Recurrence of Depigmentation in a Halo Nevus after Autologous Suction Blister Grafting

Jin-Chun Suh, M.D., Seon-Kyo Seo, M.D., Gun-Yoen Na, M.D.,PhD.

Department of Dermatology Fatima Hospital 302-1, Sin-Am Dong, Dong Gu, Taegu, South Korea

In this report we will highlight an interesting 3 year case of a halo nevus on the back of a 13-year-old Korean girl. This was a single halo nevus with a central pinkish mole and a depigmented patch, 20 mm in diameter. The patient underwent an autologous suction blister graft on the lesion. The halo nevus was completely repigmented except for the marginal rim. At a 4 month follow-up, a new whitish halo was observed around the central pinkish mole. At this point the central nevus was excised and examined with a H & E stain and an immunohistochemical stain with an anti-Ig G antibody. After the excision of the central mole, repigmentation was completed and this condition persisted at a 3-year-follow-up.


The halo nevus is characterized by a pigmented nevus with a surrounding depigmented zone. Its course of development is variable, 25% of all cases are associated with vitiligo, whereas a few cases may be associated with melanoma or a dysplastic nevus. However, in most cases the central nevus will disappear in time, the leukodermic areas will remain depigmented for an unpredictable period yet repigmentation may take place eventually. Usually there is no recommended treatment for a halo nevus except for the periodic examination of the affected area. In contrast the results of this case would seem to suggest that a central mole should be removed.

CASE REPORT

This case outlines the 3 year history of a single halo nevus on the back of a 13-year-old Korean girl. The halo nevus had a central pinkish mole and a depigmented patch. 20 mm in diameter(Fig.1). An autologous suction blister graft(ASBG) was performed, as previously described. In brief, a blister was made at the halo nevus by the application of liquid nitrogen. Blisters were then made at the donor site using suction on the patient's buttock. After removing the roof of the blister at the recipient site, the roof of the suction blister was grafted onto the recipient site. Two weeks after grafting, a topical corticosteroid application was recommended. At a 3 week follow-up, the leukodermic area was repigmented except for the marginal rim. At a 4 month follow-up, a new central whitish halo was observed around the central pinkish mole. At that time, the pinkish central papule was excised and examined using hematoxylin-eosin staining and immunohistochemical staining which contained an anti-human Ig-G antibody. The direct immunohistochemical staining process is outlined below. Before immunostaining, four-micron paraffin wax sections were cut from the block and mounted on slides using the avidin-biotine complex method. The sections were incubated with a biotinylated rabbit anti-human IgG antibody (DAKO, Carpinteria, CA, USA). After 30 minutes incubation, the sections were washed twice in PBS and soaked for 30 seconds. Then the avidin-biotin complex was developed for 5 minutes using avidin peroxidase. Finally the an-
tibody binding was visualized using 3,3-diaminobenzidine (DAKO LSAB Kit, Carpinteria, USA). The omission of the primary antibody served as a negative control. The histology of the central mole showed many nevus cells in the dermis intermingled with a dense population of lymphoid cells. The immunoreaction of the rabbit anti-human IgG was located in the nevomelanocytes and epidermal melanocytes (Fig. 2). The lymphoid cells were negative. After the excision of the central pinkish mole, repigmentation was completed and this condition persisted at a 3-year-follow-up.

**DISCUSSION**

The mechanism of depigmentation in a halo nevus is still not clearly understood. Bergman et al. demonstrated that the mononuclear lymphoid cells were a predominance of T cells with a relatively high proportion of cytotoxic/suppressor T cells. Mitchell et al. observed that lymphocytes isolated from patients with halo nevi or melanoma reacted to
human melanoma cells in a culture, whereas in patients with vitiligo and normal controls they did not. Copeman et al. found that a cytoplasmic antibody against melanoma was present in patients with resolving halo nevus yet absent in patients who had vitiligo without a halo nevus, juvenile melanoma or ordinary nevi. Berman et al. reported that, after incubation with diluted patient serum, an indirect immunofluorescence examination of skin from a patient’s giant congenital nevus and from the depigmenting halo revealed deposition of both IgG and C3 on the nevus cells. IgG and C3 deposition was only detected on the nevus cells that were located within the epidermis and dermis of the nevus and within the dermis of the depigmenting halo, and not on normal epidermal melanocytes. The deposition of IgM and complement on the nevus cells which can be determined by a direct immunofluorescence study, and the presence of the IgM antibody against nevus cells in a patient’s serum which can be detected by an indirect immunofluorescence study were both described by Tokura et al. However, other authors have failed to demonstrate a specific structural deposition of IgG, IgM, IgA, C3, and fibrin using direct immunofluorescent studies. Some have suggested that perinevus vitiligo is linked to a local toxicity that affects the melanocytes yet not the nevus cells and that nevus involution is linked to an immune reaction.

We have been able to successfully demonstrate the presence of immunoglobulin G on the nevomelanocytes and epidermal melanocytes of the central nevus using direct immunohistochemical staining. To our knowledge, this finding has not yet been recorded and does not coincide with any other reports. Our results on the resolutional stages may be explained by the very early age of the halo nevus. Many authors suggest that both humoral and cellular factors may be responsible for nevus destruction, and that a diffusible humoral factor may be responsible for the destruction of epidermal melanocytes in a surrounding depigmented halo. The authors suspect that 'the diffusible humoral factor' could perhaps be immunoglobulin G. It is clear that further study of the halo nevus at an early stage is needed.

Copeman et al. postulated that a halo nevus represents the accelerated and successful rejection phase of a pigmented mole which is developing a malignant change. The most common condition associated with a halo nevus is vitiligo, although a few cases may be associated with melanoma or dysplastic nevus. As can be seen in this case, repigmentation was completed after ASBG, a new pale halo then developed again, yet after the central nevus was excised the spread of depigmentation halted and repigmentation was completed. Nevertheless, a central mole has a pinkish color implying a regression phase, and these findings would seem to suggest that a central mole continuously contributes to the development of a leukodermic patch. Therefore, the authors believe the central mole induces a halo depigmentation so that early excision of the mole will halt a development of a depigmentation patch.
REFERENCES


