT which arose in a 55-year-old woman and we underline the rare occurrence of the tumour over the age of 30.

CASE REPORT

A 55-year-old woman was examined for an enlarging pink-red nodule on her left cheek (Fig. 1). The mass was scarcely movable on the underlying tissues and firm in consistency; its diameter was about 1 cm. The lesion was completely removed by surgery. Histological examination showed a symmetrical non-encapsulated proliferation of epithelioid or plump spindled cells in the dermis with a predominantly nested pattern. The nests dissected collagen bundles and epidermal appendages in the dermis and extended into the subcutis. The cells showed neither cytological atypia nor mitotic figures (Fig. 2). The epidermis was intact but a true grenz zone could not be detected because the nests were closely related to the dermo-epidermic junction. Periodic acid – Schiff staining failed to demonstrate the presence of mucin within the lobules. The following immunohistochemical stainings were performed

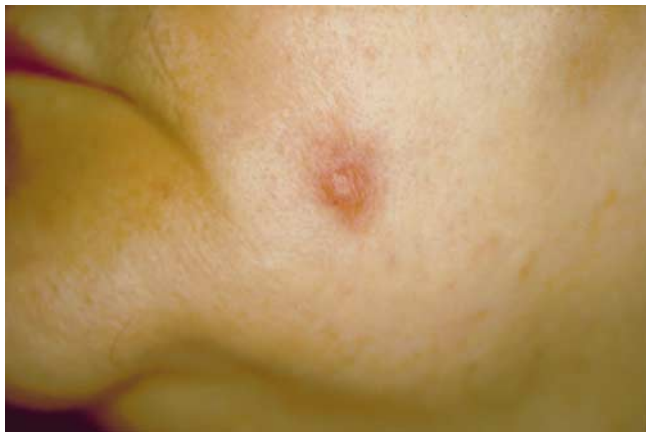

Fig. 1. Clinical appearance of CNT on the cheek.

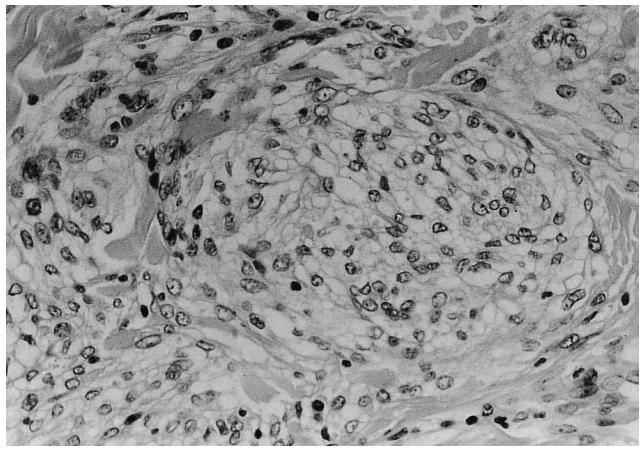

Fig. 2. Epithelioid and plump spindled cells without cytological atypia and mitotic figures (× 200).

and showed negative results: S-100, HMB-45, pan cytokeratin, EMA, α-smooth-muscle actin, desmin, CD-68, CD-34, chromogranins, GFAP and synaptophysin. NSE was positive. A diagnosis of CNT was made based on histological findings

DISCUSSION

CNT is a neoplasm with a controversial histogenesis. It is considered to be a variant of classical nerve sheath myxoma. In fact, based on its histological background three subtypes of nerve sheath myxoma have been described: a pure myxoid, a pure cellular and an overlap one, based on cellularity, mucin content and growth pattern. Myxoid subtype (mature nerve sheath myxoma) is most common in middle-age and is typically located on the face and upper extremities, but can occur anywhere on the body. Its main characteristic histological pattern is the presence of myxoid micronodules loosely clustered in a fibrous matrix in the reticular dermis. The immunohistochemical profile is: S-100 (+), collagen type IV (+), vimentin (+).

CNT (immature nerve sheath myxoma) differs from the myxoid type due to its earlier onset (the mean age is 24 years but examples have been reported in children), its characteristic histological pattern with fascicles of cells infiltrating collagen bundles and its variable immunohistochemistry. Barnhill et al. (2) hypothesized that the myxoid variant might represent an older stage of CNT but they noticed that the anatomic sites of predilections of the two variants are different and thus the idea of a distinct subtype rather than myxoid alteration of CNT was supported. Ultrastructural studies (3) support the view that CNT is primarily composed of undifferentiated cells with partial features of Schwann cells, smooth muscle-cells, myofibroblasts and fibroblasts, but that this tumour and classical nerve sheath myxoma are, however, related and represent a spectrum.

We believe that the main interest of this case is the age of the patient (55 years) which is unusual for the development of a CNT. In the series of Calonje et al. (4) a 65-year-old patient is reported. Differential diagnosis with minimal deviation neurotropic melanoma and, mainly, metastatic malignant melanoma must be considered at this age. A positive reaction for S-100 protein and HMB-45 would strongly favour a diagnosis of malignant melanoma.

REFERENCES

1. Rosati LA, Fratamico CMF, Eusebi V. Cellular neurothekeoma. Appl Pathol 1986; 4: 186 – 191.
2. Barnhill RL, Dickersin R, Nickeleit V, Bhan AK, Muhlbauer JE, Phillips ME, Mihm MC. Studies on the cellular origin of neurothe-keoma: clinical, light microscopic, immunohistochemical, and ultrastructural observations. J Am Acad Dermatol 1991; 25: 80 – 88.
3. Argenyi ZB, Kutzner H, Seaba MM. Ultrastructural spectrum of cutaneous nerve sheath myxoma/cellular neurothekeoma. J Cutan Pathol 1995; 22: 137 – 145.
4. Calonje E, Wilson-Jones E, Smith NP, Fletcher CDM. Cellular "neurothekeoma": an epithelioid variant of pilar leiomyoma? Morphological and immunohistochemical analysis of a series. Histopathology 1992; 20: 397 – 404.

Accepted September 2, 1998.

R. Strumìa[1], A. R. Lombardi[2] and L. Cavazzini[2]
Departments of [1]Clinical Dermatology and [2]Medical Sperimental and Diagnostics, University of Ferrara, Via Savonarola 9, IT-44100 Ferrara, Italy.