COMMENTARY

# A Concise Approach to Childhood Hypopigmentation

In this issue, van Geel *et al.*[1] published a review on differential diagnoses of hypopigmentation disorders in children. Childhood hypopigmentary disorders pose several diagnostic challenges. Firstly, the abnormalities could present with asymptomatic, subtle clinical change difficult to be recognized by patients, parents, and clinicians. Secondly, the range of the differential diagnosis is broad and histological examination of the lesion is rarely diagnostic. Thirdly, there are several conditions that typically present with inflammatory component but sometimes present with hypopigmented lesions. Fourthly, it could occur as a primary disorder or be associated with various medical conditions. Therefore, a rational approach is required in evaluating children with a hypopigmented lesion.

Hypopigmentation disorders in children can be classified based on the extent of involvement. In diffuse hypomelanosis, the total area of the skin is affected. Disorders in this group are usually due to mutations of gene responsible for melanin biosynthesis or melanosome formation and the number of melanocytes is usually normal. The prototype in this group is oculocutaneous albinism in which the patient's skin colour can be ranged from complete pigment loss to partial pigmentation. Other differential diagnoses include Hermansky-Pudlak syndrome, Chediak-Higashi syndrome, Griscelli syndrome, Elejalde syndrome, Cross syndrome, Prader-Willi syndrome, Angelman syndrome, Ectrodactyly-Ectodermal dysplasia-Cleft lip/palate syndrome, Menkes syndrome, copper and selenium deficiency, phenylketonuria, homocysteinuria, and histidinemia. These disorders are typically associated with systemic manifestations e.g., eye abnormalities, haematological abnormalities, neurological abnormalities, and immune deficiency. Therefore, a complete history taking and thorough physical examination are mandatory in children presenting with diffuse hypopigmentation.


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Localized and discrete lesions can be classified into amelanosis and hypomelanosis based on the finding under Wood's light examination. Some entities in this group can be associated with systemic manifestations (e.g., tuberous sclerosis, incontinentia pigmenti, etc.). Non-melanotic conditions (e.g., nevus anemicus, Bier spots) should be excluded by diascopy test. Among amelanotic conditions, the differential diagnosis includes vitiligo, Vogt-Koyanagi-Harada syndrome, Alezzandrini syndrome, piebaldism, Waardenburg syndrome, Tietz syndrome, halo nevi, and postinflammatory depigmentation. Hypomelanotic lesions can be categorized into linear and non-linear lesions. Linear lesions include pigmentary mosaicism, incontinentia pigmenti stage IV, linear morphea, linear lichen sclerosus, Goltz syndrome, and Menkes syndrome (female carrier). The differential diagnosis of localized non-linear lesions includes ash leaf macules in tuberous sclerosis, nevus depigmentosus, postinflammatory hypopigmentation, pityriasis alba, pityriasis versicolour, hypopigmented mycosis fungoides, tuberculoid leprosy, hypopigmented sarcoidosis, leucoderma at sites of vaccination, cryothrapy for warts as well as topical medications like potent corticosteroids.

Numerous inflammatory skin conditions such as pityriasis lichenoides chronica, lichen striatus, chicken pox, and atopic dermatitis tend to develop postinflammatory hypopigmentation rather than hyperpigmentation, especially in dark-complexioned individuals. The inflammatory and hypopigmented lesions often coexist, but when the inflammatory lesions are absent, it is more difficult to make the diagnosis. Thus, serial physical examinations are necessary.[2] Moreover, some typically adult-onset conditions e.g., tuberculoid leprosy, hypopigmented mycosis fungoides,


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and sarcoidosis can sometimes present in childhood and should be also kept in mind.[3,4]

Investigations in children presenting with hypopigmented lesions depend on the presumptive diagnosis. In diffuse hypomelanosis, the investigations should be focused on the associated systemic findings. Molecular genetic analysis and karyotyping of the affected skin and blood may be considered. In localized hypomelanosis and amelanosis, it is readily diagnosed by clinical findings. Histological examination is usually non-diagnostic. However, it may be useful in some conditions e.g., inflammatory disorders, hypopigmented mycosis fungoides, leprosy, and sarcoidosis.

The mainstream management in patients with diffuse hypopigmentation is sun avoidance, use of broad-spectrum sunscreen, and treatment of associated systemic involvement. In localized and discrete hypopigmented conditions, the management depends on the nature of the disease. For hypopigmented lesions associated with inflammatory dermatoses, the underlying dermatitis should be treated. Infectious conditions should be treated as such. Autologous epidermal cellular grafting results in good to excellent repigmentation in segmental vitiligo, halo nevi, and piebaldism (> 80% of patients achieved more than 75% repigmentation) with good long-term stability.[5] Its results in nevus depigmentosus and generalized vitiligo were inferior, and relapse is possible.[6] Cosmetic camouflage and micropigmentation may be the only option in some hypopigmented disorders. Additionally, psychosocial issue is equally important. Children with lesions in exposed areas may be subject to humiliation, psychic trauma, or even feeling of social rejection. Good communication among the physician, patient, parents, and schoolteachers is essential and should never be overlooked in taking care of this group of patients.

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