Pityriasis rubra pilaris secondary to viral infection

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ABSTRACT

Introduction: Pityriasis rubra pilaris is a rare, chronic dermatosis presenting with erythema and papular eruptions as well as characteristic perifollicular hyperkeratosis and 'islands of sparing' (normal appearing skin). In the literature, there are reports of cases where symptoms of infection preceded the onset of pityriasis rubra pilaris.

Objective: Presentation of the case of a 67-year-old patient with type I pityriasis rubra pilaris to highlight the possible association between pityriasis rubra pilaris with infectious agents.

Case report: A 67-year-old patient was referred to hospital because of rapidly progressive erythematous papular skin rash preceded by symptoms of an upper respiratory tract infection. On physical examination, attention was drawn to perifollicular papules covered with cap-like scales as well as confluent erythema and features of hyperkeratosis on the hands and feet. Based on the clinical presentation and histopathological findings, type I pityriasis rubra pilaris was diagnosed and treatment with acitretin was initiated.

Conclusions: Infectious agents have the potential to be a trigger for pityriasis rubra pilaris. Acitretin continues to demonstrate efficacy in the treatment of pityriasis rubra pilaris.

Key words: pityriasis rubra pilaris, infections, pathophysiology, treatment.

INTRODUCTION

Pityriasis rubra pilaris (PRP) is a rare chronic papular dermatosis of incompletely understood aetiology. Although the disease is considered idiopathic in most patients, there are many documented cases in the literature in which the onset of PRP was preceded by symptoms of viral or bacterial infections [1].

OBJECTIVE

The aims of this case report are to present the case of a patient hospitalised in the Clinical Department of Dermatology (OKD) of the University Hospital in Krakow, who developed symptoms of PRP after a viral infection, and provide insights into the nature of the disease.

CASE REPORT

A 67-year-old male patient, a retired entrepreneur, was admitted to the OKD for the diagnosis and treatment of rapidly progressive erythematous papular skin rash. The first skin lesions, presenting as erythema on the face and palms, appeared approximately 2 weeks prior to hospitalisation. Subsequently, ery-

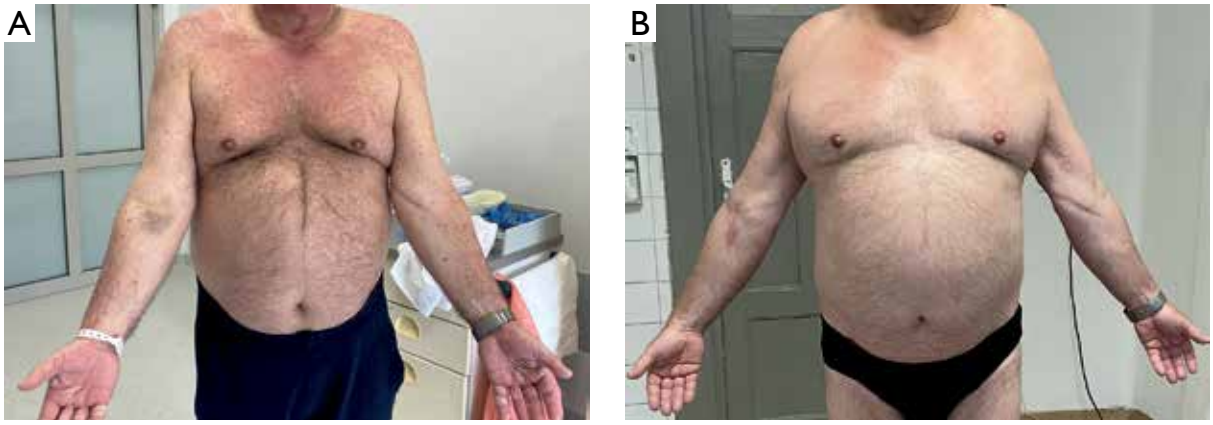

Figure 1. A – Confluent erythematous papular lesions with 'islands of sparing' on the trunk. Erythema and hyperkeratosis on palmar skin. **B** – Clinical improvement after initiation of acitretin treatment

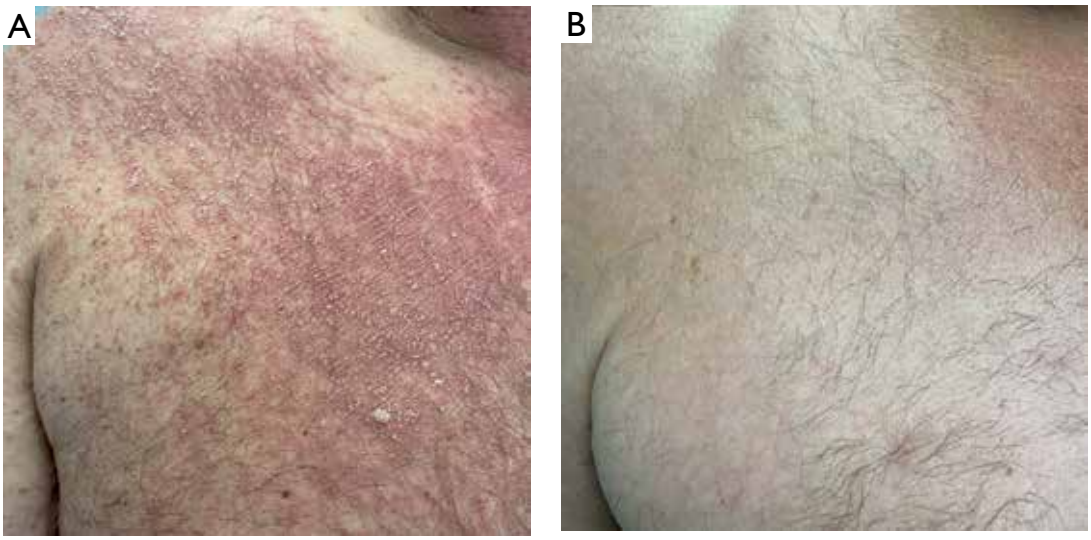

Figure 2. A – Perifollicular erythematous papules covered with cap-like scales on the chest. **B** – Clinical improvement after initiation of acitretin treatment

thematous papular eruptions spread symmetrically over the scalp, neck, chest (particularly in the upper region), back, and upper limbs. The emergence of the lesions was accompanied by a mild sensation of skin itching and burning. At the local dermatology outpatient clinic the patient was prescribed anti-inflammatory treatment, but his condition failed to improve. Upon admission to the hospital, physical examination revealed confluent facial erythema with a glossy surface and bran-like scaling. Other findings included erythematous papular lesions with a tendency to evolve into erythroderma, located on the scalp, neck, upper chest, back, and on the extensor surfaces of the forearms, thighs, and lower legs (fig. 1 A). It was noted that the lesions had a predilection for sun-exposed areas (photodistribution) and that certain patches of skin ('islands') remained unaffected by the disease. The papular eruptions had a perifollicular distribution and were covered with cap-like scales (fig. 2 A). On the extensor surface of the knees and elbows, the eruptions coalesced into hyperkeratotic plaques. In addition, erythematous keratotic lesions with bran-like scale were present on the palmar surface of the hands and soles of the feet (figs. 3 A and 4 A). Pathologically affected skin exhibited a subtle orange tinge. On clinical examination, there were no significant deviations in the patient's baseline parameters.

The onset of skin lesions was preceded by an upper respiratory tract infection treated with paracetamol, pseudoephedrine, dextromethorphan, and oseltamivir. Oseltamivir was prescribed based on clinical symptoms, without confirmation of influenza diagnosis by other diagnostic modalities.

The patient had no family history of chronic skin diseases, autoimmune or allergic conditions.

On admission to hospital, a panel of laboratory tests was performed (peripheral blood count, C-reactive protein, general urine analysis, lipid profile, cre-

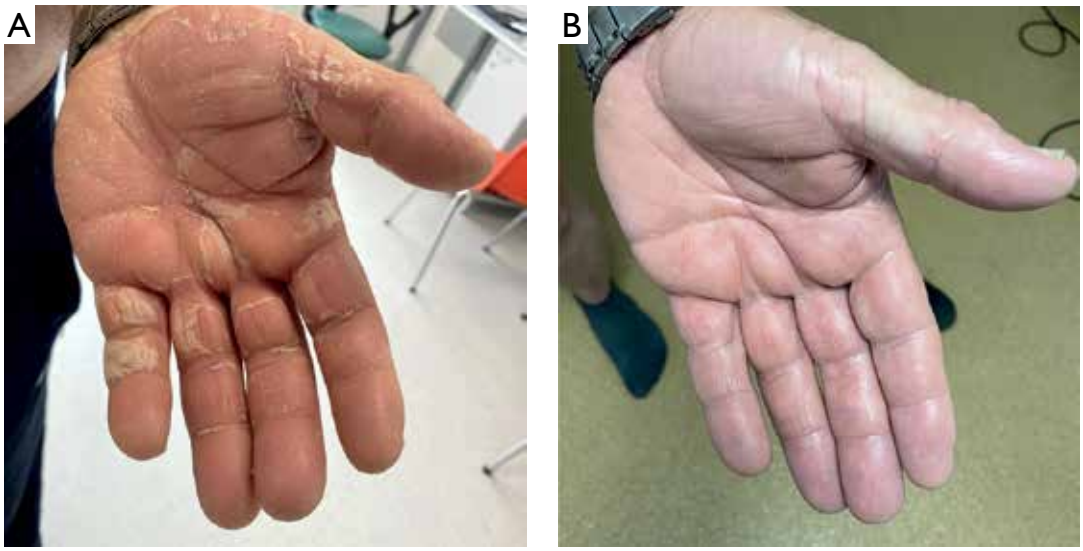

Figure 3. **A** – Erythema with prominent hyperkeratosis on the palm. **B** – Clinical improvement after initiation of acitretin treatment

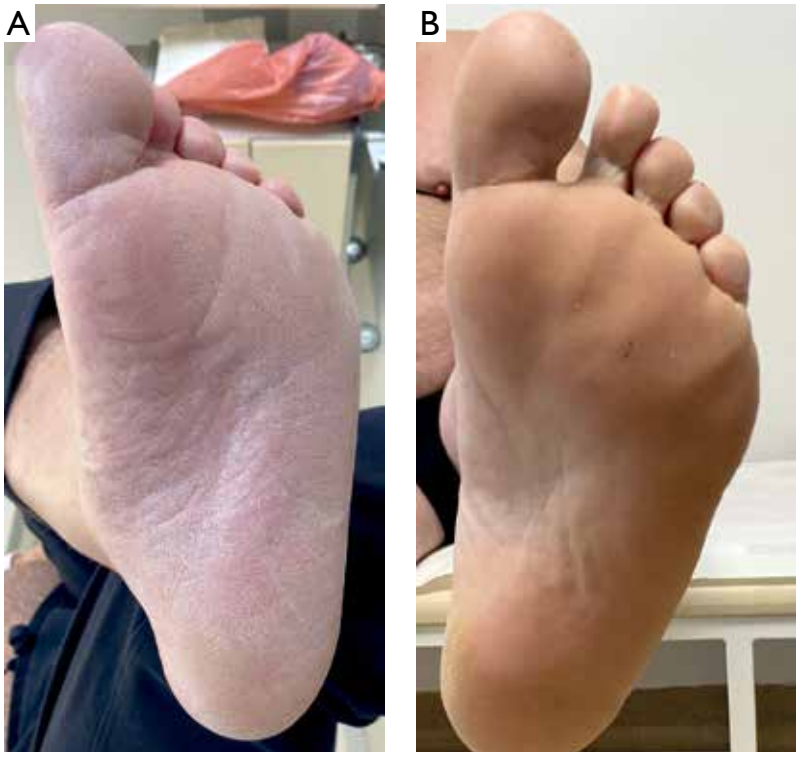

Figure 4. **A** – Erythema with prominent hyperkeratosis on the sole. **B** – Clinical improvement after initiation of acitretin treatment

atinine, urea, sodium, potassium, liver aminotransferases, TSH, fasting glucose, hepatitis B and C tests, HIV test). The only deviation from normal ranges was an elevated fasting glucose level.

Immunological tests were performed, including the determination of antinuclear antibodies by Indirect Immunofluorescence (IIF) and immunoblot assay (ANA Profile 3). The IIF test revealed antinuclear antibodies with granular fluorescence pattern at a titre of 1 : 320 and antibodies with a midbody pattern at a titre of 1 : 640, but the immunoblot assay did not reveal any specific autoantibodies.

Based on the clinical presentation and patient's history, a suspicion of PRP was raised. To verify the diagnosis, a skin specimen containing an affected hair follicle was obtained from the patient's chest.

Skin lesions located on the trunk and limbs were treated using an ointment with clobetasol propionate and 5% urea, followed by a lactic acid formulation. The facial lesions were treated with eucerin cream and methylprednisolone aceponate ointment.

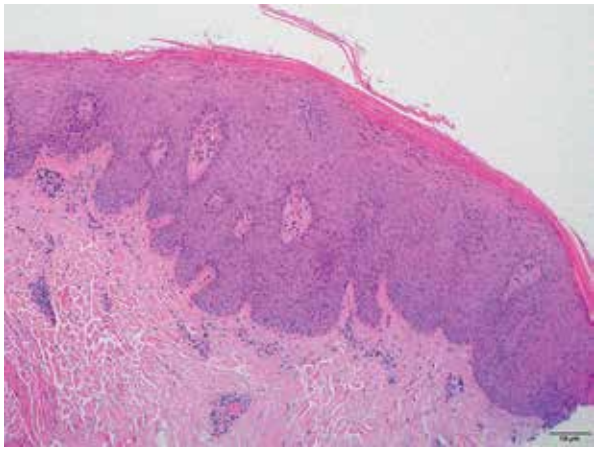

Figure 5. Histopathological image. Epidermis covered with alternating para- and orthokeratosis. Acanthosis with narrow dermal papillae. Mild perivascular inflammatory infiltrate in the dermis (H + E; 20×)

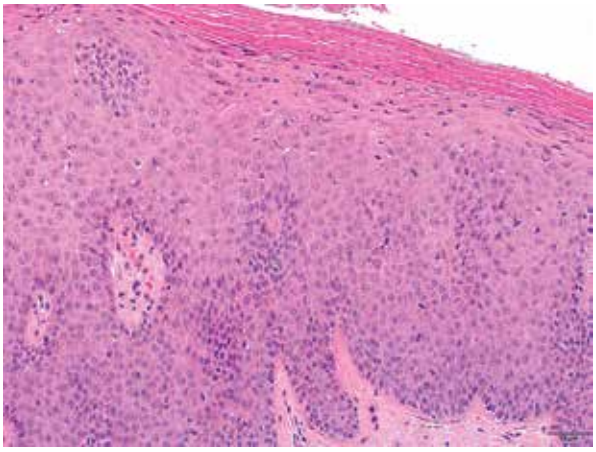

Figure 6. Histopathological image. "Checkerboard" pattern of the cornified layer in the epidermis (H + E; 40×)

Following the biopsy result confirming the diagnosis (figs. 5 and 6), the decision was made to initiate treatment with acitretin at a dose of 35 mg/day (i.e. 0.33 mg/kg/day for the patient's weight of 105 kg), resulting in a gradual marked improvement. The lesions located on the face and chest resolved completely without leaving any skin discolouration (fig. 1 B). Erythematous papular lesions on the back and upper limbs regressed partially. The severity of erythema decreased, the papules flattened, and the scaling resolved (fig. 2 B). Moderate erythema persisted solely on the posterior surface of the patient's thighs. Hyperkeratosis on the palms and soles resolved (figs. 3 B and 4 B). Since the treatment was effective and well-tolerated, acitretin therapy was continued with a gradual dose reduction. The patient remains under dermatological follow-up.

DISCUSSION

PRP is a rare inflammatory papular dermatosis characterised by prominent perifollicular keratosis. The first patient with PRP reported in the medical literature is believed to be James Shooter, who was admitted to St Bartholomew's Hospital in London in 1828. His condition was misdiagnosed by Dr. Claudius Tarral as generalised psoriasis vulgaris. Hyperkeratotic papules with central hair involvement on the skin of the dorsal surface of the proximal and middle phalanges, which were described by the physician, are more consistent with a diagnosis of PRP. The patient was treated with vesicants (cantharidin patch). However, in addition to being ineffective, the therapy caused episodes of heavy bleeding [2]. The first person to note that the condition was clinically distinct from psoriasis, who coined the term "pityriasis pilaris", was the French dermatologist Marie-Guillaume-Alphonse Devergie in 1856 [3]. In 1889, Ernest Besnier established the definitive name of the condition by appending the adjective "rubra". In a comprehensive, richly illustrated study totalling 120 pages, he described a series of nine patient cases [4].

PRP is classified as a rare dermatosis (estimated prevalence: 2.5/1,000,000 population) [5]. The disease is believed to affect men and women equally, as supported by the findings reported by Piamphongsant and Akaraphant [6]. In their 2020 study, Halper *et al*. examined the PRP patient population affiliated with a dedicated Facebook group and noted a small predominance of women (54.5%) [7]. PRP is characterised by a bimodal distribution, with peaks in incidence occurring during the first decade of life in children and between the fifth and seventh decades in adults [8, 9].

The aetiopathogenesis of PRP is not yet fully understood. Initially, based on observations of similar symptoms in Chinese soldiers and Ugandan prisoners exposed to vitamin A deficiency, it was hypothesised that the deficit of this vitamin or its carrier (retinol-binding protein – RBP) was the key factor in the pathophysiology of PRP [10–12]. However, subsequent studies reporting normal vitamin A and RBP levels in PRP patients, along with the failure of therapy with vitamin A derivatives in a proportion of PRP cases, overturned this hypothesis [13–15]. Current research investigating the aetiopathogenesis of PRP focuses on mutations in the *CARD14* (caspase recruitment domain family member 14) gene located on chromosome 17 (17q25) and CARD14-induced activation of the IL23-IL17A cytokine axis. Mutations of the *CARD14* gene have been identified in the genotype of individuals with the familial variant of PRP and in some patients without a family history of

PRP [16, 17]. The mutations have a proven link with heightened NF-κB (nuclear factor κB) expression in keratinocytes, triggering the activation of the IL23-IL17A cytokine axis and resulting in overexpression of the chemokine CCL20 and interleukin IL-17C [18-20]. NF-κB is a protein complex acting as a transcription factor, which plays a pivotal role in the inflammatory process observed in both PRP and psoriasis. NF-κB-dependent intracellular signalling pathways are implicated in the production of inflammatory cytokines by Th17 lymphocytes, dendritic cells, and keratinocytes themselves [21]. Interestingly, NF-κB is activated in response to viral and bacterial antigens as well as by proteins of some viruses (e.g. the human immunodeficiency virus) that interact directly with elements of its intracellular signalling pathway [22, 23]. NF-κB-mediated activation of the IL23-IL17A axis was also confirmed in patients without CARD14 mutations [20]. It is important to highlight that mutations in the CARD14 gene and the activation of the IL23-IL17A axis were also identified in patients diagnosed with psoriasis [24]. In 2021, Shao *et al*. argued that overexpression of the phospholipases PLA2G2F, PLA2G4D, and PLA2G4E (phospholipase A2 group IIF, IVD, IVE) was a crucial element in the aetiopathogenesis of both diseases [25].

The infectious agent most extensively linked with PRP is the human immunodeficiency virus. PRP can be an early sign of HIV infection, and in some patients antiretroviral therapy causes the skin lesions to subside [26–29]. However, the exact pathogenetic mechanism linking the two conditions remains unknown. Since other dermatoses with a follicular component (including hidradenitis suppurativa, acne conglobata, or lichen spinosus) are also characterised by a more common coexistence with AIDS, hair matrix cell infection or HIV-induced disruption of follicular keratinisation have been proposed as potential underlying causes of this link [26, 30, 31].

Following frequent reports of PRP development after bacterial and viral infections, the possibility of PRP being triggered by immune system stimulation is also considered [1]. There are literature reports of patients experiencing a remission of PRP symptoms after treatment of the accompanying infection [32, 33]. Interestingly, patients with chronic periodontitis were shown to have increased lymphocyte differentiation towards Th17 cells, which, in addition to their protective function against infectious agents, are also an important element in the pathogenesis of PRP and other inflammatory skin diseases [34].

There are literature reports describing the association between the onset of PRP and treatment with tyrosine kinase inhibitors, topical TLR agonists, phosphatidylinositol-3-kinase inhibitors, antivirals, biologics, PD-1 inhibitors, VEGF inhibitors, statins, insulins, angiotensin-converting enzyme inhibitors, and COVID-19 vaccines [35, 36]. There have also been sporadic reports linking PRP with autoimmune diseases and cancer [1].

PRP typically presents with fine erythematous papules with severe keratosis, in a perifollicular distribution. The lesions tend to coalesce into plaques over a large area, leaving 'islands' of unchanged skin ('suberythroderma' with nappes claires). Occasionally, the plaques are covered with fine scale. Affected skin areas may have an orange tinge. Initially, the lesions typically manifest on the scalp and face, resembling seborrhoeic dermatitis, before gradually spreading to other body regions, with an occasional propensity for erythroderma. These changes are often accompanied by palmoplantar keratoderma [37]. Clinically, PRP is divided into six types (table 1): five proposed by Griffith in 1980 and the sixth added by Miralles *et al*. in 1995 [8, 27].

Our patient was diagnosed with type I PRP, which is characterised by a good prognosis (in 80% of patients with this variant, symptoms resolve within three years) [38].

The histopathological findings of PRP are diagnostic and include [1, 39, 40]:

- alternating vertical and horizontal ortho-and parakeratosis ("checkerboard" pattern),
- focal or confluent hypergranulosis,
- irregular acanthosis with short narrow dermal papillae,
- epidermal thickening over dermal papillae,
- moderate lymphocytic infiltrates around the vessels of the superficial dermal plexus,
- follicular keratotic plugs with parakeratosis around the follicular openings,
- mitoses in the suprabasal layer.

Some patients present with signs of local acantholysis, eosinophilic infiltration or dense lymphocytic infiltration, resembling histopathologically the changes seen in lichen planus [41]. When performing a diagnostic biopsy in a patient with suspected PRP, the biopsy specimen should contain a hair follicle.

Dermoscopic examination of PRP lesions typically reveals yellow-orange areas surrounded by linear and dotted vessels accompanied by centrally located white keratotic plugs [42]. In contrast to lesions typically seen in psoriasis, dermoscopic evaluation of PRP eruptions significantly more commonly reveals an orange background, white keratotic plugs, linear vessels around the periphery of lesions, and scale conglomerates. Psoriatic lesions are significantly more frequently characterised by a pink background, dotted vessels, and white scale [43].

A number of conditions should be considered in the differential diagnosis of PRP, including seborrhoeic dermatitis, psoriasis, atopic dermatitis, cuta-

Table 1. Pityriasis rubra pilaris: clinical types [8, 26, 38, 39]

Types	Clinical characteristics
I. Classical generalised adult variant (most common type)	Characterised by typical clinical course and good prognosis. Skin lesions may be accompanied by ectropion, NA changes (longitudinal ridging, nail plate thickening and discolouration, subungual hyperkeratosis) and joint pain.
II. Atypical adult variant	Rare variant with a frequently chronic course. Body skin eruptions may resemble ichthyosis, and skin exfoliation secondary to palmoplantar keratoderma may have a 'roof-tile' appearance. In type II PRP, the lower limbs are the most frequently affected body area. There is no typical pattern of progression starting with the involvement of upper body parts and gradually moving downward. Skin lesions are often accompanied by hair loss.
III. Classic generalised juvenile variant	Affecting patients between the ages of 5 and 10. It is characterised by a typical course and a good prognosis, with lesions resolving within 1 year.
IV. Circumscribed juvenile variant	The most common variant in the paediatric population, occurring mainly in prepubescent children. Characterised by well-demarcated erythematous plaques with prominent follicular hyperkeratosis occupying mainly the extensor surface of the elbow and knee joints, and occasionally the dorsal surfaces of the hands and feet, as well as the buttocks. The prognosis tends to be poorer, and in many patients the disease persists over a span of many years.
V. Atypical juvenile variant	A very rare variant often associated with familial occurrence (inherited through an autosomal dominant pattern) and characterised by a chronic course. Typical skin changes include well-demarcated erythematous plaques on the skin of the cheeks and chin, typically with sparing of nasolabial folds, as well as ichthyosis-like changes on the skin of the trunk and limbs. Some patients develop scleroderma-like lesions on the skin of the hands and feet.
VI. HIV-associated variant	Frequently coexisting with other dermatoses with a follicular component, such as hidradenitis suppurativa, acne conglobata, or lichen spinosus.

NA – nail apparatus, PRP – pityriasis rubra pilaris, HIV – human immunodeficiency virus.

neous T-cell lymphoma, follicular lichen planus, hypereosinophilic syndrome, Wong's dermatomyositis, and graft-versus-host disease mimicking PRP [1, 44]. PRP lesions in patients who have been treated with vitamin A derivatives may occasionally resemble the eruptions characteristic of erythema gyratum repens [1]. In patients with type IV disease, the lesions need to be differentiated from lichen spinulosus, Darier's disease, pemphigus foliaceus, follicular keratosis, and epidermal nevus [1, 45].

Traditional first-line treatment of PRP was based on retinoids (first generation: isotretinoin and alitretinoin, and second generation: acitretin) [1, 46]. While some literature reports suggest that isotretinoin has a superior therapeutic efficacy compared to acitretin [46], based on the authors' personal experience, treatment with acitretin yields more favourable therapeutic outcomes. At present, biological therapies with inhibitors of interleukin-17 (such as ixekizumab, secukinumab, brodalumab), interleukin-23 (ustekinumab, guselkumab, risankizumab, tildrakizumab), and TNF-α are also used with considerable success [46–50]. In addition, there have been initial reports of upadacitinib as an effective therapeutic agent [51]. Another option is methotrexate treatment, though it appears to be less effective compared to retinoid therapy [46, 52]. A variety of responses to PRP phototherapy have been reported in the literature, including exacerbation, lack of efficacy, and reduction in symptoms severity [53–55]. Other reported therapies with lower efficacy include azathioprine, cyclosporin A, apremilast, mycophenolate mofetil, penicillin, dimethyl fumarate, extracorporeal photophoresis, and intravenous immunoglobulins [1, 46]. Topical glucocorticosteroid preparations prove effective for lesions covering a limited area of the skin [9]. Regular application of emollients and keratolytic agents on scale-covered skin is also recommended.

CONCLUSIONS

We report the case of a patient diagnosed with PRP, which was likely triggered by an infectious agent in the upper respiratory tract. Over the past few years, there have been significant advances in the understanding of the pathophysiology of the disease, resulting in the expansion of available PRP therapies. Retinoids continue to be a well-established and effective treatment modality.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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