Dermatology Series: Skin of a lighter color

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Case history

A young woman presented with symmetrical skin color loss from around her eyes (Fig. 1) and the backs of her hands. It began in her teens and has been slowly progressive. Her hands are sometimes red and itchy after sun exposure. Her mother had a patch of white hair and became gray at an early age.

Q.1 What causes skin color loss?

**Hypopigmentation** is a lessening or lightening of skin color without total depigmentation. Common causes of hypopigmentation are pityriasis alba, a form of non-inflammatory atopic eczema predominantly on the face of children by either race or sun and post-inflammatory hypopigmentation.

In some countries, the patient may be concerned about the possibility of leprosy.

**Depigmentation** is the total absence of skin color due to loss of melanocytes. This may be post-traumatic such as it may be seen after firm cryosurgery - melanocytes are particularly sensitive to killing by cold. Halo naevi are common in adolescence and involve depigmentation around a benign melanocytic naevus with subsequent fading of the naevus. The topical use of monobenzyl ether of hydroquinone as a skin lightening product can cause depigmentation even at sites distant from the application. This patient has vitiligo.

Q.2 How can vitiligo be diagnosed?

Vitiligo is usually diagnosed clinically by:
- the age of onset
- the degree of color loss
- and the distribution of loss.

It commonly begins after 20 years of age. Both hypopigmented and depigmented macules can occur in vitiligo, often with a hyperpigmented edge. The skin within the macules is otherwise normal. In vitiligo the color loss is usually symmetrical, but can be dermatomal. Common sites of involvement include the hands and wrists, the face (especially periorbital and perioral skin) and hyperpigmented skin such as the genitalia and axillae. Depigmentation may occur at a site of skin trauma, for example, the Koebner (isomorphic) phenomenon. Wood's light examination clearly demonstrates the color contrast in vitiligo. Biopsy is not diagnostic as loss of melanocytes is seen in other depigmenting conditions. Autoimmune thyroid disorders, alopecia areata, pernicious anemia and diabetes mellitus occur more frequently in association with vitiligo than in the general population and screening for these diseases should be considered. A positive family history for autoimmune diseases can often be elicited.

Q.3 What therapeutic advice can we give the patient?

Sun protection is particularly important as the depigmented skin cannot tan and hence burns easily. Non-melanoma skin cancer may arise within vitiligo. Minimizing sun exposure also reduces the contrast with the adjacent normal skin if this is not permitted to tan. Cosmetic cover-up can be helpful for the patient from a quick and practical point of view on exposed sites. These include carefully blended foundations to match the normal skin color or the use of skin color lighteners.

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Figure 1 Depigmentation in the periorbital region.
stains which do not wash off. The success of active
treatment to some degree depends on the size of the
area of pigment loss and whether hairs in the area
remain pigmented (Fig. 2). Melanocytes migrate back
into the areas of vitiligo from the pigmented edges and
from pigmented hairs within the white area - the latter
resulting in a speckled appearance (Fig. 3). Treatment
options include the application of potent topical corti-
costeroids and psoralen plus ultraviolet A light (PUVA)
photochemotherapy. The psoralen may be applied
topically or taken orally and the UVA obtained either
from sun exposure or from a UVA light box. However,
treatment is long term and repigmentation unlikely to
be complete. The natural history is for the vitiligo to
progress slowly for years and then to stabilize. Sponta-
neous repigmentation particularly in sun exposed sites
may occur, but this is unusual.

**Figure 2** Depigmentation of occipital skin but with
sparing of hairs which remain pigmented.

**Figure 3** Speckled appearance due to repigmentation from
hair follicles following psoralen plus ultraviolet A light.