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MEDICINES USED IN THE TREATMENT OF VITILIGO

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Abstract. The characteristics of vitiligo in 41 adults presenting to a university dermatology clinic in Sheffield, UK were studied in a case review. Of 41 patients, 29 women and 12 men, there were 37 European Caucasians, three Asian Caucasians and one black African. Symmetrical and acrofacial types of vitiligo predominated but there were no segmental cases.

Keywords: vitiligo, acrofacial, trauma, melanin, vitamin.

Age of onset was before 20 years in 17, with a mean of 28 years; the oldest onset was 74 years. The average duration of the disease was 16 years. Autoimmune thyroid disease was present in 14 cases (34%). Only seven (18%) gave a family history of vitiligo. A precipitating factor was identified in nine (22%), including pregnancy, sunburn and skin trauma. Topical steroid treatment was given in 33 and narrow-band ultraviolet B was prescribed in nine. This series is unusual in having no cases of segmental vitiligo, perhaps because no children are included, and in having a high prevalence of thyroid disease.

Vitiligo is a disease that causes loss of skin color in patches. The discolored areas usually get bigger with time. The condition can affect the skin on any part of the body. It can also affect hair and the inside of the mouth.

Normally, the color of hair and skin is determined by melanin. Vitiligo occurs when cells that produce melanin die or stop functioning. Vitiligo affects people of all skin types, but it may be more noticeable in people with brown or Black skin. The condition is not life-threatening or contagious. It can be stressful or make you feel bad about yourself.

Treatment for vitiligo may restore color to the affected skin. But it doesn't prevent continued loss of skin color or a recurrence.

Vitiligo signs include:

1) Patchy loss of skin color, which usually first appears on the hands, face, and areas around body openings and the genitals

2) Premature whitening or graying of the hair on your scalp, eyelashes, eyebrows or beard.

3) Loss of color in the tissues that line the inside of the mouth and nose (mucous membranes).

Vitiligo can start at any age, but usually appears before age 30.

Depending on the type of vitiligo you have, it may affect

1) nearly all skin surface: with this type, called universal vitiligo, the discoloration affects nearly all skin surfaces.

2)many parts of the body: with this most common type, called generalized vitiligo, the discolored patches often progress similarly on corresponding body parts (symmetrically).

3) only one side or part of the body: this type, called segmental vitiligo, tends to occur at a younger age, progress for a year or two, then stop.

4) one only or few areas of the body: this type is called localized (focal) vitiligo.

5) the face and hands: with this type, called acrofacial vitiligo, the affected skin is on the face and hands, and around body openings, such as the eyes, nose and ears.

It's difficult to predict how this disease will progress. Sometimes the patches stop forming without treatment. In most cases, pigment loss spreads and eventually involves most of the skin. Occasionally, the skin gets its color back.

Vitiligo occurs when pigment-producing cells (melanocytes) die or stop producing melanin — the pigment that gives your skin, hair and eyes color. The involved patches of skin become lighter or white. It's unclear exactly what causes these pigment cells to fail or die. It may be related to: a disorder of the immune system (autoimmune condition), family history (heredity), a trigger event, such as stress, severe sunburn or skin trauma, such as contact with a chemical.

Skin Layers of melanin: Melanin is a natural pigment that gives your skin its color. It's produced in cells called melanocytes.

People with vitiligo may be at increased risk of: social or psychological distress, sunburn, eye problems, hearing loss.

See your health care provider if areas of your skin, hair or mucous membranes lose coloring. Vitiligo has no cure. But treatment might stop or slow the discoloring process and return some color to your skin.

The physical presence of developed, amelanotic, non-scaly, chalky-white macules with transparent edges in a characteristic dispersion in the mouth, tips of the lower extremity, genitalia, and segment and sites of friction usually yields an unambiguous identification of vitiligo. Additional chemical testing is usually not required to establish vitiligo identification. A skin biopsy or additional testing is rarely required other than to rule out other illnesses. Non-invasive methods for determining whether a condition lacks melanocytes include in vivo confocal imaging and a skin sample. According to the histopathology of a vitiligo patch's center, the epidermis's melanin pigmentation has completely disappeared, and no melanocytes are found. Lymphocytes were only occasionally seen at the lesions' expanding edges. Portable ultraviolet (UV) illumination equipment that generates ultraviolet A (UVA), such as a Wood's lamp, could aid in the diagnosis of_vitiligo. It aids in the destruction of localized melanocyte and detects regions of depigmentation that may never be visible to human sight, particularly in those with

light skin. Under Wood's light, the vitiligo spots glow brightly blue-white and have distinct borders. Dermoscopy was used to distinguish vitiligo from other depigmenting diseases. Several hypopigmentation syndromes lack residual perifollicular pigmentation and telangiectasia, which are typical vitiligo features.

1) Topical treatment corticosteroids:

Corticosteroids have a significant medicinal impact in vitiligo by regulating and suppressing the inflammatory response. Topical corticosteroids (TCS) are the first-line treatment for vitiligo, whether potent (betamethasone valerate) or highly potent (clobetasol propionate). The therapeutic effects are stronger in sun-exposed areas, whereas acral zones typically produce poor results.

2) vitamin D3 Analogous (D3A):

Topical vitamin D3 analogues (D3A) are not effective as a stand-alone treatment for vitiligo due to their immunomodulatory properties, which decrease T cell function, promote melanocyte formation, and induce melanogenesis. Nonetheless, they are useful as supplements to other treatments. The optimum dosage for four weeks when applying the ointment and eight weeks when applying the cream is 100 g weekly on 30% of the body area, plus a combination of calcipotriol 0.005% and betamethasone 0.05%.

Vitiligo is a multivariate skin condition with a complicated pathophysiology. Despite recent significant advances in human knowledge of this condition, the origin and pathophysiology of vitiligo remain unknown. There are still questions about what causes melanocyte degeneration, and more research is needed to fully understand the etiology of vitiligo. It is critical to understand the biological messengers and molecular processes that result in metabolic abnormalities, melanocyte destruction, and autoimmune disorders to find novel treatment objectives and medications that may arrest the spread of the illness and possibly treat vitiligo. Natural cytokine-targeting treatments have been shown to be effective

in treating conditions such as psoriasis and vitiligo. As a result, attacking the interferon (IFN)-chemokine axis with current or future medicines is appealing and intriguing. The inconsistent therapeutic progress and recurring nature of vitiligo medication can be discouraging at times. Customized treatment plans must be developed based on the type of vitiligo, whether it is active, and the side effects of the drug. There are only a few vitiligo treatments available, and none of them can reliably cause repigmentation in every individual. Additional scientific and therapeutic research is required to develop new therapeutic strategies and gain a better understanding of the vitiligo etiology. Many new medicines are on the horizon, and the majority of information about them is provided by case studies or episodes. Additional randomized controlled trials are required to accurately assess their effectiveness.

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