A Case of Acute Reticulate Hyperpigmentation on the Face and Neck

Hoon Kang, M.D., Jun Young Lee, M.D., Chung Won Kim, M.D.

Department of Dermatology, Catholic University Medical College, Seoul, Korea

Multiple, reticulated, dark brown colored, macules and patches suddenly developed on the face and neck of a 48-year-old Korean woman two days after a traffic accident. On physical examination and laboratory tests including serum melanocyte-stimulating hormone, estrogen and progesterone level, no abnormalities were found except cervical pain. Histological examination of hematoxylin-eosin stained sections revealed increased melanin pigmentation in the basal layer, but the number of melanocytes was not changed in DOPA stained section. Topical application of 2% hydroquinone and 20% azelaic acid ointments had been applied successively for two months each without any apparent improvement.

Herein we present a case of reticulate hyperpigmentation on the face and neck, which is very acute and whose causative factors are not certain. (Ann Dermatol 7:(3)244-247, 1995)

Key Words : Reticulate hyperpigmentation

Disease which shows pigmentation on the face and neck originates from various conditions such as melasma, pigmented contact dermatitis, pregnancy, and medications. Most pigmented diseases usually progress slowly and have cause-related characteristic patterns and sites1, but our case developed spontaneously after a traffic accident, showed atypical reticulate pattern of hyperpigmentation and was confined to the face and anterior neck area. Herein we report a case of atypical reticulate hyperpigmentation with no certain cause.

REPORT OF A CASE

A 48-year-old female was referred to our department because of sudden hyperpigmentation on the face and neck. Four days previously she had been admitted to the neurosurgical department because of cervical pain after a traffic accident.

Two days later, asymptomatic, multiple, reticulated, dark brown colored macules and patches developed spontaneously on the face and neck (Fig. 1).

Her past history showed she had experienced localized hyperpigmentation on both maxillary areas 3 years earlier, but it had soon disappeared. There was no history of previous medication, ultraviolet (UV) radiation, pregnancy, endocrinologic disease or inflammation. Family history was not contributory.

Laboratory examinations including complete blood count, urinalysis, blood chemistry were all within normal limits or negative. In addition, serum cortisone, alpha-melanocyte stimulating hormone (α-MSH), progesterone and estrogen level were not contributory. Chest roentgenogram, brain CT and cervical MRI showed no abnormal findings.

Histologic examination of hematoxylin-eosin (HE) stained section revealed increased melanin pigmentation in the basal layer (Fig. 2), but the number of melanocytes was not changed in the DOPA stained section (Fig. 3).

We tried application of 2% hydroquinone topically for 2 months but there was no clinical improvement. Although we substituted 20% azelaic acid

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Reprint request to: Hoon Kang, M.D., Department of Dermatology, Kangnam St. Mary's Hospital Catholic University Medical College Kangnam-gu, Banpo-dong, 505 Seoul, 137-040, Korea
for 2% hydroquinone, we could not find any apparent improvement. Two years after discharge, she still shows light brown colored patches on the face and neck.

**DISCUSSION**

Diseases which cause abnormal excessive pigmentation can be classified into melanotic, melanocytic, and nonmelanotic types. Melanotic is the case which shows an increase in the amount of melanin without an increase in the number of melanocytes. Melanocytic means an increase in the number of melanocytes, and nonmelanotic means pigmentation caused by physical factors. Generally, pigmentation of the skin indicates melanotic rather than melanocytic. Melanotic pigmentation occurs when the production and size of normal melanosomes transferred to the epithelium and the lifetime of the epithelium increase. In our case, the increase in amount of melanin along the basal cell layer of the epidermis was found on HE stained section, but the number of melanocytes was not increased on DOPA stained section. So we assumed the pigmentation of our case to be classified as melanotic.

Clinically, melasma should be considered in differential diagnosis. Melasma mostly shows the pigmentation symmetrically on the brow, chin, upper lip, and temporal area of the face and it is known that it is related to exposure to UV radiation, ingestion of contraceptives, use of cosmetics, pregnancy, and hormones such as MSH, estrogen, and progesterone. Clinically and histologically our case shows a similarity to melasma. But it is distinguishable from the usual form of melasma in some respects such as the hyperpigmentation of our case.
was distributed on the whole face and anterior neck including the area under the chin which is an unexposed area, and the lesion developed suddenly and spread rapidly.

The causes of disease which shows melanotic pigmentation on the facial area could be UV light, contactants, pregnancy, medications, and endocrine disease. But it is hard to determine the cause when the shape and the distribution of the pigmentation is not typical. Pigmentation caused by UV radiation can be divided into immediate tanning and delayed tanning. Immediate tanning shows pigment change in a few minutes by oxidizing the existing melanin or its antecedent, while delayed tanning shows pigmentation in 48-72 hours by producing new pigment. UV radiation is an unlikely cause in this case. And even if the patient had been exposed to UV radiation during a traffic accident, it is hard to believe that such a little amount of UV radiation would cause sudden pigmentation. The possibility of pigmentation caused by UV lights can also be excluded because pigmentation was found on the area under the chin but not found on an exposed area.

MSH accelerates the production of melanin by increasing cyclic AMP from normal melanocytes, and high estrogen level induced by pregnancy or taking contraceptives can cause hyperpigmentation on a small area of the body. However, there were no changes in the level of MSH, cortisone, estrogen, or progesterone and we could not find any other endocrinologic problems in our case. Some medications and chemical compounds, including chemotherapeutic agents (bleomycin, 5-FU, met-hotrexate, BCNU etc.), metals (arsenic, gold, lead, chrome etc.) tetracycline, azo and anilin dye, also cause hyperpigmentation. However, we could not find any drug history.

It is known that the inflammatory mediators and cytokines directly influence the production of melanin. When the inflammation is caused by UV lights, contact dermatitis, atopic dermatitis, or psoriasis, many inflammatory mediators and cytokines including prostaglandin, leukotriene, basic fibroblast growth factor, and interleukin-1 are released from the keratinocyte and accelerate the production of melanin. However, pigmentation caused by cytokine or inflammatory mediators usually occurs after 30 days. Therefore, this is not the cause in this case.

Goldman et al., reported that melanophores of fish, amphibian, and reptile have adrenaline receptors, and cause the pigmentation on a specific stimulus. Also, Latties et al., noted that the tyrosinase which is in the eyeballs of rabbits is affected by the adrenergic nerve system. Therefore, he thought that catecholamine had an effect on the pigmentation. Although we could not find the exact mechanism of the pigmentation in our case, we suppose that it was caused by neuropeptides released from the nerve-endings in the face and cervical region after an intensive physical and psychological shock caused by a traffic accident. However, we consider that the exact mechanism should be further investigated.

REFERENCES


