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Unique clinical presentations of pityriasis rosea: aphthous ulcers, vesicles and inverse distribution of lesions

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Abstract

Pityriasis Rosea (PR) is a common skin disorder encountered in daily practice. Although its etiology has not been established, there has been widespread research into possibilities. The lack of its characteristic manifestations, specifically the herald patch and truncal involvement, can lead to pitfalls in diagnosis. Whereas other conditions in the differential diagnosis should be considered, PR can at times also manifest in an atypical manner. We wish to illustrate three cases of PR including those that presented with aphthous ulcers, vesicles, and an inverse pattern.

Keywords: pityriasis rosea; inverse pityriasis; herpetiform; aphthous; vesicular pityriasis

Introduction

Pityriasis Rosea is an acute, self-limiting, inflammatory exanthem that is asymptomatic but can be mildly pruritic. It consists of salmon-colored macular and papular lesions with scales that fold over when stretched, known as the 'hanging curtain sign' [1]. PR prototypically manifests as an initial 2-6cm solitary erythematous plaque situated on the trunk, known as the herald patch. This is followed days to weeks later by secondary eruptions of smaller lesions along the lines of cleavage on the trunk, known as the 'Christmas-tree' distribution [2]. PR targets the trunk and extremities while sparing the palms, soles, and scalp [1,3]. It has a slight female predilection and favors the 10-35 years age group with a peak in adolescence [3-5]. There is no racial predilection for the disease [5]. Whereas the hallmark of PR is the

primary herald patch, the lack thereof can lead to an incorrect diagnosis. We wish to highlight some atypical presentations of PR with cases that involve aphthous ulcers, vesicles, and inverse patterns.

Discussion

Possible Etiologies

Although no etiology has been confirmed for PR, there has been widespread research conducted of possible causes. In particular, HHV 6 and 7 have been extensively studied as precipitating factors. HHV 6 and 7 are commonly acquired in childhood with seroprevalence in the general population being 80-90% [6]. These viruses remain latent and hidden from the immune system, reactivating in adult life or during states of immunoincompetence. Broccolo et al. examined this relationship in 2005 and detected HHV 6 and 7 DNA in 17% and 39%, respectively, of PR plasmas [6]. Broccolo et al. also found that HHV 7 was associated with a higher peripheral blood mononuclear cell (PBMC) load than controls [6]. In 2009, Kirac et al. studied serological parameters along with blood and tissue samples of PR patients, finding that these patients showed higher levels of DNA positivity in their blood for HHV 6 and 7 with HHV 7 being more predominant in tissue samples [7]. Although these and other studies have found a correlation between HHV 6 and 7 [8], a number of others have not [9,10,11].

In addition to HHV 6 and 7, HHV 8 has been studied as a possible etiology. In 2006, Chuh et al. tested patients with PR for HHV 8 with PCR and anti-IgG along with anti-IgM antibodies with indirect fluorescence [12].

Chuh et al. reported that PCR was negative in all patients and that although none had positive anti-IgM antibodies, only 4 had positive anti-IgG with no significant rise in titers; their conclusion was that PR is not associated with HHV-8 [12]. However, a later study in 2009 by Prantsidis et al. found that 7 of the 34 patients with acute, typical PR tested positive with DNA sequencing for HHV-8 genome [13]. Along with the aforementioned types of HHV, CMV, parvovirus B-19, and EBV have been examined as possible etiologies with no relationship being established [14,15]. An exception is a study by Bonafé et al. from 1982 finding that 42% of their patients with PR had antibodies against EBV early antigen, versus the controls [16].

Along with the viral etiology, a number of drugs have been reported to cause PR-like eruptions, such as adalimumab [17], bupropion [18], captopril [17], etanercept [19], imatinib [20,21], isotretinoin [22], lamotrigine [23], and metronidazole [24]. Whereas the clinical manifestations are similar to idiopathic PR, drug-induced PR lesions are itchy, diffuse, and confluent [25]. The herald patch and Christmas tree distribution are usually lacking as well [25,26]. Oral lesions and post-inflammatory hyperpigmentation are also associated with drug-induced eruptions [26]. The drug-induced lesions also present with increased eosinophils in the blood and skin infiltrate [27]. Thus, whereas no cause has been established to date, the literature contains a variety of potential factors that can lead to PR.

Diagnosis

The diagnosis of PR is clinical and done so by a careful history and physical exam [28]. The questions to ask include when the lesion first erupted, what medications the patient has been using, and if there are any signs of prodromal symptoms such as an upper respiratory infection [28]. Chuh et al. also proposed a set of diagnostic criteria that include discrete circular or oval lesions, scaling on most lesions, and collarette scale with central clearance of most lesions [29]. Thus, whereas the typical cases of PR are relatively straightforward to diagnose, other conditions should be considered. In atypical cases of PR, secondary syphilis is the most important to rule out. Nummular eczema should also be considered. Pityriasis lichenoides chronica (PLC) shares a similar

distribution to PR but lacks the herald patch and lasts for months to years before regressing [35]. Pityriasis rosea can also be mistaken for guttate psoriasis, tinea versicolor, and lichenoid drug eruption.

Atypical Presentations: PR with Aphthous Ulcers

Lesions of the oral mucosa associated with the typical oval macules with a collarette of scale may be more common than presented in the literature [32,36]. Although some state that the aphthae could be a separate entity from PR, the lesions tend to erupt and resolve at the same time as the PR lesions, supporting their single etiology when co-occurring [33]. The aphthae, appearing as shallow ulcers with a red rim in the buccal mucosa or soft palate, can be sore or asymptomatic [31,33-35].

Guequierre and Wright describe 5 types of oral lesions they have encountered including erythematous macules, erythematous macules with desquamation, erythematous annular lesions with raised borders, annular punctate hemorrhagic lesions, and a large erythematous plaque across the entire palate [36]. Costello observed a young boy with typical manifestations of PR along with a superficial, erythematous lesion on the mucosa, and a rectal temperature of 101.2oF [37]. Rosenbaum reported a man presenting with eruptions on the trunk and inguinal area with hemorrhagic lesions on the soft palate that increased in size up to 7mm and eventually resolved a few days after the cutaneous lesions [38]. Kestel described an 18-year-old man with biopsy-proven PR and ulcerative and hemorrhage lesions on the buccal mucosa [31]. Kay encountered a woman with PR and shallow ulcerations with an erythematous margin [39]. We encountered a woman in her 20s with classic PR lesions of the trunk and herpetiform aphthae of the soft palate (**Figure 1**).

Atypical Presentations: PR with Vesicles

Vesicles in PR are commonly located on the palms and soles, resembling dishydrosis, or on the face mimicking varicella [40, 41]. They are not associated with any prodromal symptoms, such as fever, malaise, or headache [40]. Vesicles in PR more frequently present in children and infants [31,35]

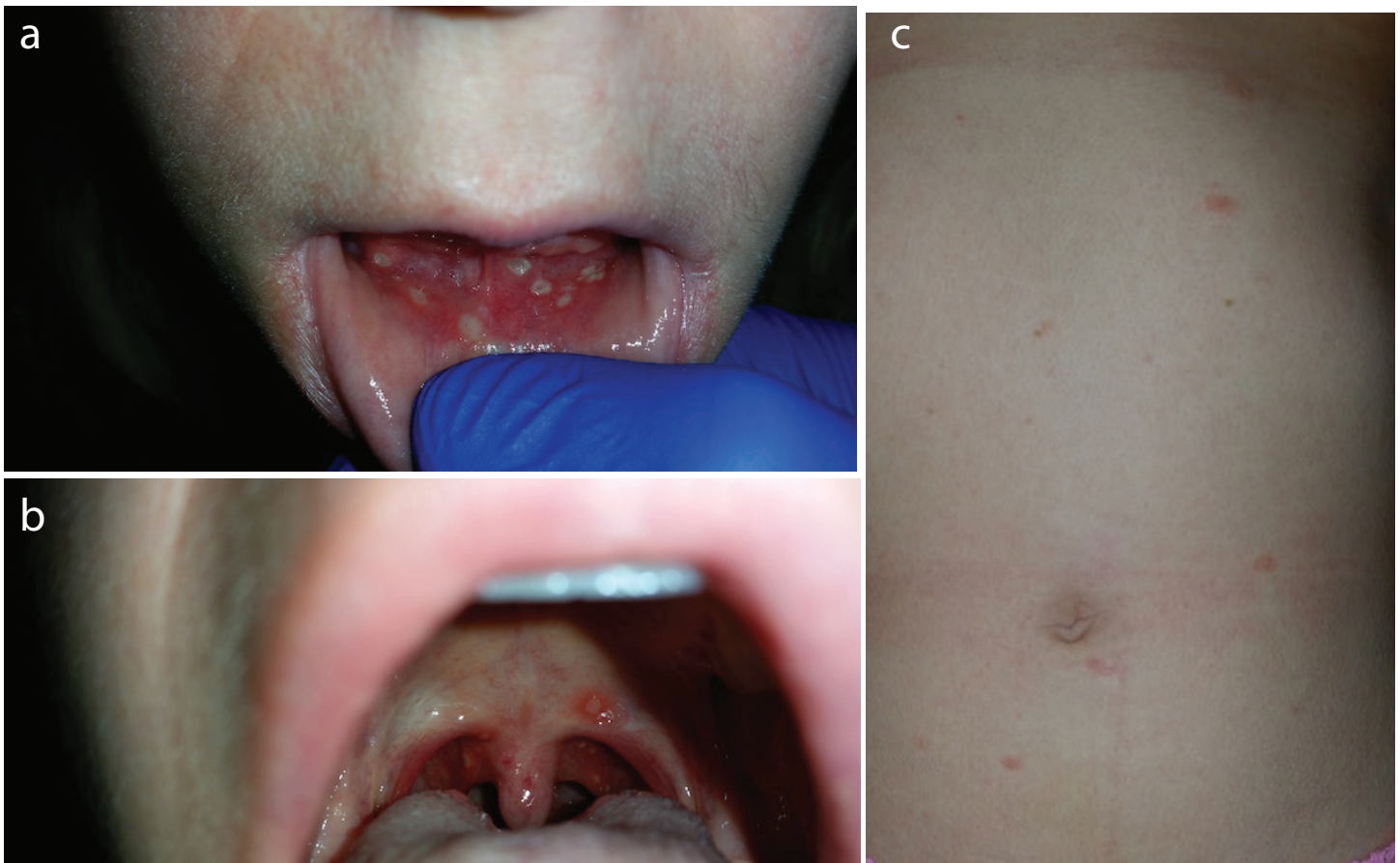


Figure 1 a) Herpetiform aphthous ulcers on the buccal mucosa. b) Herpetiform aphthous ulcers of soft palate. c) Typical pityriasis rosea rash on trunk of same patient.

Garcia et al. encountered two cases of young men with vesicular eruptions under 9mm involving their feet and ankles followed by a progression to PR [42]. Whereas Garcia notes that this was not a typical case of vesicular PR with lesions appearing on the trunk, both the vesicles and papulosquamous lesions resolved together [42]. Spiller et al. reported a 30-year-old woman with a herald patch of ten days and vesicles on bilateral palms [43]. Anderson reported a case of a man that presented with an oval, scaly, pruritic truncal rash with superimposed vesicles and bullae [44]. Balci et al. described the case of a 20-year-old woman with vesicles on the erythematous macules and plaques [45]. Finally, Bari et al. reported a case of a 7-year-old girl with vesicles and bullae on purpura located on the buttocks and extremities that resolved spontaneously within 5 weeks [4]. We treated a teenager with vesicles of the hands and feet (including palms and soles) and typical PR lesions of the trunk (Figure 2).

Atypical Presentations: Inverse Pityriasis Rosea

With inverse PR, the disease affects areas that are normally spared [31]. Most notably, inverse PR targets the face, extremities, and flexural areas while sparing the trunk [41,46,47]. Children and adolescents are particularly susceptible to developing the atypical variant [5].

Amer et al. conducted an observational study in 50 African American children and found that 30% of the children had facial involvement whereas 8% had scalp involvement along with residual pigmentary changes [48]. In addition, Vano-Galvan et al. encountered a 12-year-old African American boy with a facial papular eruption that resolved after five weeks, also leaving residual hyperpigmentation [49]. Trager reported a case of a 2-year-old girl with a mild pruritic rash located on the axillae and pubic area with some lesions on the trunk leading to the diagnosis of inverse PR [5]. In addition, Ermertcan et al. reported a child lacking a herald patch but with a

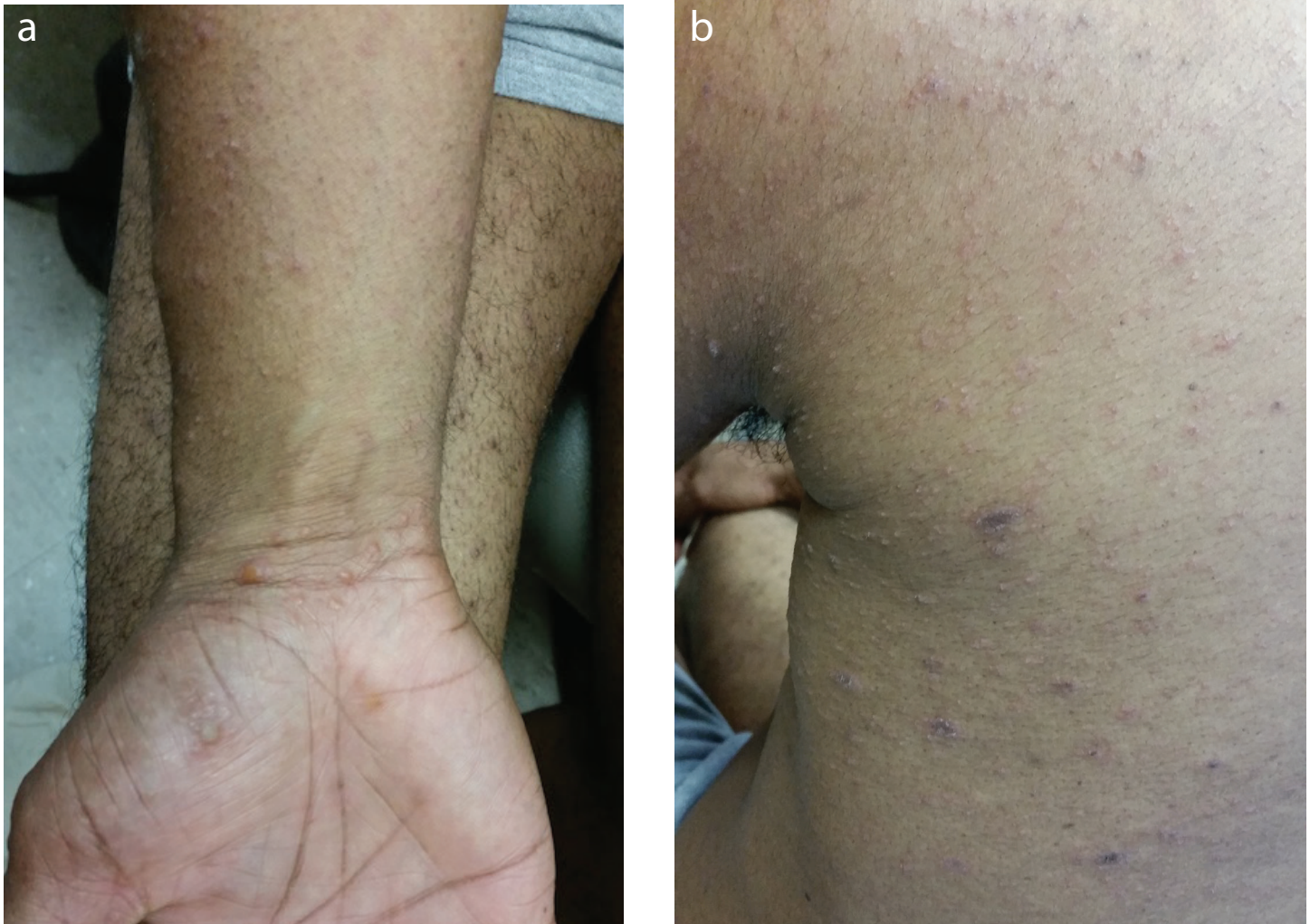


Figure 2. a) Vesicular lesions on the ventral forearm. b) Typical pityriasis rosea rash on trunk and axilla of same patient.

red-brown maculopapular eruption concentrated on the flexural areas that was treated symptomatically with antihistamines, corticosteroid cream, and emollients. The eruption eventually regressed in two months [50]. We treated a teenager with lesions of classical PR that manifested confluent red plaques of his antecubital fossae with characteristic collarettes of scale (**Figure 3**).

Therapy

Since PR is a self-limited disease, aggressive treatment is usually unnecessary with reassurance alone being sufficient. The skin disorder tends to regress within 2-12 weeks. First line therapies for PR include topical corticosteroids, emollients, and oral antihistamines [17]. These treatments are often aimed at relieving pruritus. A 2009 Cochrane collaboration paper concluded, however, that no PR therapy had sufficient evidence for efficacy [51]. Common therapies cited in the literature as being helpful include emollients, topical antihistamine



Figure 3. Inverse pityriasis rosea on flexor surface of the arm.

topical corticosteroids, ultraviolet light (in the form of UVB, UVA1, or sunlight), systemic antihistamines, oral erythromycin, and systemic antiviral agents [51]. We typically use topical steroids for itch and valacyclovir 1g by mouth three times daily for 10 days with good success.

Conclusion

Thus although PR is a common skin disorder, this classical eruption may be accompanied by atypical manifestations, including acral vesicles, an inverse distribution, and oral aphthae. The presence of these atypical features should prompt consideration of other conditions associated with the respective lesions but ultimately should not deter the physician from considering pityriasis rosea as the leading diagnosis.

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