Infection-provoked psoriasis: Induced or aggravated (Review)

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Abstract. Psoriasis is a common chronic, immune-mediated, inflammatory skin disorder, with a reported prevalence of 0.0-2.1% among children and 0.91-8.50% among adults, worldwide. Psoriasis is induced by several environmental factors, including infection, alcohol consumption, drugs, trauma, acute withdrawal of systemic or potent topical corticosteroids, body mass index and endocrine disorders. Increasing evidence suggest that a variety of microorganisms play key roles in the induction and exacerbation of psoriasis. Pathogens, such as *streptococci* and *staphylococci* are considered causal factors, presumably via superantigen activation of skin-seeking T cells. In addition, fungal pathogens, such as *Candida* and *Malassezia*, and viral agents, such as human immunodeficiency virus, hepatitis C virus infection and human papillomavirus, are also closely associated with psoriasis. Recently, several types of pathogens, such as *Helicobacter pylori* infection, *Zika* virus and scabies, have been reported to potentially trigger psoriasis. The present review discusses the underlying molecular mechanisms by which these infections influence psoriasis to provide a better understanding of the pathogenesis of psoriasis.

Contents

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1. Introduction

Psoriasis is one of the most common inflammatory skin diseases and is considered to have several causal agents, including infection, alcohol consumption, drugs, trauma, acute withdrawal of systemic or potent topical corticosteroids, body mass index and endocrine disorders (1). It is recognized that certain species of bacteria, such as, *streptococci* and *staphylococci*, are associated with the induction and aggravation of psoriasis (2). However, the specific mechanisms are constantly being updated. In recent years, pathogens, including the bacterial species *Helicobacter pylori*, the fungi species *Malassezia* and *Candida*, viruses [human immunodeficiency virus (HIV), human papillomavirus (HPV) and hepatitis C virus infection (HCV)] and the *Sarcoptidae* species of mites (Table I) are considered to be associated with the occurrence and development of psoriasis (3). The present review aims to discuss all the possible pathogens reported associated with the onset or exacerbation of psoriasis, along with their potential and molecular mechanisms. In conclusion, information on the current understanding of the role and therapeutic targets of the infection during the onset and acute exacerbation of psoriasis is provided.

2. Bacteria

Streptococci. In the first report published 50 years ago, two thirds of patients with guttate psoriasis (GP) had an acute sore throat 1-2 weeks prior to the outbreak, and serological evidence of a preceding streptococcal infection (4). β-haemolytic streptococcal throat infection is associated with the onset and acute exacerbation of psoriasis (5). In a prospective study, patients with psoriasis suffered from a sore throat 10 times more than household controls (6). *Streptococcus* infection stimulates T cell proliferation through bacterial superantigens, without prior intracellular processing by antigen-presenting cells (7). Infiltrating T cells, which subsequently induce psoriatic skin lesions, derive from tonsils where streptococcal infection promotes a skin-homing phenotype,

in which streptococcal and skin-specific epitopes function through molecular mimicry (8).

The human cathelicidin interleukin (IL)-37 peptide, a multifunctional regulator of components of the innate immune response, is overexpressed in patients with psoriasis (9). Following induction of streptococcal infection, monocytes expressing the surface markers CD14 and CD16 are usually activated in patients with GP. Peptidoglycan (PGN) is a major component of the cell wall of *Streptococcus*, and an increasing number of PGN-containing cells have been detected in psoriatic lesions (10). Qian *et al* (11) assessed the function of human IL-37 in the activity and differentiation of PGN-induced monocytes and subsequently investigated its association with GP. The results demonstrated that the levels of $CD14^+$ and $CD16^+$ monocytes, circulating IL-37, the soluble form of triggering receptor expressed on myeloid cells (sTREM-1), and anti-streptolysi O are significantly higher in patients with GP compared with healthy controls. In addition, the percentages of $CD14^+$ and $CD16^+$ monocytes are positively correlated with serum sTREM-1 levels and Psoriasis Area and Severity Index (PASI) scores (12). Therefore, the authors concluded that IL-37 plays an important role in psoriasis via synergistic action with PGN to direct the polarization and differentiation of monocytes into a proinflammatory phenotype (13,14). In addition, co-culture of psoriatic skin lymphocyte-associated antigen (CLA^+) T cells and lesional epidermal cells revealed that IL-17A is the primary inducer of *ZC3H12A* expression in keratinocytes treated with the supernatant derived from *Streptococcus pyogenes*-activated psoriatic precursor lesions (15).

A systematic review published in 2001 reported no satisfactory evidence of the efficacy of antibiotics in patients with chronic plaque psoriasis or acute GP, mainly due to the lack of satisfactory results compared with the placebo (16). A new and updated Cochrane review published in 2019 described the association between treatment with anti-streptococcal agents for chronic plaque psoriasis or GP (17). However, both the efficacy and safety of anti-streptococcal interventions remain unclear. These trials present a high risk of bias, only include a small number of unrepresentative participants and report limited measurements on the outcomes of interest. These studies failed to investigate the effects of streptococcal infection, and a key intervention (amoxicillin) was not assessed. Thus, further studies are required to assess the efficacy and tolerance of penicillin V or amoxicillin among both children and young adults with GP.

Several case series have recommended treatment with tonsillectomy for selected patients with GP and pustular psoriasis (18,19). The results of a randomized controlled trial demonstrated that tonsillectomy is an important alternative to the current treatment of psoriasis for a selected group of patients, which provides long-lasting improvements (20). However, several studies with long-term follow-up are warranted to accurately determine the benefits of tonsillectomy in psoriasis (20,21). In addition to the association between streptococcal infection of the throat and psoriasis, other case reports of GP in children with perianal streptococcal disease have been published (22-24). A dental focal infection may be an important causative or exacerbating factor for pustulosis palmaris et plantaris (PPP), and dental examinations along with tonsillar examinations, are recommended prior to treatment for PPP (25).

Staphylococci. In a systematic review, patients with psoriasis were 4-5 times more likely to be colonized by *Staphylococcus aureus* (*S. aureus*) on the skin and 60% more likely to be colonized in the nasal cavity compared with healthy controls [relative risk and 95% confidence interval (CI), skin: 5.54 (3.21-9.57); nares: 1.60 (1.11-2.32)] (26). In addition, *S. aureus* isolates secrete more than one staphylococcal enterotoxin, including staphylococcal enterotoxins A, B, C and D (SEA, SEB, SEC and SED), and toxic shock syndrome toxin-1 (27). In another study, *S. aureus* isolated from patients with psoriasis secreted high levels of staphylococcal enterotoxin. Notably, the PASI scores of these patients were significantly higher compared with toxin negative *S. aureus* isolates (28). Göçmen *et al* (29) reported that both *S. aureus* colonization and the presence of toxin positive and *agr* genes may be critical for disease activity in patients with psoriasis. Compared with patients with atopic dermatitis (AD) or lichen planus, a greater inflammatory response was induced in patients with psoriasis following the topical application of staphylococcal toxins to tape-stripped, clinically uninvolved skin (30). Moriwaki *et al* (31) demonstrated that *SLURP1* participates in the pathophysiology of psoriasis by inhibiting the growth of *S. aureus*. Furthermore, T cells activated by *S. aureus* have been cultured from skin lesions of individuals with psoriasis. Unlike the total count of PGN-containing macrophages, the number of macrophages carrying *S. aureus*-specific PGN in these skin lesions is limited, suggesting that T cells specific to PGN from *S. aureus* may not participate in the pathogenesis of the disease (32).

Helicobacter pylori (H. pylori). *H. pylori*, a microaerophilic gram-negative bacterium, naturally colonizes the human gastric mucosa (33). Considered a global health problem, the incidence rate of *H. pylori* infection is up to 80-90% in some developing countries (34). Recently, several studies have reported an association between certain skin diseases and *H. pylori* infection, including AD, chronic spontaneous urticaria (35), rosacea (36), alopecia areata (35) and psoriasis, which may provide novel therapeutic strategies for these skin diseases. Notably, a recent systematic review reported that *H. pylori* infection is associated with the pathogenesis of psoriasis as these patients have higher PASI scores (37). The function of *H. pylori* infection in the pathogenesis of extradigestive manifestations is attributed to the systemic effect of the local inflammation caused by the bacterium (38). The bacterium colonizes the gastric mucosa and subsequently releases several cytotoxic substances to induce an inflammatory response (39). Thus, gastric infection with *H. pylori* may induce systemic effects that include increased permeability of the gastric mucosa to food antigens, immune regulation, autoimmune mechanisms and damage to vascular integrity (40). Researchers have proposed that the diversity of *H. pylori* immunopathogenesis may result from an imbalance in Th1 and Th2 responses (41). The enterotoxin secreted by *H. pylori* binds to the T cell receptor and induces the expression of the T cell skin homing receptor and skin lymphocyte antigen, which results in the occurrence of T cell superantigen (42,43).

Table I. List of pathogens associated with the onset or exacerbation of psoriasis.

Bacterium
Streptococci
Staphylococci
Helicobacter pylori
Porphyromonas gingivalis
Chlamydiae
Fungi
Candida
Malassezia
Viruses
HIV
HPV
HCV
Human herpesvirus
Coxsackie B
Chikungunya
Zika virus
Human parvovirus B19
COVID-19
Other
Sarcoptidae

HIV, human immunodeficiency virus; HPV, human papillomavirus; HCV, hepatitis C virus.

3. Other bacterial agents

Periodontitis is a type of chronic gingivitis caused by an excessive inflammatory response to microbial colonies in dental plaques (44). It affects approximately one third of adults >30 years and approximately half of adults >50 years (45). Recently, the association between periodontitis and immune-mediated inflammatory diseases has attracted great interest (46). It is speculated that bacterial colonization in the oral cavity triggers an overactive immune response in susceptible hosts, causing a permanent inflammatory process associated with autoimmune disease (47,48). This association has been extensively investigated in rheumatoid arthritis (RA), in which patients with periodontitis are 2-8 times more likely to have RA than patients without periodontitis (49).

A systematic review demonstrated that the risk of psoriasis is significantly higher among patients with periodontitis (50). The causative agent is speculated to be *Porphyromonas gingivalis*, a common gram-negative anaerobic periodontal pathogen, due to its unique ability to produce peptidyl arginine deiminase, the major enzyme that promotes the post-translational citrullination of peptides (51). Excessive levels of citrullinated peptides subsequently induce the production of anti-citrullinated peptide antibody (ACPA), a critical autoantibody involved in the pathogenesis of inflammatory diseases, such as RA synovitis (52). However, excessive citrullination of peptides is unlikely to increase the risk of psoriasis, as ACPA plays no role in the pathogenesis of this inflammatory skin disease (53). An informative analysis would be to determine whether patients with psoriasis and periodontitis have a risk of developing psoriatic arthritis compared with the general population with psoriasis (54). Although the exact molecular mechanism underlying the increased risk of psoriasis among patients with periodontitis remains unclear, several potential explanations have been proposed. First, it is likely to be associated with a common pathology between psoriasis and periodontitis, as an exaggerated immune response to the presence of a microbiome on the epithelial surface is observed in both cases, which may indicate a common genetic susceptibility that modulates dendritic cell activity and Toll-like receptor expression (55). Another explanation is that the bacteria and their products involved in periodontal infection induce the activation of Th17 cells and increase IL-17 expression (54). However, further studies are required to accurately determine the molecular mechanisms underlying this risk (50,56,57).

Chlamydiae is a type of intracellular microorganism that tends to elicit persistent infection and may be the cause of neoplastic changes in host cells (58). Several studies performed over the last 5 years have reported a close association between *Chlamydophila psittaci (Cp)* and ocular adnexal malignant lymphomas, Waldeyer ring lymphomas and cutaneous diffuse large B-cell lymphomas (59,60). Fabris *et al* (61) investigated the association between a subclinical *Cp* infection and autoimmune diseases, such as inflammatory polyarthritis. In the study, patients with seronegative polyarthritis, followed by psoriatic arthritis, had a higher prevalence of *Cp* infections. Subsequently, Stinco *et al* (62) reported that 17% (11/64) of patients with psoriasis had a subclinical *Cp* infection, while only 0.4% (1/225) of the healthy control group had a subclinical *Cp* infection. *Chlamydiae* is known to contribute to a strong immunogenic response that triggers several diseases (63). Similarly, the subclinical *Cp* infection is speculated to provide a basis for the staging of psoriasis by mimicking the host epitope, and its mechanism involves the function of bacterial superantigens (62).

4. Fungi

Candida. *Candida spp*. have been isolated from 15% of skin specimens from patients with psoriasis compared with just 4% of specimens from healthy controls (P=0.045), and in 60% of oral specimens from patients compared with 20% of specimens from healthy controls (P<0.01) (64). Notably, patients with psoriasis have lower levels of serum IgM, IgA and IgG against *Candida albicans (C. albicans)* compared with controls, suggesting that psoriasis may be associated with a decreased humoral immune reaction to *Candida* (64). *Candida spp*. may aggravate psoriasis by secreting toxins, and the presence of *C. albicans* in the gut has been speculated to induce the formation of superantigens, contributing to nonspecific T-cell activation and cytokine production, thereby triggering the psoriatic process (65). In addition, a previous study demonstrated that the outcome of over half of the 50 patients with psoriasis improved following treatments with oral nystatin (66).

Malassezia. *Malassezia spp*. are considered external triggers that induce the pathogenic features of psoriasis (67). Patch

testing was performed with sonicates of heat killed *Malassezia*, and the intact uninfected skin of 10 patients with inactive psoriasis developed clinical and histological skin lesions similar to psoriasis (68). In addition, Elewski (69) reported a patient with psoriasis who developed active lesions in skin lesions of pityrosporum folliculitis. Several studies have demonstrated an improvement in scalp psoriasis following treatment with oral ketoconazole, combined with a decrease in the amount of yeast (70,71). Taken together, these findings support the association between psoriasis and *Malassezia spp.* Previous studies on the prevalence of *Malassezia spp.* in patients with psoriasis identified several species, including *M. globosa*, *M. furfur* and *M. sympodialis* (72,73). Mechanistically, *Malassezia spp.* upregulates the expression of heat shock protein 70, transforming growth factor-β1 and integrin chains in keratinocytes, the expression of which is higher in psoriatic plaques colonized *Malassezia plaques* compared with noncolonized psoriatic plaques (74). However, further studies are required to determine whether *Malassezia* can induce the development of psoriatic lesions.

5. Viruses

HIV. HIV infection is an independent risk factor for 102,070 patients with psoriasis following elimination of age, sex and comorbidities (adjusted OR, 1.80; 95% CI, 1.38-2.36) (75). Over 80% of HIV-positive patients with psoriasis have recurrent lesions when their $CD4^+$ lymphocyte count reaches <450. Furthermore, previous studies have reported that $CD4^+$ counts <200 cells/mm^3 are associated with a 9-fold higher risk of clinically active psoriasis (76,77). However, the association between $CD4^+$ lymphocyte counts and psoriasis remains controversial. While $CD4^+$ lymphocyte counts are very low in the terminal stages of HIV infection, psoriatic lesions may regress completely (78). The imbalance of the $CD4^+/CD8^+$ lymphocyte ratio in patients with HIV may result in the disturbance of homeostasis, favouring psoriatic symptomatology (79). HLA-DR, which is rarely expressed in the epidermis of healthy individuals, plays an important role in antigen presentation. However, human keratinocytes can release interferon gamma (IFNγ) to induce HLA-DR expression (80). HIV infection increases HLA-DR expression in human keratinocytes by excessively secreting IFNγ, which results in the migration of leukocytes into skin tissues and promotes the reaction to streptococcal and staphylococcal superantigens, thus contributing to the psoriatic phenotype (81-83). Furthermore, certain viral proteins, including the negative regulatory factor protein and envelope glycoprotein GP120, function as superantigens in HIV infection, which increases the burden of psoriasis observed in patients with HIV (84,85). In addition, psoriasis is more difficult to treat and recurs more frequently in HIV-infected individuals compared with noninfected individuals (86).

HPV. Jain *et al* (87) reported that genital warts (HPV infection) potentially trigger inverse psoriasis. However, these results may have been due to coincidence given that patients were lost to follow-up after the clearance of skin and genital lesions. Recently, a nationwide study performed over 12 years indicated that individuals with HPV have an approximately 2-fold increased risk of developing psoriasis compared with the general population. Furthermore, individuals with HPV infection have a higher risk of developing psoriasis with age compared with heathy individuals (88). However, the molecular mechanism by which HPV infection increases the incidence of psoriasis remains unclear. It has been speculated that the inflammatory state following HPV infection upregulates the synthesis of nerve growth factor (NGF), which influences the pathological features of psoriasis, such as keratinocyte proliferation, angiogenesis and T-cell activation. Notably, high NGF expression has been observed in psoriatic-prone skin compared with normal skin (89).

HCV. Imafuku and Nakayama (90) investigated the association between psoriasis and HCV infection. The results of this study demonstrated that HCV infection triggers psoriasis, particularly late-onset psoriasis, potentially due to upregulated expression of tumor necrosis factor-α (TNF-α), a common mediator of the two conditions (91). Supporting this hypothesis, treatment of psoriasis with TNF-α antagonists, without deterioration of HCV, was reported. Farag *et al* (92) demonstrated that 90 patients with psoriasis who were infected with HCV had significantly increased PASI scores ($P<0.001$), suggesting that HCV infection may lead to the exacerbation of psoriasis. In addition, a positive association between the viral load and PASI scores was observed. The skin load, rather than the presence of HCV infection, may play a critical role in the pathogenesis of psoriasis. This cutaneous load is positively associated with the duration of disease (92). HCV infection not only increases psoriasis severity, but may also play a role in its different clinical aspects. Chun *et al* (93) recently confirmed the immunological association between HCV and psoriasis. It was demonstrated that patients with psoriasis who are HCV-positive have increased expression levels of the inflammatory genes responsible for psoriasis development, including *cathelicidin*, Toll-like receptor 9 and IFNγ. However, the incidence of psoriasis and HCV substantially vary between race and nations. Further studies are required to investigate the association between psoriasis and HCV and determine the molecular mechanisms underlying the two conditions.

Human herpesvirus (HHV). Epstein-Barr virus (EBV) is a HVV that is carried by 90% of the population worldwide, and generally persists throughout an individual's lifetime, without causing disease (94). The results of a cross-sectional study demonstrated that neither severe psoriasis alone nor in combination with immunosuppressive therapy are associated with an increase in EBV or HHV6 replication in white blood cells (95). Loh *et al* (96) reported that a patient developed symptoms of glandular fever 3 weeks prior to the emergence of GP. In this case, serological evidence indicated a recent EBV infection and IgM positivity. Jiyad *et al* (97) reported that a 79-year-old woman presented with significant EBV viremia and generalized pustular psoriasis (GPP) and serial measurements of EBV DNA indicated an association with the deterioration of her clinical condition. The authors speculated that reactivation of EBV may have triggered the development of GPP.

Furthermore, Yoneda *et al* (98) reported that two patients developed GPP after infection with cytomegalovirus (CMV). Increasing evidence suggests that CMV may be associated with GPP (99,100). TNF-α expression has been observed in GPP lesions, where CMV DNA fragments were also detected in the same site (101). In addition, overexpression of CMV IE protein in keratinocytes is followed by an increase in TNF-α activity (102). TNF-α, along with other cytokines, such as interferon-c (IFN-c) and IL-6, may be injurious, and the complex interaction of TNF-α *in vivo* potentially elicits the occurrence of GPP (103). However, Kirby *et al* (104) reported that CMV, HHV6 and HHV7 infections are unlikely to play important roles in the pathogenesis of psoriasis. In addition, limited studies have reported that infection with varicella-zoster virus and herpes simplex virus may be associated with the occurrence of psoriasis (105,106). However, further studies are required to determine the molecular mechanism underlying the association between HHV infection and psoriasis (107).

6. Other viruses

Korzhova *et al* (108) reported that 25 patients with psoriasis presented antibodies to the *Coxsackie B* virus in diagnostic titres, with several patients having displayed a progressive increase in the titres of the aforementioned antibodies, which suggests an association between psoriasis and *Coxsackie B* virus. In addition, antibodies to *Coxsackie B1* and *B5* viruses have been demonstrated to occur more frequently among seropositive patients, underscoring their particular importance in the development of psoriasis (108).

Chikungunya (CKG) infection, which is provoked by an RNA virus and transmitted by *Aedes* (*A. aegypti and A. albopictus*), is a re-emerging viral disease (109). CKG fever is perceived as an epidemic in Africa and Asia. CKG is characterized by a sudden fever with severe arthralgia, constitutional symptoms and skin eruption, which lasts for 1-7 days (110). It is also characterized by several cutaneous manifestations in axillae and perineal regions, including maculopapular eruption, centrofacial melanosis and aphthous-like ulcers (111). Inamadar *et al* (112) described two cases of an exacerbation of psoriasis following a suspected CKG infection. Furthermore, Seetharam and Sridevi (113) reported that five patients developed psoriasis 5-14 days after CKG infection, and two other patients experienced an exacerbation of existing psoriasis. The authors proposed that the virus acts as a superantigen and activates cellular immunity through innate or acquired pathways, thereby promoting immunological changes that produce different clinical manifestations in insusceptible individuals. Collectively, these findings suggest that CKG infection triggers psoriasis, particularly in endemic areas.

Zika virus (ZIKV), an emerging arthropod-borne virus, belongs to the Flaviviridae family, and its epidemic scope is spreading throughout the tropical and subtropical regions of the world since the year of 2007 (114). Although ZIKV is mostly asymptomatic, the typical symptoms of infection consist of a mild fever, headache, fatigue, rash, arthritis and/or joint pain, myalgia and conjunctivitis (115,116). To the best of our knowledge, Paniz Mondolfi *et al* (117) was the first to report a case of psoriasis that appeared 3 weeks after an uneventful regression of acute ZIKV infection. Based on the most recent experimental data on the biology of ZIKV infection in the skin, the authors hypothesized that ZIKV may have directly induced the development of GPP by stimulating keratinocyte-derived inflammatory mediator production and a multipotent T-cell driven immune reaction in the cutaneous milieu (118).

Human parvovirus B19 (PVB19), a single-stranded DNA virus, causes several clinical manifestations, including influenza-like illness, acute-onset polyarthropathy and rash (119). Several studies have reported the association between PVB19 infection and various chronic autoimmune and cutaneous diseases, including rheumatoid arthritis, systemic lupus erythematosus and multiple sclerosis (120,121). Yazici *et al* (119) indicated a statistically significant association between PVB19 infection and psoriasis. Notably, 79% patients with psoriasis presented specific anti-PVB19-IgG antibodies (119). Another study also supported the potential association between psoriasis and PVB19 infection, whereby PVB19 infection may induce the pathogenesis of psoriasis; however, the underlying molecular mechanism remains unclear (122). Future studies with larger sample sizes and measurements of immune parameters are required to determine the association between PVB19 infection and psoriasis.

In late December 2019, the world was introduced to the novel coronavirus disease (COVID-19). The novel SARS-CoV2, which humans encountered for the first time, binds ACE receptors to trigger pneumonia (123). Kutlu and Metin (124) reported a case of psoriasis exacerbation induced by treatment with hydroxychloroquine and oseltamivir in a patient with COVID-19. Although hydroxychloroquine may aggravate psoriasis, the presence of severe psoriasis within a short period may indicate that COVID-19 infection plays an important role in the pathogenesis of psoriasis (125,126). Plasma concentrations of inflammation-related cytokines, including IL-2, IL-7 and IL-10, granulocyte colony-stimulating factor, interferon-induced protein 10, monocyte chemokine 1, macrophage inflammatory protein 1 and tumour necrosis factor, have recently been reported to be potentially associated with the exacerbation of psoriasis in patients infected with SARS-CoV2 (127).

7. Other microorganisms

Scabies, which is induced by a parasitic infection carried by the mite *Sarcoptes scabiei,* is a pruritic cutaneous disease, and several systemic diseases have been reported to be associated with scabies infection, such as chronic kidney disease and chronic obstructive pulmonary disease (128). Notably, scabies and psoriasis are both characterized by pruritic skin lesions (129,130). In 1986, Keipert (131) reported that an individual with crusted scabies developed psoriasis. Liu *et al* (132) performed a nationwide study and demonstrated a potential association between scabies and psoriasis. Patients with scabies were reported to have an increased risk of psoriasis (hazard ratio, 3.03). A Th17 cell-mediated inflammatory pathway was speculated to function in patients with this condition. In addition, previous studies have reported elevated levels of IL-2, IL-4, IL-6, IL-17 and IFN-C in patients with both scabies and psoriasis. The risk of

subsequent psoriasis may decrease with early and aggressive treatment of scabies (133-135).

8. Conclusions

Several microorganisms are associated with the onset or exacerbation of psoriasis. However, other predisposing factors, such as alcohol and drug consumption, should always be eliminated first. A better understanding of the pathogenesis of psoriasis induced by infection not only contributes to achieving a greater appreciation of the disease process but also provides guidance for treatment strategies. In some cases of infection, such as psoriasis associated with HHV, lesions may become resistant to treatment; thus, early identification and management may help avoid problems of nonadherence to some extent. The pathogenesis of infection-provoked psoriasis has not been precisely clarified. In most cases, the infection is proposed to function as a superantigen, stimulating the proliferation of T-cells without prior intracellular processing by an antigen-presenting cell. Subsequently, infiltrating T cells induce psoriatic lesions. In some patients with a viral infection, the expression levels of cytokines increase, such as TNF-α and IFNγ, which is responsible for the development of psoriasis. However, anti-infective treatments are not always effective and require further investigation. In addition, further studies are required to determine why some individuals develop psoriasis following exposure to a specific infection.

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Availability of data and materials

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Authors' contributions

YT, WX and YF conceived and designed the study. XT, NL, YY, YH and DX contributed to draft the manuscript and revised it critically for important intellectual content. WX and YF confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

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Not applicable.

Competing interests

The authors declare that they have no competing interests.

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