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Pityriasis Alba Revisited: Perspectives on an Enigmatic Disorder of Childhood

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Pityriasis alba (PA) is a localized hypopigmented disorder of childhood with many existing clinical variants. It is more often detected in individuals with a darker complexion but may occur in individuals of all skin types. Atopy, xerosis, and mineral deficiencies are potential risk factors. Sun exposure exacerbates the contrast between normal and lesional skin, making lesions more visible and patients more likely to seek medical attention. Poor cutaneous hydration appears to be a common theme for most risk factors and may help elucidate the pathogenesis of this disorder. The end result of this mechanism is inappropriate melanosis manifesting as hypopigmentation. It must be differentiated from other disorders of hypopigmentation, such as pityriasis versicolor alba, vitiligo, nevus depigmentosus, and nevus anemicus. Alleviation of the various risk factors via patient education on proper skin care and hygiene, use of lubricants and emollients, topical corticosteroid therapy in the presence of inflammation, and the novel administration of topical anti-inflammatory drugs such as calcineurin inhibitors can play a crucial role in promoting remission or resolution.

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Pityriasis alba (PA) is derived from the Greek word *pityron* and the Latin word *albus*, which signify bran or branlike and white, respectively. Widely recognized as an idiopathic hypopigmentary disorder evident as macules,[1] it was first described more than 80 years ago.[2] Mainly seen in the pediatric population, it primarily affects the head and neck region, with the face being the most commonly involved site.[1-3] Pityriasis alba is present in individuals with all skin types, though it is more noticeable in those with a darker complexion.[1,3] This condition also is known as furfuraceous impetigo, erythema streptogenes, and pityriasis streptogenes.[1] The term *pityriasis alba* remains accurate and appropriate given the etiologic elusiveness of the disorder.

Epidemiology

Pityriasis alba primarily affects preadolescent children aged 3 to 16 years,[4] with onset typically occurring between 6 and 12 years of age.[5] Most patients are younger than 15 years,[3] with up to 90% aged 6 to 12 years and approximately 10% aged 13 to 16 years.[6] The belief that there is equal prevalence and incidence among males and females[3,7-10] is questionable. For example, a point-prevalence study in Romania showed a statistically significant male preponderance of the disease (P=.007).[11] Others suggest male to female ratio estimates as high as 2 to 1.[8,12,13] The prevalence of PA in the overall pediatric population ranges from 1.9% to 8.4%.[7-9,11,13-15] In children with poorer socioeconomic backgrounds, incidence rates are even higher, with an overall prevalence of up to 90%.[9,16] Pityriasis alba has a worldwide distribution.[3,7-9,17-19]

Etiology and Pathogenesis

We believe that PA results from simultaneous exposure to different culprits or repeated exposure to any single agent, and the hypopigmentation seen in PA is due to changes in melanosis resulting from persistently poor skin hydration. The latter may be secondary to an inflammatory disorder such as atopy, or secondary to noninflammatory factors such as frequent bathing, drying soaps, or overall poor skin hygiene.

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The end result is a decrease in the number of melanocytes and melanosomes with normal melanin synthesis and melanosome transfer to epidermal cells.[3,10,20,21]

Atopic dermatitis is a risk factor for PA.[3,6,10,17,20,22-24] Atopy is more common in developed countries and may be related to socioeconomic and environmental factors. Atopic dermatitis is identified in as many as 85% of patients.[25] We believe that PA lesions may occur secondary to preexisting atopic dermatitis, a common etiologic agent in hypomelanosis.

Sun exposure may be a risk factor. Patients with PA are more likely to have substantial sun exposure and less likely to consistently use sunscreen.[12] However, sunlight simply makes PA more apparent,[22] which is especially true in individuals with higher Fitzpatrick skin types who tan more easily and are more likely to remain in the sun longer.[12] Findings for pityriasis versicolor alba are comparable[26]; the dose of sun exposure is more important than UV susceptibility.[12]

Other risk factors include xerosis, mineral deficiency, and inappropriate skin care. Pityriasis alba can be precipitated by xerosis.[7,9,10,12,17,27,28] Pityriasis alba lesions are exacerbated by dry skin.[25] There is evidence of a lower state of hydration in PA relative to normal skin.[29,30] Frequent bathing, hot baths, soaps, and wind,[12] as well as poor socioeconomic status and hygiene, have all been implicated.[9,16] Hypocupremia was found to be remarkably related to PA.[31] Given the role of copper in melanogenesis, it could conceivably be involved in the pathogenesis of PA.[31]

Clinical Manifestations

Pityriasis alba can be pruritic, though it usually is asymptomatic and often is incidentally detected.[3,5,10] It most often occurs on the face, particularly the forehead and malar ridges,[6] but it also may occur on the extremities.[3,32] Pityriasis alba usually is evident with 2 or 3 macules or patches at a time that progress in several stages. The first (early) stage begins as an erythematous patch with an elevated border that may last for weeks. The second (intermediate) stage manifests with the replacement of the patch by a smooth scaly layer.[5] The early and intermediate stages are marked by the presence of pinpoint follicular papules.[32] The third stage presents as a visible, round, hypopigmented macule 0.5 to 5 cm in diameter with well-defined borders and loosely adherent scales.[4,10] The patient usually seeks medical treatment during this stage.[4]

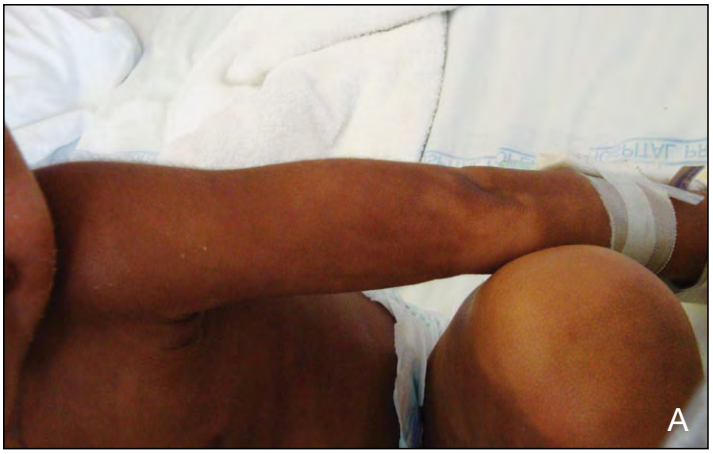
Histologic Manifestations

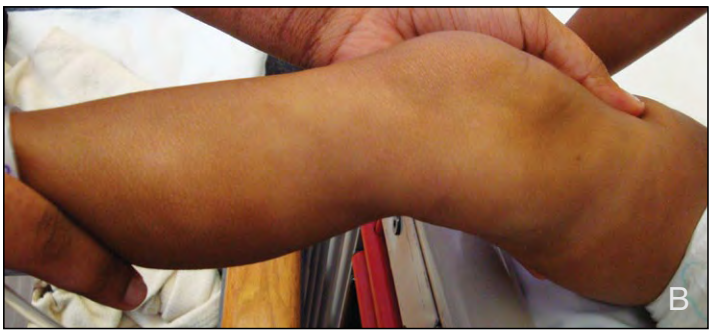
Pityriasis alba can be diagnosed using clinical findings alone. Histology aids in unclear diagnoses, though it may be quite variable[21] due to the different stages of PA.[5] Spongiosis is a consistent histologic finding.[7,10,21,27,33] Dermal perivascular lymphocytic infiltrates,[21] acanthosis, hyperkeratosis, and parakeratosis often are seen.[4,7,10,25,27,33] However, when present, the epidermal changes occur in all 3 stages of PA.[1] Each stage presents some unique histologic findings. Follicular plugging and atrophic sebaceous glands are prominent in the early stage. Damaged hair follicles are visible in the second stage. Irregular melanization, manifesting as hypopigmented macules often with small areas of hyperpigmentation, is most prominent in the late stage. Evidence of long-term dermatitis[34] as well as reduced numbers of abnormally patterned melanocytes and melanosomes appear at this stage. Follicular plugging and spongiosis, atrophic sebaceous glands, and irregular melanization of the basal layer are the key diagnostic findings, with the highest yield achieved during the first 2 stages.[34]

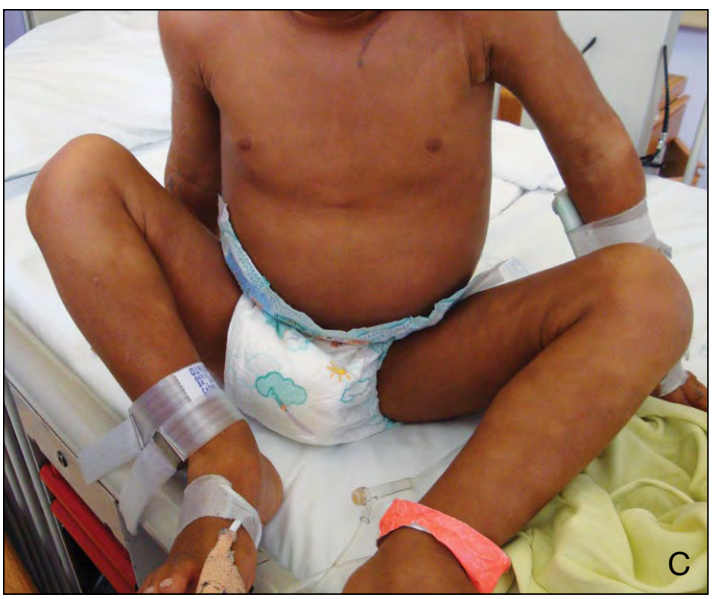
Clinical Variation

There are 2 clinical variants of classic PA (CPA): endemic PA, which occurs in children living in poor socioeconomic conditions, and atopy-related PA.[16] The improvement of endemic PA in a few months with mild topical hydrating creams, topical antimicrobials, and sunscreens, and its deterioration with the use of topical corticosteroids and calcineurin inhibitors, suggest distinctive etiologies,[16] with clear prognostic and therapeutic implications. Classic PA also must be distinguished from extensive PA (EPA). Both show fewer melanocytes and melanosomes,[10] and both manifest as hypopigmented macules with scaling. However, EPA is more common in adults, has a more generalized and symmetric distribution, tends to appear on the trunk, and usually is not associated with a history of atopy or evidence of erythema.[1] There may be both an atopic as well as an idiopathic variant of EPA.[35] It also tends to have a more prolonged course than CPA,[27] with a female preponderance.[33] The Figure illustrates an unusual case of late-stage PA with diffuse anatomic distribution, no erythema, and no facial involvement in a child. We believe this patient may have EPA, which usually occurs in adults, making it a rather unusual case. The patient did not have a history of atopy, pruritus, or pain.

Classic PA and EPA may be different diseases altogether. Extensive PA and progressive extensive or progressive macular hypomelanosis (PMH) are almost indistinguishable as separate disease entities.[33] The term *extensive pityriasis alba* may be a misnomer given the complete absence of the eczematous changes that are characteristic of CPA.[36,37] Progressive macular hypomelanosis, similar to EPA, occurs mostly in adults and demonstrates ill-defined, nonscaly, hypopigmented macules on the trunk







Hypopigmentation of the left knee and left arm (A). Late-stage pityriasis alba with smooth macules greater than 5 cm in diameter evident on the medial aspect of the patient's right lower extremity (B). Diffuse anatomic involvement of pityriasis alba is demonstrated. Hypopigmentation of the left antecubital fossa and the right leg are particularly prominent (C).

without preceding inflammation, pain, or any associated pruritus.[37,38] Progressive macular hypomelanosis is caused by *Propionibacterium acnes*, which can produce a depigmenting agent.[37,38] The fact that antimicrobials work better than anti-inflammatory drugs in achieving clinical resolution of PMH is supportive of this theory.[37-39] Progressive macular hypomelanosis can be distinguished from CPA with Wood lamp examination, which shows red fluorescence within lesional skin.[38] Histologic analysis reveals decreased epidermal melanin and abnormal melanosome distribution.[40]

Pigmenting PA (PPA) and CPA both typically occur on the face and manifest as hypopigmented macules with scaling. However, PPA appears as bluish macules with substantial dermal melanin deposition within a hypopigmented scaly region similar to CPA lesions. Up to 65% of patients with PPA also have a superficial fungal infection; many also have concurrent CPA.[22,41]

Differential Diagnosis

Conditions associated with postinflammatory hypopigmentation usually have a history of dermatitis or other rash preceding the hypopigmented macules[33,42] and include contact dermatitis, seborrheic dermatitis, and psoriasis.[38] Pityriasis alba sometimes presents with a distribution similar to psoriasis,[4,20] though the scales are flatter and much smoother and the Auspitz sign cannot be elicited in PA.[4]

Vitiligo typically is found in the perioral and periocular areas and does not present with scaling.[43-45] Unlike PA, the hypopigmented macules of vitiligo actually are depigmented with total loss of melanocytes. This finding can be easily demonstrated with a Wood lamp examination; vitiligo appears chalk white, even in individuals with a lighter complexion.[43-45]

Tinea versicolor (TV) needs to be considered in the differential diagnosis of PA.[7,46-48] Pityriasis versicolor alba is a well-known variant of TV.[48,49] Most commonly seen on the trunk, groin, and axillae, it also may be present on the face.[48] Pityriasis alba and TV are the 2 most common conditions that manifest as hypopigmented macules with scaling. However, it can be easily distinguished from PA with a potassium hydroxide preparation, which will show spores and hyphae.[7] Wood lamp examination reveals a typical yellow fluorescence not seen in PA.[48] It also tends to occur in older children and can cause widespread skin infection, particularly in seborrheic areas.[49-51]

Two neurocutaneous disorders must be distinguished from PA. Hypomelanosis of Ito appears within the first year of life as hypopigmented patches with irregular borders. Epileptic seizures may occur, though there is no correlation between their severity and the presence of the patches.[52] The so-called Fitzpatrick patches (ash-leaf spots) of tuberous sclerosis primarily occur on the trunk and usually are present at birth.[7,53] The majority of patients also have central nervous system problems.[54] A thorough investigation of other systems should be undertaken for all patients.[53]

Nevus depigmentosus, also known as nevus achromicus, appears as a hypopigmented patch that is often present at birth[7] but usually is evident before 3 years of age.[54] Its unusual distribution patterns—dermatomal, in discrete patches, or with a splashed white paint appearance—is distinctive and quite stable.[7] The trunk, particularly the back and buttocks, is most commonly involved.[21,50,54]

Nevus anemicus, a congenital pediatric disorder,[55] is not a pigmentary disorder. Increased localized vascular tone results in a relatively pale patch of skin,[7] usually secondary to an aberrant sympathetic response to medications.[16,55] Diascopy leads to a transient redistribution of blood, blanching the

Differential Diagnosis of Pityriasis Alba (PA)

- Classic PA
- Extensive PA
- Progressive macular hypomelanosis
- Pigmenting PA
- Postinflammatory hypopigmentation
- Vitiligo
- Pityriasis versicolor alba (tinea versicolor)
- Hypomelanosis of Ito
- Tuberous sclerosis
- Nevus depigmentosus
- Nevus anemicus
- Mycosis fungoides
- Nummular eczema
- Leprosy
- Pharmacologic nevi (ie, benzoyl peroxide, topical or intralesional steroids, retinoic acid)
- Hypopigmentation secondary to procedures (ie, chemical peels, dermabrasion)

normal skin while the pale areas of nevus anemicus are reddened.[7,55]

Mycosis fungoides, a condition seen mostly in individuals of darker complexion, rarely manifests as hypopigmented macules but may be confused with PA when it does.[56-58] However, the presence of nonscaly lesions on the trunk and extremities as well as excessive numbers of epidermal T lymphocytes are diagnostic.[56]

Nummular eczema; leprosy; and iatrogenic hypopigmentation from the administration of benzoyl peroxide, topical or intralesional steroids, retinoic acid, chemical peels, and dermabrasion also are in the differential diagnosis.[1,59] A clinical diagnosis is sufficient in most cases, making biopsies generally unnecessary.[5] The Table provides a summary of the differential.

Prognosis

Pityriasis alba may last anywhere from months to years and lesions can present either in the same stage or at different stages.[32] It typically has a long-term course, tends to relapse, and may pose a notable aesthetic concern for patients.[60] However, it usually resolves without treatment, though there may be some recurrence at the initial location. Pityriasis alba may be more prominently visible in dry weather, after tanning, and in patients with atopic dermatitis due to a prolonged course.[5,10] The disorder appears to be limited to the pediatric population.

Management

Classic PA is namely treated with lubricants and emollients.[3,6,17] Topical corticosteroids, such as hydrocortisone acetate 1% or desonide, have had some limited success.[1] The use of diiodohydroxyquin, hydrocortisone, and coal tar may yield acceptable results.[32] Only mild nonhalogenated topical steroids should be used for facial lesions in children.[5,61] Topical tretinoin also may be helpful.[5,10] Nonfacial PA can be treated with stronger corticosteroids, such as hydrocortisone valerate or alclomethasone diproprionate.[20] Pityriasis alba in patients with poor socioeconomic backgrounds may be alleviated by improvements in living standards and education on proper skin care. Lubricants and emollients also are useful in EPA, but topical corticosteroid therapy is ineffective. However, EPA may resolve with psoralen plus UVA therapy.[34] Both EPA and PMH seem to resolve with UVA therapy, which supports the idea that they are the same disorder.

Given their similarities, treatments initially geared toward atopic dermatitis have been tested on PA patients. A study on the use of pimecrolimus cream 1% in atopic dermatitis patients showed promising results, such as long-term control of the disease, longer disease-free periods, and decreased need for continuous treatment.[62] A study of 10 patients with Fitzpatrick skin types IV and V using pimecrolimus cream 1% showed clinical improvement within 3 weeks and near complete resolution of the lesion at the end of the 12-week treatment period. The cosmetic results also were well-accepted by patients, as there was no atrophy or residual odors.[63] An increased risk for local viral infections, such as molluscum contagiosum, has been reported.[64] A randomized controlled clinical trial of tacrolimus ointment 0.1% combined with a standard moisturizer with sun protection factor 20 has shown remarkable results.[60] Thus, calcineurin inhibitors may be preferable for facial PA and for patients with an atopic background.

There is no single preventative measure for this disorder. However, good skin care is always beneficial. The regular application of sun protection and the use of mild cleansers are excellent supplemental skin care methods for patients with PA, though they will not clear PA lesions.[1,12,20,63]

Conclusion

Pityriasis alba is a common pediatric disorder of hypopigmentation that is generally self-limited. Current risk factors include atopy, xerosis, and mineral deficiencies. The pathogenic mechanism remains elusive. Although there is no definitive treatment, management should be tailored to the different clinical variants of the disorder. Given the possibility of psychologic distress secondary to the cosmetic appearance of the disorder, PA should be treated at the patient's request.

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