Chapter

Introductory Chapter: Depigmentation

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1. Introduction

Depigmentation, lightening of the skin and mucosa, can be caused by local or systemic conditions, and there may be partial or complete loss of pigment [1]. Although depigmented patches may not matter in Caucasians, it is very serious for pigmented skin [2]. Depigmentation can also be a therapeutic goal for cosmetic treatment. Many vitiligo patients, who received depigmentation treatment, experience paradoxical jealousy because of their clean white skin. To improve facial blemishes, many people spend their money for laser, chemical peel, and cosmeceutical [3, 4].

Depigmentation can occur hereditarily or acquiredly. Hereditary diseases if depigmentation includes following diseases (**Figure 1**) [3]. Oculocutaneous albinism consists of a group of genetic disorders characterized by diffuse pigmentary dilution due to a partial or total absence of melanin pigment within melanocytes of the skin and eyes. Piebaldism is an genetic disorder characterized by poliosis and congenital, stable, circumscribed areas of leukoderma due to an absence of melanocytes within involved sites. Waardenburg syndrome is a rare genetic disease characterized by various combinations of depigmentation of skin and irides, and congenital deafness. Hermansky–Pudlak syndrome is a rare genetic disease of depigmentation showing pigmentary dilution of the skin, hair, and eyes, and serious systemic manifestations including hematopoietic, immune, pulmonary, renal, and cardiac symptoms. Chédiak–Higashi syndrome is a rare genetic disorder showing features of oculocutaneous albinism, ocular symptoms, hematologic and neurologic manifestations. Tuberous sclerosis complex is an autosomal dominant disorder characterized by neuroligic disorders and skin findings including depigmented macules. Depigmentation along the lines of Blaschko reflects mosaicism characterized by a clone of skin cells with a decreased ability to make pigment. Hypomelanosis of Ito, linear nevoid hypopigmentation and nevus depigmentosus are considered to represent manifestations of cutaneous mosaicism.

Acquired diseases of depigmentation includes vitiligo, hypomelanosis secondary to cutaneous inflammation (postinflammatory hypopigmentation, pityriasis alba, sarcoidosis, hypopigmented mycosis fungoides, lupus erythematosus and lichen sclerosus et atrophicus), infectious hypomelanosis (tinea versicolor, leprosy, kala azar...), chemical or pharmacologic hypomelanosis (chemical leukoderma, hypomelanosis by strong steroid), hypomelanosis from physical agetnts (burn, laser, abrasion...), and miscellaneous (idiopathic guttate hypomelanosis, persistent macular hypomelanosis...) (**Figure 2**) [3].

Diseases of depigmentation can occur hereditarily or acquiredly. Hereditary diseases of depigmentation were reviewed excellently by Prof. Carrasquillo and colleagues. Albinism, piebaldism, white patches of tuberous sclerosis, Hermansky-Pudlak syndrome, Chédiak-Higashi syndorme, Waardenburg syndrome, and

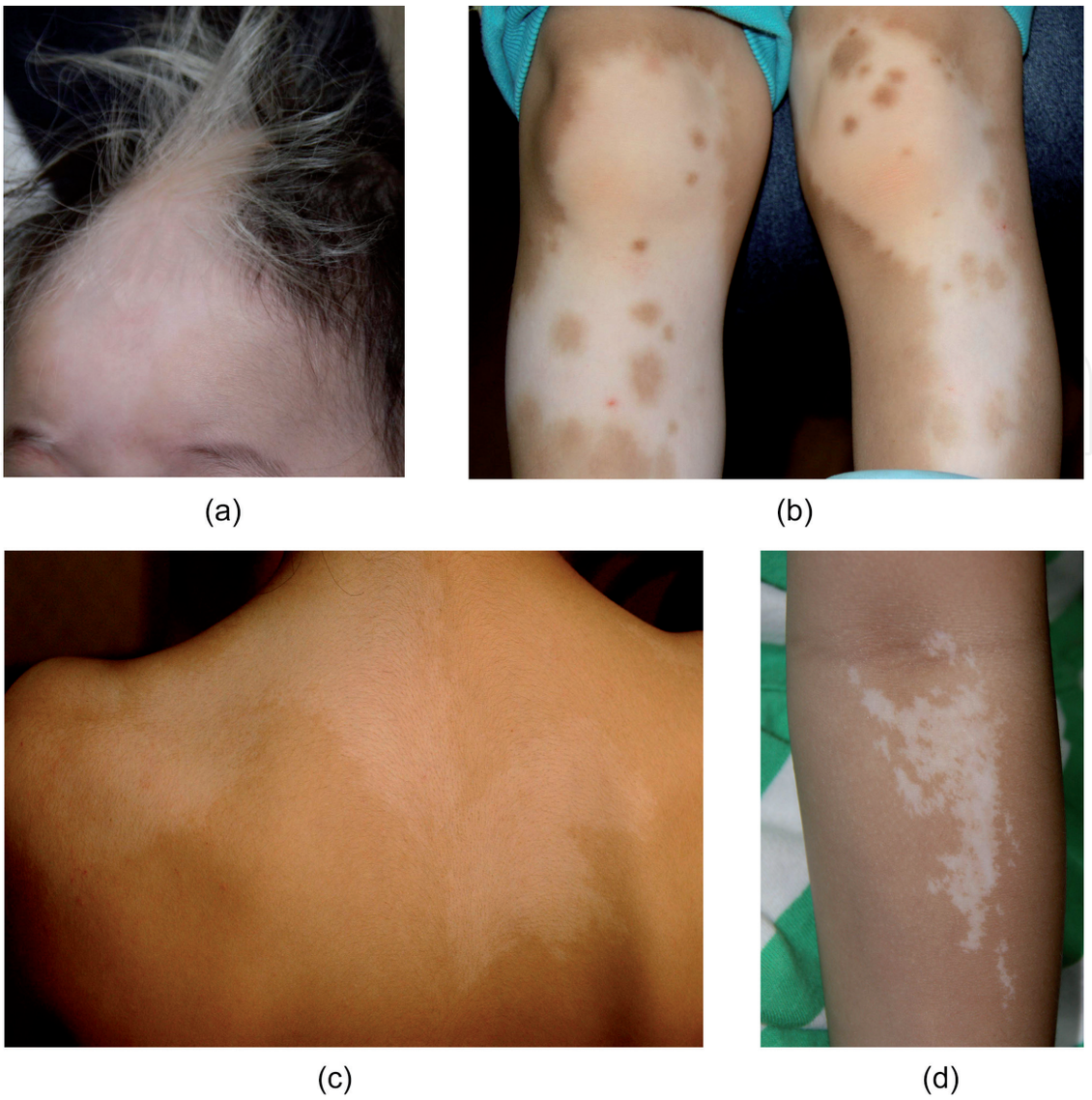

Figure 1.
Hereditary diseases of depigmentation. a. White forelock of piebaldism. b. Piebaldism affecting legs. c. Linear nevoid hypopigmentation. d. Nevus depigmentosus.

pigmentary mosaicism including hypomelanosis of Ito and nevus depigmentosus would be examples of them (**Figure 1**) [3].

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Vitiligo is a common acquired disease of depigmentation, and afflicts and frightens so many patients. In vitiligo, melanocytes that produces melanin pigment of the skin are destroyed, and it can occur systemically to affect whole body or locally/segmentally affecting part of the body (**Figure 2a** and **b**) [3].

For the treatment of vitiligo, various lasers including 308 nm excimer laser are used successfully which has been used additionally with phototherapy and topical or systemic medications [5].

In patients with extensive vitiligo, depigmentation can be an easier and cosmetically more acceptable option. It includes various chemical and physical modalities.

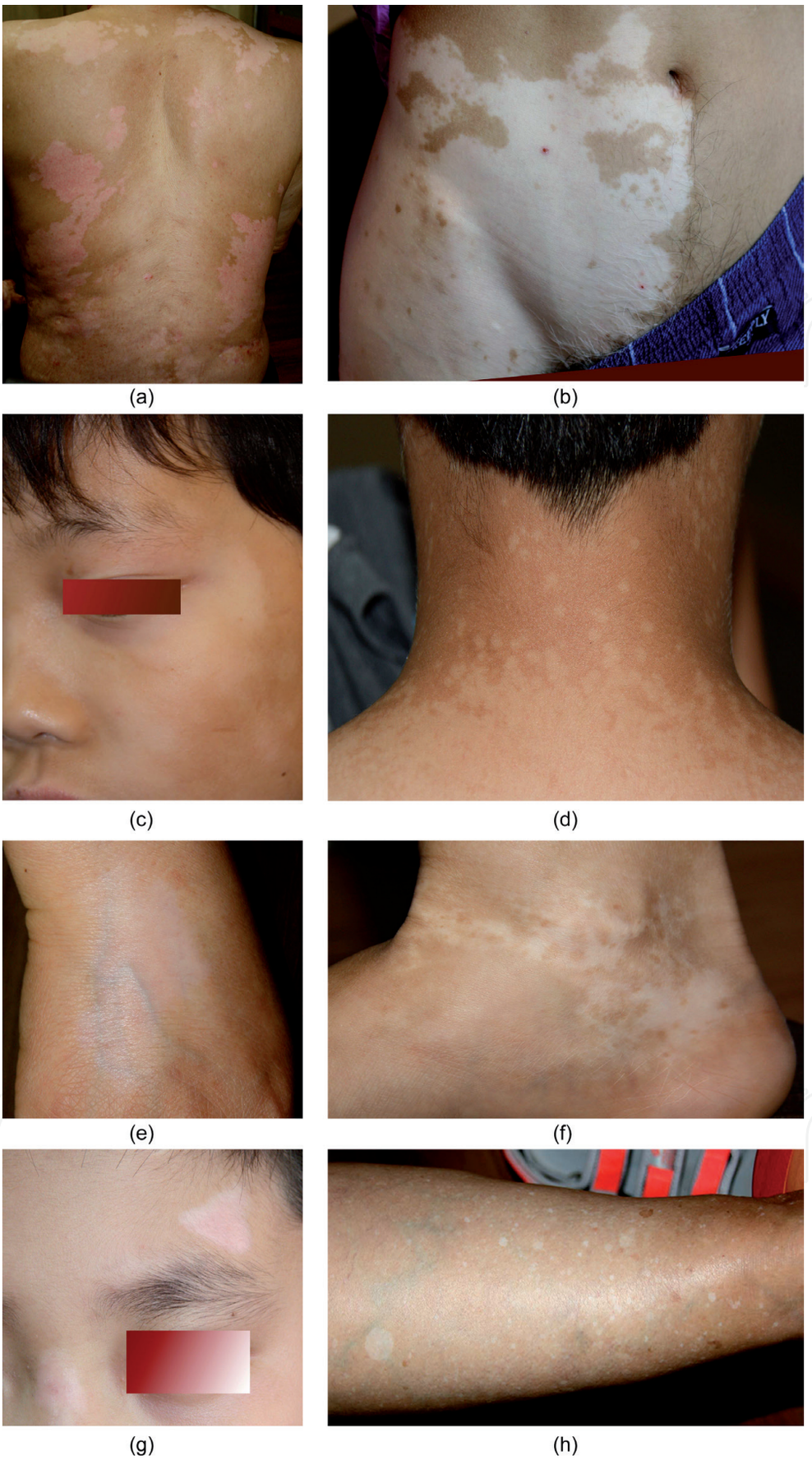

Figure 2.
Acquired diseases of depigmentation. a. Systemic vitiligo affecting the whole body. b. Segmental vitiligo affecting one side of the body with white hairs (poliosis). c. Pityriasis alba, hypomelanosis secondary to cutaneous inflammation of atopic dermatitis. d. Hypomelanosis by tinea versicolor. e. Hypomelanosis by strong steroid (injection of triamcinolone acetonide). f. Hypomelanosis from physical injury induced by burn. g. Hypomelanosis from physical injury induced by abrasion wound. h. Idiopathic guttate hypomelanosis in older patients which belong to miscellaneous hypopigmentation.

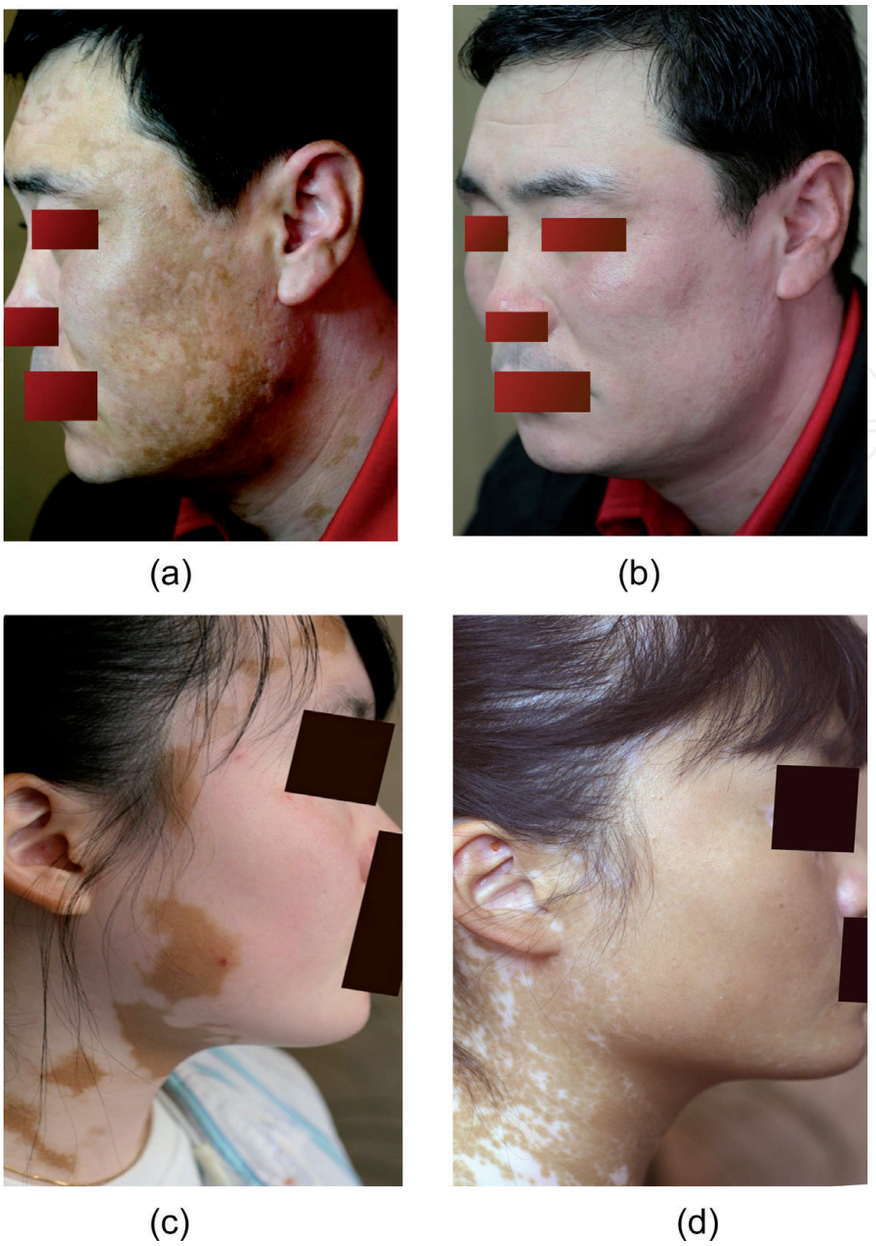

Figure 3.
Depigmentation therapies for vitiligo. a. Systemic vitiligo patient before depigmentation therapy by 20% monobenzyl-ether of hydroquinone (MBEH). b. Systemic vitiligo patient after 6 months of depigmentation therapy by 20% MBEH. c. Systemic vitiligo patient before 308 nm excimer laser therapy. d. Systemic vitiligo patient after 6 months of 308 nm excimer laser therapy.

As in **Figure 3a** and **b**, depigmentation therapy can be an ideal treatment for advanced vitiligo patients. But cases presented in **Figure 3c** and **d** suggests restoring normal pigmentation is also an excellent option, and it would be better to be decided by patient's choice.

Cosmeceutical for depigmentation is a big market field for investigators and cosmetic companies. We have tragedic experiencies of chemical leukoderma and vitiligo induced by depegmentation cometics manufactured by Kanebo containing rhododendrol [6, 7]. Researchers put tremendous efforts to overcome it and there are a lot of active researches to find out newer materials for depigmentation [4, 7].

DOI: http://dx.doi.org/10.5772/intechopen.89099

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