VITILIGO

Vitiligo is an acquired progressive autoimmune skin disorder characterized by white depigmented patches due to selective destruction of epidermal melanocytes. It is relatively common affecting approximately 1% of the population, equally prevalent between both sexes, and found in all races.

Vitiligo may appear at any time, although the onset is most commonly observed in children and young adults. Familial clustering is seen in 30% of the cases, and an autosomal dominant inheritance with variable expression and incomplete penetrance has been suggested. Vitiligo may be associated with other autoimmune disorders, such as thyroid disease, diabetes mellitus, pernicious anemia, Addison's disease, and alopecia areata.

The etiology is not completely understood.

- Immune hypothesis – aberration of immune surveillance results in melanocyte destruction.
- Neural hypothesis – a neurochemical mediator destroys melanocytes or inhibits melanin production.
- Self-destruction hypothesis – an intermediate product of melanin synthesis causes melanocyte destruction.
- Genetic hypothesis - melanocytes have an inherent abnormality that impedes their growth and differentiation.

Vitiligo appears as sharply defined milky white spots that slowly enlarge into large patches. The borders may be hyperpigmented or erythematous. The distribution is more or less symmetrical, with the most common sites of involvement being the face, neck, the distal portions of the arms and legs, the genitals, the nipples, the trunk, and around body orifices. Involvement of the mucus membrane is also frequently observed. Vitiligo is more common at sites of injury. Regimentation of body hair may be present. Scalp involvement is the most frequent, appearing as a localized patch of white or gray hair. Lesions are asymptomatic, although early lesions may be pruritus.

The following clinical variants have been described:

- Localized
    - Focal - one or more mauls in one area
    - Segmental – one or more mauls along dermatomes
    - Mucosal - mucous membrane alone
- Generalized
    - Acrofacial – distal portions of the arms and legs, plus the extremities
    - Vulgaris – scattered macules
    - Mixed
- Universal – complete or nearly complete depigmentation

The diagnosis is based on history and physical examination. Histologic examination with special stains is rarely necessary. Laboratory work up for associated autoimmune disorders may be warranted.

The differential diagnosis includes pityriasis versicolor, pityriasis alba, lichen sclerosus, leprosy, idiopathic guttate hypomelanosis, tuberous sclerosis, nevus anemicus or depigmentosus, post-

inflammatory hypopigmentation. and chemical leukoderma.
The course is usually chronic and slowly progressive. Spontaneous resolution is uncommon.

There is no definite treatment for vitiligo, and all treatments take a long time to be effective. Sun protection is important in order to avoid serious sunburns and to improve the aesthetic appearance. Aesthetic camouflage can be achieved with make up, self-tanning products containing dihydroxyacetone, and tattooing. Topical therapy with corticosteroids may be beneficial in early inflammatory lesions. Calcineurin inhibitors (tacrolimus, pimecrolimus) are an effective alternative, particularly when the disease involves the head and neck. S systemic phototherapy induces cosmetically satisfactory repigmentation in up to 70% of early or localized cases. PUVA treatment is often the most practical choice for treatment, especially in widespread vitiligo. Narrowband UV-B phototherapy was recently introduced with good clinical results.

Another novel approach is therapy with an Excimer laser at 308-nm to treat limited, stable patches of vitiligo. Systemic steroids (prednisone) have been used, although their prolonged use and toxicity are undesirable. Surgical treatment to induce regimentation involves punch grafts, mini grafts, and autologous melanocytes grafts.

If vitiligo is widespread and attempts at repigmentation do not produce satisfactory results, permanent depigmentation with 20% cream of monobenzylether of hydroquinone may be attempted in selected patients.

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