

Late Onset Ota Nevus

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Ota nevus is a dermal melanocytic hamartoma derived from neural crest melanoblast and involves skin innervated by the trigeminal nerve. Although most cases are clinically apparent at birth or around puberty, acquired lesions in adults have been rarely reported. We reported a 73 year-old Korean man with Ota nevus of the face and scalp that onset in his eighth decade and accompanied multiple solar comedo of the face. (*Ann Dermatol* 11(4) 289~291, 1999).

Key Words : Ota nevus, Late onset, Dermal melanocyte.

Ota nevus is a melanocytic pigmentary disorder involving skin innervated by the trigeminal nerve¹. Usually the onset is before the third decade of life. Occasionally, late onset Ota nevus has been reported in elderly persons. In our review of Korean literature, onset in the fourth decade has been described in two patients^{2,3}. We report herein a case of late onset Ota nevus that occurred in the eighth decade.

CASE REPORT

A 73-year-old Korean man presented with multiple, ill-defined, blue-gray patches on his left face and scalp (Fig. 1 A, B). He recalled that the lesions had slowly enlarged and grown darker over about 3 years. He did not complain of any symptoms except the aesthetic problem. Physical examination showed blue-gray patches on the left upper and lower eyelids, temple, forehead and scalp without surface change. The sclerae and mucous

membranes of the eye, nose, and mouth were not affected. Besides the pigmented lesion, multiple solar comedo of Favre-Racouchot disease and seborrheic keratosis were noted on his face. He had worked as a farmer in rice fields since his teens. He had no other medical problems and no family history of pigmentary skin diseases.

A skin punch biopsy was done from the pigmented lesions of the scalp and forehead. Histological examination showed many dendritic melanocytes containing melanin granules and pigment-laden macrophages in the upper reticular dermis (Fig. 2 A, B). These cells were dispersed between connective tissue fibers but did not disrupt them. This dermal melanocytosis was consistent with findings of Ota nevus. The patient has been followed up without treatment.

DISCUSSION

Ota nevus is a dermal melanocytic hamartoma derived from neural crest melanoblasts located in the upper portion of the dermis⁴. It usually represents unilateral, speckled, blue-black or slate-gray macular discolorations on the areas innervated by the first and second branch of the trigeminal nerve¹. It may involve ipsilateral mucous membranes of the eye, nose, and mouth, as well as sclera and tympanum. Ota nevus occurs most commonly in Asian populations and women are affected five times

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Fig. 1. Multiple blue-black patches are shown on the left side of the face (A) and scalp (B). Several flat-topped yellowish papules and nodules of Favre-Racouchot disease are seen on both periorbital areas in Fig. 1. A.

Fig. 2. Dermis contains dendritic cells (A: H&E stain, $\times 100$) with fine brown cytoplasmic melanin granules (B: H&E stain, $\times 400$).

more often than men¹. The onset shows bimodal peak incidence at birth and puberty⁵. In half of the cases Ota nevi are present at birth or become apparent in the first few months of life and in remaining cases, they appear in the first to third decades. Onset after thirty years of age has been

reported in only a few cases^{2,3,6,7}. Occurrence at the age of 30 was described in a woman after an intralesional steroid injection for ocular toxoplasmosis. Appearance of her Ota nevus at the site of injection suggested that infection and minor trauma might be triggering stimuli of acquired Ota nevus⁶. Recently,

Ota nevus was found in an 81-year-old man, which involved his left face and scalp⁷.

Late onset Ota nevus must be differentiated from several pigmentary disorders and melanoma although malignant change of cutaneous Ota nevus is extremely rare⁵. In our case, other pigmentary disorders and melanoma were excluded easily. Acquired dermal melanocytosis on the face, also referred as acquired bilateral nevus of Ota-like macule, is symmetrically distributed discrete pigmentation of the face with a late onset⁸. Unilateral localization and diffuse ill-defined patches rather than discrete macules made our case be diagnosed as Ota nevus.

In congenital Ota nevus, hamatomatous melanocytes are already present in the dermis at birth. In acquired lesions it may be hypothesized that there are pre-existing dermal melanocyte as in congenital form⁹. Mizushima et al. discovered the presence of dermal melanocytes in the uninvolved skin near the pigmented macules¹⁰. This result supports that some stimuli induce reactivation of melanin synthesis in dermal melanocytes to produce an apparent lesion. It is likely that sex hormones are involved in this stimulation. The evidences for the association of sex hormones are the female predominance, frequent onset at puberty and variation in the degree of pigmentation with menstruation. Post-traumatic or post-inflammatory onset, and darkening or increase in number of nevi with age suggest that trauma, inflammation, epidermal changes and aging may be other potent triggering factors⁹. Considering that our patient's occupation was a farmer and solar comedo co-existed on the face, cumulative long-term solar damage or aging might have played a role as a potential stimulating factor in the late development of Ota nevus.

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