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Pityriasis Alba

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Pityriasis alba (PA) is a common benign condition in children that has no definitive treatment. Its etiology and pathogenesis are still poorly understood. Recent studies have found direct correlations between the incidence of PA and atopy, amount of sun exposure, lack of sunscreen use, and frequency of bathing. It is often an incidental finding on physical examination because it is usually asymptomatic. Although treatment with emollients and mild topical corticosteroids may accelerate the repigmentation, they have limited efficacy. Without intervention, the lesions normally resolve within months to years. Extensive PA and pigmenting PA are rarer variants.

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Pityriasis alba (PA) was first recognized more than 80 years ago as a localized disorder of hypopigmentation that was less marked than vitiligo.[1] PA mostly affects the head and neck region of children. Patients are otherwise healthy and have no associated illnesses. Some authors believed that these spots were more common in individuals with darker complexions.[2] PA spots are actually a relatively common phenomenon in all skin colors but are simply more noticeable on darker skin.[2] PA does not respond to antibiotics or antifungals and has not been definitively associated with any specific etiologic agent.[2-4] A limited response is seen with corticosteroids. However, PA tends to resolve with time. With no identifiable cause, specific histologic pattern, or effective treatments, PA has remained an enigma.

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Epidemiology

PA is common, affecting between 1.9% and 5.25% of preadolescent children.[5-9] In one series of patients with PA, 81% were 15 years or younger.[2] In a different retrospective analysis of cases, 90% were aged 6 to 12 years, and 10% were aged 13 to 16 years.[10] There is no gender predisposition.[2,5-7] PA is found in all parts of the world.[2,4-7] One series shows a markedly higher incidence among school children of poorer socioeconomic background.[7]

Etiology and Pathogenesis

Many terms have been used to describe PA, including *erythema streptogenes*, *pityriasis streptogenes*, and *impetigo furfuracea*.[4] However, these names imply a known cause. Bacterial, fungal, and parasitic infections are more frequent among individuals with PA, but no definitive associations have been found.[3,10] Nutritional deficiencies also are common.[2,3,10] Some authors have suggested that xerosis and atopy are implicated in the pathogenesis of PA.[2,4,10,11] The etiology of these hypopigmented lesions remains elusive, rendering the term *pityriasis alba* accurate and appropriate.

Possible causes of PA include xerosis, sun exposure, wind, and soap.[4,7,12] A retrospective study found that patients with PA had exposed themselves to more sunlight than healthy patients and did not use sunscreen regularly.[13] Frequent bathing and hot baths also were directly related.[13] All of these correlations are supported by histologic evidence, suggesting that the state of hydration of the stratum corneum in PA is lower than that of healthy skin.[14]

PA also has been linked to vitamin deficiencies. Individuals with PA have lower serum levels of copper.[15] Because copper is a cofactor for an enzyme needed for melanin production (tyrosinase), a copper deficiency may play a role in the pathogenesis of PA.

PA may be best characterized as a form of dermatitis related to atopy.[10,16] This eczematous

disorder results in hypomelanosis. Melanocytes and melanosomes are found in decreased numbers in tissue samples, with no detectable defect in melanosomal transfer to keratinocytes.[2,11,16]

Clinical Features

Because PA is not symptomatic, many patients do not mention the presence of lesions to their physicians. It is often found incidentally.[2] There are usually 2 to 3 round macules with well-defined borders that are 0.5 to 5 cm in diameter. Most lesions appear on the face but also can occur on the upper extremities and occasionally on the lower extremities.[2] Facial spots commonly occur on the forehead (63%) and malar ridges (57%). Facial lesions are found less frequently at the angles of the mouth (37%) and on the lateral supraorbital region (35%).[10] This is in contrast to vitiligo, which has a predilection for the perioral and periocular areas. PA usually has no associated symptoms, but mild pruritus is not uncommon.[16]

PA often starts as a pink patch with an elevated border. After several weeks, the patch fades, leaving a paler spot covered by a powdery white scale.[17] This spot will progress to a smooth hypopigmented macule that can persist from 6 months to 7 years.[10] This course may be prolonged in atopic patients.[16] There is no agreement whether any seasonal variability exists; however, PA is sometimes exacerbated by dry weather. The lesions also can become more visible in summer, when the surrounding skin is tanned. Certain patients may display affected areas in all 3 stages simultaneously, while others may be seen with lesions all of the same stage. Following resolution, PA may recur at the same location.

Histology

Most cases of PA can be diagnosed clinically. Histologic analysis is often unnecessary and sometimes nonspecific. PA has areas of hyperkeratosis, parakeratosis, and acanthosis, but these are not always present.[2,5,12,16] Small amounts of spongiosis also may be found.[2,5,12] Melanocytes and melanosomes are present in decreased amounts in the basal layer of the epidermis.[2,11,13] The finding may be due to inflammation that interferes with the production of melanin.[4] A histopathologic diagnosis of PA can be made when the following features are seen: irregular pigmentation by melanin of the basal layer, follicular plugging, follicular spongiosi, and atrophic sebaceous glands.[18]

The variable histology may be attributable to the stage of PA.[17] Throughout its course, PA shows hyperkeratosis, parakeratosis, and mild acanthosis. Early on, there is follicular plugging and atrophic sebaceous glands. Perivascular lymphocytic infiltrates and edema are evident in the dermis. The intermediate stage is characterized by damage to the hair follicle and spongiotic edema. On electron microscopic examination, hypopigmented spots show reduced numbers of irregularly patterned melanocytes and melanosomes. Follicular changes are best seen during this stage, making the diagnostic value of the tissue analysis highest during this time. Late-stage PA shows a pattern typical of chronic dermatitis and also exhibits irregular melanization, with occasional hyperpigmented regions among areas of normal and hypopigmented lesions.

Atypical Forms of PA

Several variations of PA may be seen. Extensive PA is an entity characterized by features of PA distributed in a generalized fashion[13,19] and is not preceded by erythema. Extensive PA is seen more commonly in adults. The inferior portions of the torso are usually affected in a symmetrical pattern. Extensive PA is otherwise asymptomatic. Patients usually give no history of atopy. Much like classic PA, the histology is nonspecific. It shows a reduced number of functional melanocytes with a fewer number of melanosomes. Melanosomes are found in a normal distribution, and transfer to keratinocytes is undisturbed. Hyperkeratosis and parakeratosis are variable, as are intercellular edema and intracytoplasmic lipid droplets.

Pigmenting PA is a variant that may be associated with superficial dermatophyte infection and classic PA.[20,21] Pigmenting PA appears as a bluish hyperpigmentation surrounded by a hypopigmented scaly area much like classic PA. Cases almost always appear on the face and rarely occur with simultaneous extrafacial involvement. Furthermore, 65% of affected patients also have a fungal infection; 30% will have concurrent classic PA. The pigmented area is attributed to melanin deposits in the dermis, a feature not seen in classic PA.[20]

Differential Diagnosis

PA can mimic a variety of other inflammatory skin conditions associated with postinflammatory hypopigmentation. Psoriasis can be distinguished from PA based on history and physical examination. PA with a psoriasislike distribution may occur.[11,22]

PA can be mistaken for fungal infections such as tinea faciei. Tinea versicolor also can sometimes be hypopigmented.[23-25] These conditions can be distinguished by microscopic examination of a potassium hydroxide preparation of the lesion.

Nevus depigmentosus is a hypopigmented macule that can be distinguished from PA because 92.5% are present before the age of 3 and have well-defined borders.[26] Microscopy results of nevus depigmentation reveal no change in the number of melanocytes.

In vitiligo, the contrast between normal and affected skin is greater than that of PA. Also, the complete loss of melanin in vitiligo can be demonstrated by the use of a Wood lamp.[27]

Nevus anemicus, a pharmacologic nevus, is a congenital anomaly found in children that appears as well-defined patches of paler skin.[28] It may be diagnosed by stroking the affected skin; the pale areas of nevus anemicus will become erythematous. Histology results reveal normal-appearing skin.

The hypopigmented ash-leaf spot of tuberous sclerosis may be distinguished from PA[29] because it is usually evident on the trunk and extremities at birth. Patients with such findings should be investigated for other features of tuberous sclerosis.[29]

Mycosis fungoides rarely has hypopigmented macules,[30,31] but individuals with no other symptoms have been mistakenly diagnosed with PA.[32] This is seen mostly in dark-skinned individuals. Other considerations in the differential diagnosis include hypopigmentation secondary to leprosy; nummular eczema; or use of a topical steroid, retinoic acid, or benzoyl peroxide.

Treatment

Treatment for patients with PA revolves around assuring patients that the disorder is self-limiting and not dangerous.[16] No therapy is completely successful. Clinicians should recommend measures that limit an individual's exposure to possible etiologic factors, such as decreasing sun exposure, regular use of sunscreen, and reducing the frequency and temperature of baths.[14] Sunscreen also may help lessen the pronunciation of PA after sun exposure.

Universally accepted treatments include emollients and lubricants.[2,4,10] These may help lessen dryness and irritation. Topical hydrocortisone 1% or desonide can be helpful in cases with inflammation. In children, lesions on the face should only be prescribed mild nonhalogenated steroids.[33] More potent steroids, such as aclometasone dipropionate and hydrocortisone valerate, are recommended for nonfacial lesions.[11] Topical tretinoin has been used with some success.[16]

Extensive PA does not respond to topical steroids but has been found to resolve with psoralen-UVA therapy.[18] Pigmenting PA may resolve when treated with oral antifungals.[19,20]

Studies from the Middle East indicate that PA may be associated with poor socioeconomic conditions.[7] Improvement in living standards and education may help decrease the prevalence of this skin disorder.

Conclusion

PA is a common, localized, hypopigmentive disorder seen in children that has no reliable treatment but resolves with time. It is recognized more often in individuals with darker complexions but occurs equally in individuals of all skin colors. The precise mechanism behind this disorder has yet to be elucidated. Studies have uncovered associations between the incidence of PA and atopy, amount of sun exposure, lack of sunscreen use, and frequency of bathing. Limiting these factors may help decrease incidence and alleviate symptoms. Emollients and mild topical corticosteroids also help with symptoms and may have a limited effect in accelerating repigmentation. Less common variants of PA, namely pigmenting PA and extensive PA, have a better response to their respective treatments. A definitive intervention for PA may come from a better understanding of its pathogenesis.

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