Vitiligo

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Rec date: Dec 20, 2014; Acc date: Dec 28, 2014; Pub date: Jan 02, 2015

Vitiligo

Vitiligo is an acquired depigmentation disorder of unknown etiology. Frequently symmetrical, asymptomatic, white macules and patches characterize the disease. Their size may increase in time due to the loss of functioning melanocytes. Nearly 0.5% - 1% of the European and American population is affected. There is no racial or gender predilection. Vitiligo may be seen at any age impairing the quality life of the patient significantly. The disease is not limited to skin and can trigger generalized syndromes mostly involving the immune system. Numerous systemic diseases have been found to be associated with vitiligo, supporting an autoimmune pathogenesis.

Studies have revealed that 10% to 15% of patients with vitiligo develop autoimmune diseases. The prevalence of autoimmune diseases in the general population is only 1% - 2%. Also the existence of autoantibodies against melanocytes strengthens the autoimmune theory on the etiopathogenesis of vitiligo.

Vitiligo frequently occurs at sites that are normally hyperpigmented, including the face (periorificial), the dorsal surface of the hands, nipples, axillae, umbilicus, sacrum. Infrequently mucosae and internal organs having melanocytes may be involved. Leukotrichia may be observed. A Wood's lamp may be of use in determining extent and activity of vitiligo, as well as monitoring response to therapy.

Clinicians should assess the psychological and quality of life effects of vitiligo on patients. Even tough the diagnosis of vitiligo is made clinically; histopathology can help confirm the diagnosis. Absence of melanocytes, scant inflammatory infiltrate, lichenoid interface dermatitis may be associated with the clinical picture.

Vitiligo has been classified as:

1. Localized (focal, unilateral/segmental, and mucosal)
2. Generalized (vulgaris, acrofacialis, and mixed)
3. Universal

Vitiligo can also be divided into segmental and nonsegmental types. Segmental form is possibly related to dysfunction of the sympathetic nerves. Nonsegmental form may be associated with systemic involvement.

Involvement of melanocytes can be observed. This requires a multidisciplinary diagnostic approach.

The association with autoimmune thyroid disease is best established and thyroid autoantibodies can be found in the sera of vitiligo patients.

Associations with other systemic disorders include:

1. Polyglandular syndrome type I and type II
2. Addison's disease, hypoparathyroidism
3. Diabetes mellitus type I
4. Autoimmune hemolytic anemia
5. Pernicious anemia
6. Hemolytic anemia

The incidences of these diseases are lower when compared to autoimmune thyroiditis.

First line treatments for segmental vitiligo are, avoidance of triggering factors, topical corticosteroids and topical calcineurin inhibitors. Second line choices are localized NB-UVB therapy, particularly excimer monochromatic lamp or laser and for the third line treatment methods we should consider surgical techniques if repigmentation is poor.

For nonsegmental vitiligo, treatment includes the avoidance of triggering factors, NB-UVB therapy, TCS, TCI, topical vitamin D analogues, excimer light. If the result is insufficient systemic steroids or immunosuppressants can be administered. Grafting, depigmentation are the best options for nonresponsive patients.

Treatment of vitiligo is problematic and prolonged however substantial knowledge has accumulated during the past years about pathogenic mechanisms leading to better treatment options. Vitiligo should be considered as a systemic disease and the evaluation of the patient, designing the treatment should be carried our accordingly.