International Journal of Research in Medical Sciences
Pérez-García MM et al. Int J Res Med Sci. 2025 Apr;13(4):1731-1737
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20250740

Review Article

Pityriasis rubra pilaris and HIV: a diagnostic challenge-case report and critical review of the literature

Mónica M. Pérez-García, Camila Ortuz-Lessa*, Sarahi Amador-Eleuterio, Arely L. Gallegos-Ramos

Department of Dermatology, Hospital General de México "Dr. Eduardo Liceaga", Mexico City, Mexico

Received: 18 February 2025
Accepted: 01 March 2025

***Correspondence:**
Dr. Camila Ortuz-Lessa,
E-mail: camilaortuz@gmail.com

ABSTRACT

Pityriasis rubra pilaris (PRP) is a rare cutaneous disorder characterized by hyperkeratotic follicular papules and palmoplantar keratoderma. Its clinical presentation ranges from mild to severe forms, including erythroderma. There are six types of PRP. Type VI is associated with HIV, presenting with a more severe and challenging course. This variant can occur in HIV-positive patients regardless of their CD4 T cell count. This article provides a detailed review of the pathogenesis, clinical characteristics, diagnosis, differential diagnosis and therapeutic options of PRP, with an emphasis on its HIV-associated form. An illustrative clinical case is presented, analyzing the underlying pathogenesis and treatments used. Furthermore, these findings are compared with those reported in the literature to provide a broader perspective on how this disease affects immunocompromised patients.

Keywords: Auto immune deficiency syndrome, Devergie's disease, HIV, Lichen ruber pilaris, Lichen acuminatus, Lichen ruber acuminatus, Type VI pityriasis rubra pilaris

INTRODUCTION

PRP was first described by Claudius Tarral in 1835 as a variant of psoriasis.[1-3] It is an uncommon inflammatory papulosquamous skin disorder characterized by hyperkeratotic follicular papules that coalesce into scaly, orange-red plaques accompanied by palmoplantar keratoderma.[4-8] In some cases, patients develop erythroderma, leaving islands of unaffected skin.[9] It can present either acquired or familial, in childhood or adulthood, with either generalized or localized involvement.[10] Diagnosis is established through the correlation of clinical and pathological findings.

The most cited classification scheme is that of Griffiths (1980) which divides the disease into five groups.[1,3,10,12] However, in 1995, Miralles added a sixth type associated with HIV.[4,11,13] Type I: Classic Adult, Type II: Atypical Adult, Type III: Classic Juvenile, Type IV: Circumscribed, Type V: Atypical Juvenile, Type VI: HIV-Associated.

Although PRP typically affects immunocompetent individuals, it has also been documented in patients with HIV. In these cases, it is characterized by typical lesions resembling acne conglobata, hidradenitis suppurativa and lichen spinulosus, which are distinctive of its HIV-associated form.[14,15] The relationship between the two conditions presents challenges in treatment due to immune alterations and the effects of antiretroviral therapy. This article presents a clinical case of HIV-associated PRP, comparing the findings with the literature to provide a deeper understanding of how this disease affects immunocompromised patients.

CLINICAL CASE

A 50-years-old male patient, diagnosed with HIV in 2023, is under antiretroviral treatment with a CD4+ count of 530. He presented with disseminated dermatosis on his trunk and upper and lower extremities, sparing the palms and soles. The lesion consisted of multiple erythematous-

squamous plaques with well-defined borders and thick scales. Based on the clinical suspicion of plaque psoriasis, treatment with topical calcipotriol/betamethasone, salicylic acid cream and UVB phototherapy was initiated, without improvement.

In the next follow-up visit, the dermatosis showed a tendency to generalization, with erythematous-squamous plaques having irregular borders, fine white-yellowish scales and islands of spared skin (Figure 1, 2). HIV-associated type VI Pityriasis rubra pilaris was suspected and an incisional biopsy confirmed the diagnosis, showing laminar orthokeratosis, "checkerboard" pattern parakeratosis, marked acanthosis, hypergranulosis, lymphocytic, inflammatory infiltrate and dilated capillaries in the dermis.

Treatment with isotretinoin 20 mg every 12 hours was started after ruling out Guselkumab due to a history of hepatitis B virus. The patient is currently showing significant clinical improvement, with minimal residual lesions.

EPIDEMIOLOGY

Clinical findings compatible with PRP were first recognized in the 19th century 10, but its incidence and prevalence are still not well documented. PRP can appear at any age, with two peaks of incidence: one at six or seven years old and the other between the fifth and sixth decades of life.[1,16,17] It affects all races and genders equally, although some reports suggest a slight male predominance.[1,5,10,17]

The incidence varies widely, being 1 in 500 new patients in Kenya, 1 in 5000 in the United Kingdom, 1 in 50,000 in India and between 1 in 3500 to 5000 in the United States.[1,5,8,10] 18 HIV-associated PRP commonly affects young men, both homosexual and heterosexual.[8,11]

ETIOPATHOGENESIS

The pathogenesis of PRP is not fully understood, but triggering factors such as infections, medications, neoplasms and autoimmunity have been identified.[1,19,20] The IL-23/TH17 axis plays a key role, as PRP responds favorably to the therapies targeting IL-17 and IL-23.[3,10]

Most cases of PRP are sporadic; however, the familial form has been associated with dominant autosomal mutations in the CARD14 gene.[3,8,19] This gene is part of the PSORS2 locus, which is involved in familial pustular psoriasis.[5,10] Gain-of-function mutations in CARD4, located on chromosome 17q25, lead to excessive activation of nuclear factor kappa B (NF-kB) in active B cells, regulating genes involved in immune and inflammatory responses.[1-3,20] Although these mutations have been primarily identified in familial cases, they have also been reported in individuals with sporadic PRP.[1,6]

Although some pathogenic mechanisms of psoriasis and PRP overlap, some studies have shown that the same dysregulated agents in the skin of PRP patients are also presents in psoriasis patients, supporting the success of biologic treatments in PRP.[5,10]

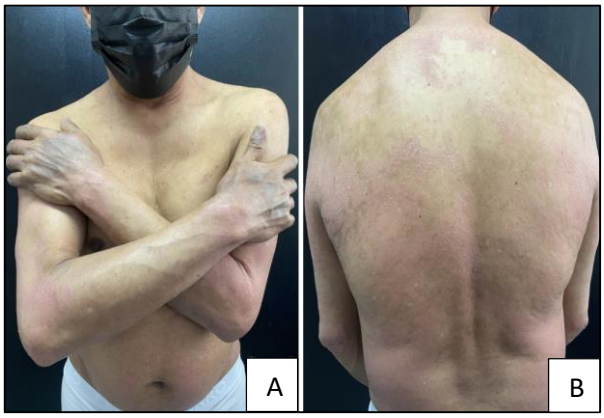

Figure 1 (A and B): Clinical images of patient with HIV-associated PRP (front and back view).

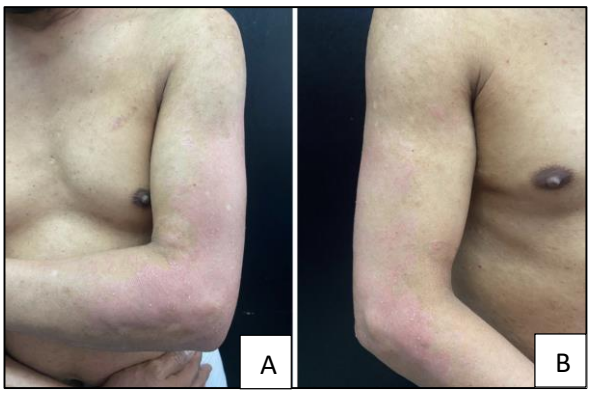

Figure 2 (A and B): Clinical images of patient with HIV-associated PRP (extremities).

The role of viral and bacterial infections has been described in the context of HIV infection.[6,9] It has been proposed that immune system alterations in these patients may influence the onset and severity of PRP.[22] HIV induces an immunosuppressive state, favoring abnormal proliferation of keratinocytes and skin inflammation. The inflammation and dysfunction in the skin barrier typical of PRP may be exacerbated by chronic HIV infection. This condition could be linked to an abnormal immune response to antigenic triggers, such as HIV itself, as well as follicular inflammation generated by the infection in the hair follicle region.[11,15,18]

Infections by Staphylococcus aureus and Streptococcus pyogenes have been linked to cases of juvenile PRP, with resolution of skin lesions observed following treatment with specific antibiotics.[1,6] This has led to the theory that the bacterial superantigens could trigger PRP, although no conclusive studies have been conducted to date.[18]

Additionally, the apparent therapeutic response to antibiotics could be mistaken for spontaneous resolution.

Drugs such a tyrosine kinase inhibitors (TKI) and phosphoinositide 3-kinase inhibitors (PI3K) have frequently been associated with the development of PRP.[3,10] In a systematic review of patients with drug-induced PRP, the interval between the initiation of treatment and the onset of PRP ranged from three days to five months.[23] Hypotheses include drug-induced changes in immune regulatory surveillance, epidermal growth and the production of pro-inflammatory cytokines.[10,23]

An underlying malignant neoplasm may also be a potential cause of PRP, suggesting that it could be a paraneoplastic eruption.[3,21] In fact, several dermatologic conditions have been associated with underlying malignant neoplasms and a review reported that malignant neoplasms were the second most frequent cause of PRP.[21] Although current studies do not specify the types of neoplasms involved, the hypothesis is that antigenic stimulation may be responsible, either by neoplastic cells or by the collapse of immunoprivileged sites, resulting in the release of previously sequestered autoantigens.[3] However, cutaneous manifestations in these cases tend to be atypical and the range of associated tumors is varied.[5]

There are case reports linking PRP with autoimmune diseases such as myasthenia gravis, autoimmune thyroiditis, celiac disease and vitiligo.[1] The association between these diseases and PRP may be due to shared T-cell dysregulation.[3]

Although the role of vitamin A deficiency and metabolic abnormalities is still unclear, serum vitamin A levels in patients with PRP are typically normal.[1] However, retinol-binding protein (RBP) levels were low in 11 PRP patients and their relatives.[18] High doses of vitamin A have not always led to PRP remission.

Recently, COVID-19 vaccination has been included among the potential triggering factors.[3,21] Although the induction mechanism is poorly understood, it is possible that the foreign epitopes of the vaccine induce a hyperactive immune response in predisposed individuals.

CLINICAL FEATURES

The clinical presentation of PRP varies considerably, ranging from mild and localized forms to severe forms that extensively affect the skin, including diffuse erythroderma.[5] Common symptoms include intense pruritus, skin tightness, burning, stinging and pain. In some cases, it may be self-limiting and asymptomatic.

The key characteristic features are hyperkeratotic follicular papules and palmoplantar keratodermia.[17,24] Almost all forms of PRP can evolve into erythroderma within two to three months, except for the juvenile circumscribed form.[25] Scaling is frequent, tends to be fine and pityriasiform on the upper body and thick and lamellar on the lower body.[19,25]

The hyperkeratotic follicular papules, 1 to 2 mm in diameter, present a central keratotic plug and may merge to form plaques of a reddish-orange color.[2,6,10,24] Although it has been proposed that the presence of these papules on the dorsal fingers is pathognomonic, this finding is often absent.[10] Palmoplantar keratodermia is characterized by diffuse, waxy, yellowish hyperkeratosis.[10]

TYPES OF PRP BASED ON AGE AND PRESENTATION

Classic PRP in adults

Presents with inflamed plaques, islands of sparing, pityriasiform scales and erythroderma. The involvement progresses in a cephalocaudal direction.[8,18] Ectropion, hair loss and nail abnormalities such as nail plate thickening, splinter hemorrhages, subungual hyperkeratosis or xanthonychia are observed.[1,3,18] This is the most common form in adults and tends to resolve spontaneously after several years.[10]

Atypical PRP in adults

Less frequent, characterized by ichthyosiform and thick, laminated scales on palms and soles.[3] It primarily affects the lower extremities and may be associated with alopecia.[1,15] Its course is more prolonged, lasting up to two decades.[18] Eczematous lesions can complicate the diagnosis.[8]

Classic PRP in childhood

Like the adult form, but with a lower incidence of erythroderma an ectropion.[3] It mainly presents between 5 and 10 years of age.[1,18] Previously considered self-limiting, recent studies suggest a variable course, with 72% of cases persisting for an average of 58 months.[3,16]

Circumscribed PRP in childhood

The most common pediatric variant, characterized by well-defined plaques with follicular plugs on the knees, elbows, hands, feet and buttocks.[1,18] Palmoplantar keratodermia, which can extend to the Achilles tendon, is common.[3,10] Its recurrent and remitting course is more frequent in prepubertal children.

Atypical PRP in childhood

An uncommon subtype, usually developing in early childhood and the most frequent type in familial PRP. It is characterized by facial involvement with erythematous plaques on the cheeks and chin, hyperkeratotic follicular papules and ichthyosiform or psoriasiform scales on the trunk and extremities.[1,18] It may present with distal onycholysis and onychogryphsis.[3] Its course is chronic.

PRP associated with HIV

Characterized by a low or normal CD4+ T-cell count.[10,22] Although it shares clinical findings with classic PRP, it is usually more severe and difficult to treat. An HIV-associated tetrad is observed, including PRP, acne conglobata, hidradenitis suppurativa and lichen spinulosus.[1,11,15] In its early stages, erythematous follicular papules progress to erythroderma, with areas of unaffected skin.[3,6] A characteristic finding is prominent follicular plugging with the formation of spicules.[9,14] This nodulocystic folliculitis pattern helps differentiate it.[14]

It is proposed that the underlying mechanisms involve abnormal immune response to HIV and the resulting follicular inflammation.[11,15,18] In these patients, deforming and atypical patterns are observed, such as peripheral corneal mucinosis, explosive cystic acne, lichen planus and nodulocystic lesions with occasional nail dystrophy.[15] Suppurative hidradenitis may be triggered due to chemotherapy- induced neutropenia.[15] It manifests as erythematous plaques in unusual areas, such as the face trunk or extremities.[6,8]

HIV-infected patients, especially those not receiving adequate treatment, are more susceptible to developing more aggressive forms, such as generalized or erythrodermic PRP.[5]

HISTOPATHOLOGY

In PRP, common histopathological findings include alternating orthokeratosis and parakeratosis, both vertically and horizontally, forming what is referred to as a "checkerboard" pattern.[1,3,6,16,17] Additionally, focal or confluent hypergranulosis, keratotic, follicular plugging, psoriasiform hyperplasia and a granular layer that may be normal or thickened are frequently observed.[8,10,19] Mitotic figures above the basal epidermal layer, acantholysis and a mild perivascular lymphocytic inflammatory infiltrate in the dermis are also seen.[4,16,18] Other additional findings include epidermal spongiosis and focal acantholytic dyskeratosis.[20]

DIAGNOSIS

The diagnosis is based on the identification of consistent clinical and histopathological features, considering the age of onset, progression of skin involvement symptoms and family or medication history, to rule out potential pharmacological inductions or hereditary causes.[10] Both psoriasis and PRP can initially present with erythematous, scaly plaques accompanied by pruritus.[7]

Although there are no specific laboratory tests or genetic markers for a definitive diagnosis, it is primarily based on clinical suspicion and dermatopathological correlation.[11] Skin biopsies, preferably taken from areas with scaly, follicular and hyperkeratotic papules, are essential for confirming the diagnosis.[10] It is recommended to take two 4 mm punch biopsies due to the histopathological changes that may be found.[10] Genetic testing for the CARD14 gene has been suggested for patients with PRP onset in the first year of life, children who persist with symptoms beyond three years or those with a family history of PRP.[5,10] Additionally, since PRP may be associated with HIV, it is recommended to screen adolescent and adult patients for possible HIV infection.[5,10,22]

Dermatoscopy has proven useful in differentiating PRP from psoriasis. It was observed that patients with PRP had follicular plugs and peripheral yellow-orange halos without visible vascular structures, while psoriasis displayed regularly distributed dotted vessels.[3,19] However, histopathological examination remains crucial for confirming the diagnosis. In patients with darker skin, PRP may be misdiagnosed due to its atypical presentation with follicular papules and brown-reddish erythema instead of the classic pink-orange hue, as well as unconventional distributions and early onset.[10,26]

DIFFERENTIAL DIAGNOSIS

Given that patients with HIV are more susceptible to opportunistic skin infections, it is crucial to perform a differential diagnosis. In its early stages, PRP can be confused with several common skin conditions in immunocompromised patients.

Seborrheic dermatitis

Although PRP is characterized by a cephalocaudal progression, like seborrheic dermatitis, the latter primarily affects the scalp, eyebrows and nasolabial folds with erythematous and scaly parches.[1,5] The absence of follicular hyperkertosis, palmoplantar keratodermia and differences in distribution help differentiate it from PRP.[5]

Erythrodermic psoriasis

Erythroderma in PRP can be confused with that of other etiologies, with psoriasis being the most common.[18] Other causes include atopic dermatitis, drug reactions and cutaneous T-cell lymphoma. Patient history, physical examination and skin biopsy are essential and if lymphoma is suspected, further studies such as flow cytometry or T-cell clonality tests should be performed.[10]

Chronic plaque psoriasis

Although both conditions present erythematous, inflamed plaques with micaceous scales, psoriasis commonly localizes on the elbows, knees, scalp, palms and soles and lacks the characteristic features seen in PRP.

Wong-type dermatomyositis

This rare disease shares clinical and histopathological features with PRP, such as follicular keratotic papules on the trunk and keratoderma of the soles.[5] Skin biopsy and

muscle enzyme testing are useful for differential diagnosis.[10,18]

Erythema gyratum repens

This paraneoplastic disorder, associated with lung carcinoma, presents with migrating skin eruptions in annular and arcuate plaques, often accompanied by palmoplantar keratoderma.[10]

PROGNOSIS

PRP is not associated with systemic involvement or a high mortality risk, but it does present significant morbidity and a considerable impact on patients' quality of life. While the disease can persist for years, some cases experience spontaneous resolution, although unpredictably. In these cases, the disease course typically begins with a severe, inflammatory and erythrodermic phase that persists for several months, followed by a slow and gradual improvement in the subsequent years.[27] Rapid resolution, within less than a year, is more common in children than in adults.[27]

The prognosis in HIV-positive patients is generally more severe, with an increased risk of complications such as cutaneous sepsis.[14,15,28] Additionally, HIV-associated PRP increases the risk of squamous cell carcinoma.[15]

Furthermore, the emotional and psychological burden of living with PRP, especially in patients with HIV, can significantly affect quality of life.

TREATMENT

The treatment of PRP includes biologic medications, oral retinoids and methotrexate as the main pillars.[27] Most studies focus on type I PRP, while experience with other types is more limited.[20] Systemic retinoids are the first-line treatment for PRP, followed by methotrexate as a secondary option.[19] However, with the introduction of biologic treatments, which are generally reserved as third-line option due to their cost and insufficient clinical evidence, retinoids and methotrexate have been considered as therapeutic alternatives.[7,16,24,25]

Although topical treatments are insufficient on their own, they are used as adjuncts in severe cases.[27] Emollients such as cream or ointments improve patient comfort and should be applied several times a day.[16] In patients with thick keratoderma and fissures, a 40% urea cream, particularly under occlusion, is recommended.[5,20,27] Other topical agents that have been tried include retinoids, calcipotriol and calcineurin inhibitors.[1,7,20] As for topical corticosteroids, their efficacy is not fully clear when compared to emollients. A trial of two weeks with a medium-potency corticosteroid on the trunk and extremities and lower-potency steroid on the face and folds, is suggested. If no improvement occurs within two weeks, treatment should be discontinued.[5,27] Topical corticosteroids seem to be more effective in pediatric patients or those with limited disease, such as type IV PRP.[1,3] In contrast, systemic corticosteroids are not useful and may exacerbate the disease.[18]

In chronic and symptomatic cases, it is recommended to continue both topical and systemic treatment to control inflammation.[5] Systemic therapy is generally initiated immediately in adults with generalized or highly symptomatic PRP, while a more conservative approach is taken in children.[27]

Recently, biologic agents have shown promising results in the treatment of PRP with therapies such as anti-IL-17 and anti-IL-23 demonstrating rapid and effective responses in selected cases.[7,29] IL-17A inhibitors, such as ixekizumab and secukinumab 3 and brodalumab, which blocks the IL-17 receptor, have shown good results.[27] On the other hand, IL-23 inhibitors, such as Ustekinumab which inhibits the IL-12 and IL-23 p40 subunit, guselkumab, risankizumab and tildrakizumab which target the p19 subunit of IL-23, have also demonstrated efficacy.[1,3,7]

A 2019 systematic review on the use of biologic products in refractory PRP revealed that biologic therapy, whether monotherapy or combination therapy, achieved a 75% improvement.[13] However, since the review was limited to case reports and small series, definitive recommendations cannot be made. A retrospective study indicated that patients treated with biologics had better survival rates and fewer short-term complications.[30] IL-23 inhibitors are suggested for patients with refractory PRP.[7]

Oral retinoids, such as isotretinoin, acitretin and etretinate, are first-line treatment in some regions due to their antiproliferative, immunomodulatory and anti-inflammatory effects mediated by nuclear retinoic acid receptors (RAR).[1,6,19] In one study, 61.1% of patients experienced an excellent response to isotretinoin, which has a shorter half-life, making it favorable for patients.[3,18] The recommended dose for adults with PRP is 0.5 to 1 mg/kg per day.[19,20]

Methotrexate is reserved for patients who cannot access or tolerate biologic therapy or oral retinoids.[19,20] The combination of methotrexate with oral retinoids in patients with type I PRP has been reported to improve outcomes, although with a higher risk of hepatotoxicity.[18]

Other additional treatments include tumor necrosis factor alpha (TNF-α) inhibitors and phototherapy. Biologic TNF-α inhibitors, such as infliximab, adalimumb and etanercept, have shown efficacy in the rapid resolution of erythema and scaling.[3,12,29] Although they were the first used in PRP, their use has decreased in favor of more specific biologic inhibitors.[27] TNF-α inhibitors are used as second-line treatment for refractory PRP, but their use has declined in favour of newer therapies.[7,19] Phototherapy with ultraviolet A or B light has been shown to be ineffective or even exacerbate PRP in most cases, although

some documents successes when combined with acitretin.[1,18,19,20,27] A documented case of exposure to ultraviolet B light, which exacerbated the disease, showed sustained remission following treatment with ultraviolet A light.[31] Phototherapy combined with oral retinoids has also been tried with some success in case series.[1] Systemic treatment may be discontinued in patients who achieve clinical remission and restarted if signs of disease activity reappear.[27]

In patients with HIV-associated PRP, treatment is more complex. Although evidence of efficacy is limited, the general approach is followed with specific adjustments for the patient´s immunological status. Treatment must be carefully monitored due to possible interactions with antiretroviral therapy. Improvement has been documented with the use of triple antiretroviral therapy, such as zidovudine, lamivudine and saquinavir or zidovudine, lamivudine and nevirapine, along with acitretin.[4,8,18,27] However, treatment outcomes vary and successful treatment has not been documented without the inclusion of antiretroviral therapy.[27] Ixekizumab presents an attractive option for patients with HIV-associated PRP, as it is not associated with lipid or hepatic abnormalities and avoids the generalized immunosuppression observed with other therapies, such as methotrexate, azathioprine and cyclosporine.[13,20]

Finally, it is important to recognize the psychological impact on patients with PRP, who often experience depression, anxiety, shame, isolation and loss of self-esteem.[16,32] One study revealed that 38% of patients experienced passive suicidal ideation and 4% active suicidal ideation during the disease's course. This underscores the importance of an integrated approach that also addresses the emotional well-being of patients.[5]

CONCLUSION

HIV-associated PRP is a rare but severe manifestation that presents significant challenges in terms of diagnosis and treatment. The complex interaction between HIV and PRP treatments requires a personalized approach, especially in patients with suboptimal viral load control. Despite advances in understanding its pathogenesis and emerging therapeutic options, there is still no standardized management protocol. Further clinical studies are needed to assess the efficacy and safety of current treatments to improve outcomes for these patients.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Wang D, Chong L, Chong WS, Oon HH. A review on pityriasis rubra pilaris. Am J Clin Dermatol. 2018; 19(3):377-90.
2. Ortiz AM, Bernedo PI, Casas AH. Pitiriasis rubra pilaris: A propósito de un caso. Rev Argent Dermatol. 2020;3:103.
3. Joshi TP, Duvic M. Pityriasis rubra pilaris: An updated review of clinical presentation, etiopathogenesis and treatment options. Am J Clin Dermatol. 2024;25(2):243-59.
4. Williams A, George A, Thomas EA, Koshy JM. Pityriasis rubra pilaris type 6: A case report in an AIDS patient. Indian J Sex Transm Dis AIDS. 2020;41(1):100-1.
5. StatPearls.Pityriasis rubra pilaris. Available from: Pityriasis Rubra Pilaris – StatPearls. NCBI. 2025.
6. Chandra R, Arif A, Antonius CS. Pitiriasis Rubra Pilaris yang Berhubungan dengan HIV. Cermin Dunia Kedokteran. 2019;47(6):455-8.
7. Chandy RJ, Chokshi A, Tan I, Feldman SR. Biologics for treatment of pityriasis rubra pilaris: A literature review. J Cutan Med Surg. 2024;28(3):269-75.
8. Sehgal VN, Srivastava G, Dogra S. Adult onset pityriasis rubra pilaris. Indian J Dermatol Venereol Leprol. 2008;74(4):311-21.
9. González-L A, Velasco E, Pozo T, Del Villar A. HIV-associated pityriasis rubra pilaris responsive to triple antiretrovial therapy. Br J Dermatol. 1999;140(5):931-4.
10. UpToDate. Pityriasis rubra pilaris: Pathogenesis, clinical manifestations and diagnosis, 2024. Available at: https://www.uptodate.com/contents. Accessed on 18 November 2024.
11. Nair PA, Sheth N. Atypical Adult-Oset Pityriasis rubra pilaris in an HIV-positive adult male. Indian J Dermatol. 2018:63(6):522-4.
12. Lerebours-Nadal L, Beck-Sague CM, Parker D, Gosman A, Saavedra A, Engel K, et al. Severe, disfiguring, pityriasis rubra pilaris in a woman in the Dominican republic: histopathologic diagnosis and response to antiretroviral therapy. J Int Assoc Provid AIDS Care. 2016;15(1):11-4.
13. Kranyak A, Shuler M. Pityriasis rubra pilaris cleared with ixekizumab in an HIV-positive patient. JAAD Case Rep. 2022;27:55-57.
14. De D, Dogra S, Narang T, Radotra BD,Kanwar AJ. Pityriasis rubra pilaris in a HIV-positive patient (Type 6 PRP). Skinmed. 2008;7(1):47-50.
15. Zeinab MA, Azadeh G, Seyed NE, Ronak M, Safoura S, Alireza J, et al. A comprehensive review on HIV-associated dermatologic manifestations: from epidemiology to clinical management. Int J Microbiol. 2023;4:6203193.
16. Ross NA, Chung H-J, Li Q, Andrews JP, Keller MS, Uitto J. Epidemiologic, clinicopathologic, diagnostic and management challenges of pityriasis rubra pilaris: A case series of 100 patients. JAMA Dermatol. 2016;152(6):670-5.
17. Montero-Menárquez J, Samranch VA, Puig SL. Pityriasis rubra pilaris: a multicentric case series of 65 spanish patients. Actas Dermosifiliogr. 2024;115(8):761-5.

18. Klein A, Landthaler M, Karrer S. Pityriasis rubra pilaris: a review of diagnosis and treatment. Am J Clin Dermatol. 2010;11(3):157-70.
19. Moretta G, De Luca EV, Di Stefani A. Management of refractory pityriasis rubra pilaris: challenges and solutions. Clin Cosmet Investig Dermatol. 2017;10:451-457.
20. Roenneberg S, Biedermann T. Pityriasis rubra pilaris: algorithms for diagnosis and treatment. J Eur Acad Dermatol Venereol. 2018;32(6):889-98.
21. Zhou T, Muqrin AA, Abu-Hilal M. Updates on pityriasis rubra pilaris: a scoping review. J Cutan Med Surg. 2024;28(2):158-66.
22. Seenivasan V, Janaki VR, Sentamilselvi G. Human immunodeficiency virus associated follicular syndrome. Ind J Dematol. 1998;43(4):67.
23. Mufti A, Lytvyn Y, Maliyar K, Sachdeva M, Yeung J. Drugs associated with development of pityriasis rubra pilaris: A systematic review. J Am Acad Dermatol. 2021;84(4):1071-81.
24. Sagut P, McIntyre EM, Elston DM. Pityriasis rubra pilaris. J Am Acad Dermatol. 2024; 92(2):376-8.
25. Mancilla-Gudiel PM, Arenas R. Pitiriasis rubra pilar: una revisión. Dermatol CMQ. 2020;18:78-81.
26. Ji-Xu A, Leigh DK, Maloney NJ, Worswick S. Clinical course, diagnostic patterns and treatment outcomes in patients with pityriasis rubra pilaris. J Am Acad Dermatol. 2022;87(6):1450-1.
27. UpToDate. Pityriasis rubra pilaris: Prognosis and management, 2024. Available at: https://www.uptodate.com/contents/pityriasis-rubra-pilaris. Accessed on 28 December 2024.
28. Abdullayeva Z. "HIV-associated Pityriasis rubra pilaris type 6: First case report from Turkey. J Clin Images Med Case Rep. 2023;4(11):2711.
29. Sood S, Akuffo-Addo E, Yeung J, Mufti A. Biologic treatment options for pityriasis rubra pilaris: An evidence-based systematic review. J Am Acad Dermatol. 2023;89(6):1306-8.
30. Kettering C, Khosravi H, Ortiz C, English JC 3rd. Drug survival of systemic and biologic monotherapy treatments for pityriasis rubra pilaris: A retrospective observational study. J Am Acad Dermatol. 2022;86(5):1142-3.
31. Kaskel P, Peter RU, Kerscher M. Phototesting and phototherapy in pityriasis rubra pilaris. Br J Dermatol. 2001;144(2):430.
32. Velasco RC, Shao C, Greiling TM. Patient-reported cutaneous signs and symptoms of adult pityriasis rubra pilaris and correlation with quality of life and clinician-reported severity: A cross-sectional study. J Am Acad Dermatol. 2024;90(1):200-2.

Cite this article as: Pérez-García MM, Ortuz-Lessa C, Amador-Eleuterio S, Gallegos-Ramos AL. Pityriasis rubra pilaris and HIV: a diagnostic challenge-case report and critical review of the literature. Int J Res Med Sci 2025;13:1731-7.