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Review Article

A Review of the Latest Findings of Candida Colonization and Candidiasis in Patients with Psoriasis and Its Management

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A B S T R A C T

Psoriasis is an immune-mediated disease causing raised, scaly patches on the skin, especially the elbows, knees, and scalp due to systemic inflammation. The disease affects more than 125 million people worldwide. Psoriasis is associated with problems such as depression, reduced quality of life, cardiovascular diseases, stroke, lymphoma, diabetes mellitus, and metabolic syndrome. Psoriasis has a complex pathogenesis that is not yet fully understood. Many studies have shown the importance of the microbiome for the exacerbation of psoriasis. On the other hand, psoriasis and some other treatments can increase the colonization of some microbial agents in the body. *Candida* is commensal yeast that forms part of the natural microflora of the skin and mucous membranes. It has been shown that people with psoriasis are susceptible to candidiasis. In this review article, the causes of candidiasis, its diagnosis, and treatment in patients with psoriasis, as well as psoriasis aggravation methods by *Candida* species have also been investigated. To this end, keywords such as "psoriasis", "*candida*", " oral candidiasis", "cutaneous candidiasis", "vulvovaginal candidiasis", "epidemiology of *Candida"* and "balanitis" were searched. Articles published in national and international scientific databases, namely Google Scholar, PubMed/MEDLINE, Elsevier databases, Scopus, Science Direct, IranMedex, and SID from 2001 to 2023 were used. Oral candidiasis, cutaneous candidiasis, vulvovaginal candidiasis, and balanitis are diseases that affect people with psoriasis. On the other. This fungus may aggravate psoriasis through toxin production and activation of T lymphocytes independent of antigen presentation.

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Introduction

Psoriasis is a common inflammatory skin disease characterized by thick and erythema-raised plaques with silvery scales, affecting about 0.6-4.8% of the worldwide population [1, 2]. Psoriasis affects both males and females at about the same rate, but in males' onset is earlier than in females [3]. Clinical symptoms of psoriasis begin between the ages of 20 and 30 years, although children and adolescents may also be affected [4]. These symptoms are more common in elbows, knees, and scalp [3]. Psoriasis is the most common autoimmune disease, which, due to the long duration of the conflict, will cost a lot for the patient [5]. In addition, patients may experience problems such as depression, anxiety, malnutrition, sexual dysfunction, reduced quality of life, cardiovascular diseases, stroke, lymphoma, diabetes mellitus, and metabolic syndrome [2, 5]. High levels of triglycerides and cholesterol have also been reported in some people with psoriasis [6]. There is still no definitive treatment for this disease and only symptomatic treatment is used for affected people [5]. The pathogenesis of this disease is not completely understood, but an interaction between genetic and environmental factors has been recognized [3]. Among the environmental factors that have been considered in the pathogenesis of this disease, we can mention microbial factors and the formation of antibodies against them [5]. *Candida* species are one of the most important microbial agents playing a role in the creation or exacerbation of psoriasis which has been discussed in some articles [7-9]. Therefore, we decided to study the published articles on the role of *Candida* species in psoriasis and prepare a comprehensive report on the role of *Candida* species in the disease. In this study, we will provide the necessary suggestions in the field of diagnostic methods, improving the treatment of patients and preventing its recurrence.

Overview of *Candida* species and its history

Candida species is one of the most important fungal colonizers on the skin and mucosal surfaces of the human body such as the genitourinary tract, oral cavity, and gastrointestinal tract [10-14]. Many species of the *Candida* genus are commensal members of the human normal microflora; however, following some alterations in the host, some of these *Candida* species can also become opportunistic pathogens, by taking advantage of various predisposing factors, including locally or systematically impaired immune system, diabetes and use of steroids and broad-spectrum antibiotics [15, 16]. There are approximately 200 different species in the *Candida* genus, but only a few are harmful and can cause infections in humans [17]. *C. albicans*, *C. parapsilosis*, *C. glabrata*, C. *tropicalis*, *C. krusei*, *C. dubliniensis*, *C. famata*, and especially *C. albicans* are species that can cause disease in humans [15, 18, 19]. *Candida* genus was first described by Wilkinson in 1849; subsequently, Hausmann showed in 1875 that it is responsible for vaginal and oral candidiasis. Christine Marie Burkhout was the one who described the species albicans in 1923 during her thesis at the University of Utrecht. The word Albican is

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derived from the Latin word Albicare, meaning white, and *Candida* is derived from the Latin word 'Toga [17]. Over time, the genus *Candida* began to be classified and many other species were discovered. Currently, there are more than 200 species [20, 21].

Epidemiology of *Candida* species

Although the number of epidemiological studies on *the Candida* genus is still increasing worldwide, there is little information in some parts of the world, mainly due to the lack of accurate, specific, and sensitive diagnoses in routine clinical laboratories. The epidemiology of *Candida* species can be different depending on the geographic region, age, sex, underlying disease, and sampling location. In the Middle East and North Africa, An evident shift has been observed in the epidemiology of *Candida* species associated with invasive candidiasis in these countries from the predominant *C. albicans* toward the non-albicans species. Among them, while *C. tropicalis* prevails in Lebanon, Saudi Arabia, and UAE, *C. parapsilosis* is the most common species in Turkey, Kuwait, and Egypt [22]. In the Asia-Pacific region, *C. albicans* is the predominant *Candida* species causing invasive candidiasis/candidemia in Japan, Australia, Korea, Hong Kong, Singapore, Malaysia and Thailand whereas *C. tropicalis* is the most common *Candida* species in Pakistan and India [23]. In Iran, *C. albicans*, *C. parapsilosis*, *C. glabrata* and C. *tropicalis* are mentioned as causes of candidemia [24].

***Candida* colonization in patients with psoriasis**

Candida colonization in patients with psoriasis has been investigated by several studies. Various studies have shown more oral *Candida* colonization in patients with psoriasis compared to healthy people [25-28]. In these studies, the prevalence of oral *Candida* species isolation in patients with psoriasis has been reported to range widely between 23% and 78% [26-28]. So far, research has focused mainly on oral *Candida* colonization, while data on skin and intestinal colonization is limited. In conducted studies, psoriasis patients and controls did not differ significantly in the rate of *Candida* spp. Isolated from the skin [9, 26]. In the study of Elsner et al., the *candida* isolated from the stool of patients with psoriasis was significantly more than that of control subjects [29]. No significant relationship between age, sex, duration of disease, and colonization rate in patients has been reported in the mentioned articles [26, 29]. Reports on the relationship between the severity of the disease and the *Candida* colonization rate are different [26, 29, 30]. In Elsner et al. and Leibovici et al. studies, there was no significant relationship between the severity of the disease and the amount of *Candida* colonization [26, 29], but in Picciani et al.'s study, a significant association between the clinical severity of psoriasis and *Candida* colonization was seen [30].

Possible role of *Candida* species in psoriasis

Although *Candida* species are involved in the initiation or exacerbation of psoriasis, the exact mechanism is not known [5]. In this article, we will refer to some of the mentioned mechanisms. *Candida* spp. antigens, especially *C. albicans* surface proteins, have superantigen-like effects that lead to the activation of T lymphocytes independent of antigen

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presentation and excessive release of pro-inflammatory cytokines and exacerbation of psoriasis [9, 31]. Some studies have focused on the identification of somatic protein profiles of various *Candida* species isolated from psoriatic patients. In these studies, 12 somatic proteins have been identified in *C. albicans* (11 to 93.3 kDa), 12 somatic proteins in *C. parapsilosis* (11 to 109.6 kDa), 12 somatic proteins in *C. guilliermondii* (11 to 74.1 kDa), 12 somatic proteins in *C. lipolytica* (11 to >180 kDa), 7 somatic proteins in *C. tropicalis* (11 to 74.1 kDa) and 45 somatic proteins in *C. famata* (18 to > 180 kDa) [2, 32]. In these studies, it has not been determined which of these proteins can play a role in triggering or aggravating psoriasis; therefore, additional studies are required in this matter. *Candida* species can also aggravate psoriasis through toxin production [5]. Overgrowth of *Candida* in the intestine and other parts of the body causes a large amount of toxins to enter the blood. In a normal state, *Candida* toxin is neutralized by the liver, but when this toxin is high, the liver is not able to neutralize it. As a result, the toxin is deposited in the skin and causes the aggravation of lesions in psoriasis [6].

Candidiasis in patients with psoriasis

In addition to the colonization and exacerbation of psoriatic lesions, *Candida* species can cause a disease called candidiasis in different parts of the body of patients with psoriasis [33]. Infection in patients may be due to the nature of the disease or due to treatment [5]. The advent of some specific immunomodulating treatments for psoriasis has highlighted the association of psoriasis with various infections [34]. Interlukine(IL)-17 plays an important role in preventing candidiasis [9]. Some IL-17 inhibiting drugs such as Ixekizumab and Secukinumab used in the treatment of psoriasis, can increase candidiasis in patients with psoriasis [9, 35]. Disturbance in the production of anti-Candida antibodies has also been reported, which may be effective in increasing candidiasis [25]. *Candida* species can cause the following diseases in psoriasis patients:

Oral Candidiasis

Four types of oral candidiasis may be seen in people with psoriasis, which include: thrush, denture stomatitis, chronic hyperplastic, and perleche [33]. Patients with oral candidiasis can be asymptomatic or may present various clinical features such as sore throat, difficulty swallowing, and halitosis [33, 36]. This form of the disease can usually be diagnosed through physical examination and clinical history [33, 37]. Traditionally, microscopic examination using potassium hydroxide and lesion swab culture on selective media can also help in diagnosis [33, 38]. The diagnosis and treatment of all forms of candidiasis are listed in Table No. 1

Cutaneous candidiasis

Cutaneous candidiasis is a superficial infection of the skin and tends to occur in warm and moist areas of the skin including groin, abdominal skin folds, inframammary skin, and interdigital spaces [39, 40]. The mortality rate of cutaneous candidiasis is relatively low; however, if infections remain untreated for a long time, they may cause systemic candidiasis, with a mortality rate of approximately 25–50% [39]. Cutaneous candidiasis can be diagnosed

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based on the clinical appearance of the skin, the presence of pseudohyphae in wet potassium hydroxide mounts, and positive fungal culture from scraping of the affected areas [33, 40] (Table 1).

Vulvovaginal candidiasis (VVC)

Vulvovaginal candidiasis is a global issue and is the second most common cause of vaginitis in women after bacterial vaginosis [41]. It is estimated that 75% of women experience at least one VVC infection during their lives and 40%–45% will have two or more episodes [42]. Concomitant fungal infections can worsen psoriasis; therefore, VVC needs to be identified and treated quickly in these patients [33]. Vulvovaginal candidiasis can be diagnosed based on clinical symptoms and physical examination (Table 1). Diagnosis is confirmed by direct microscopic examination and culture. Most cases of this disease are uncomplicated and easily treated. A longer and different treatment is needed in complicated cases such as recurrent disease, infection due to a non-*albicans candida* species, and severe disease [43].

Candidal balanitis

Candidal balanitis is defined as inflammation of the glans penis in the presence of *Candida* species and typically affects sexually active adult males [44, 45]. The disease often occurs in uncircumcised men patients with diabetes and patients with primary inflammatory dermatoses, such as psoriasis [46]. Candidal balanitis can be diagnosed based on the clinical appearance and direct microscopic examination and culture may be helpful in some cases [33, 47] (Table 1). The sensitivity of the microscopic examination can vary depending on the sampling method, and the "adhesive tape" method is more accurate than swabbing [47].

Table 1. Guideline for the diagnosis and treatment of candidiasis

Disease	Symptoms		Treatment
Oral candidiasis	Thrush	Thick, white plaques on the tongue, buccal and gingival mucosa that are easily scratched	Clotrimazole troches, nystatin suspension, oral fluconazole, Itraconazole solution, posaconazole suspension
	Denture stomatitis	Inflammation and erythema of the oral mucosal areas covered by the denture	
	Chronic hyperplastic	White patch on the gums and inside the cheeks that cannot be scratched easily	
	Perleche	Erythema or fissuring at the corners of the mouth	
Cutaneous candidiasis	Thin, bright-red plaques that can be erosive, dry, scaly, oozing, or macerated. Pustules and collarette scales may also appear. Itching or burning may be present as well.		Clotrimazole cream, oral fluconazole
Vulvovaginal candidiasis	Pruritus, hyperemia, vaginal discomfort and leucorrhea, burning, soreness, cheese-like or watery vaginal discharge, dyspareunia, and vaginal and vulvar erythema		Clotrimazole cream, Butoconazole cream
Candidal balanitis	Redness, soreness, or swelling of the penis White, shiny patches at the top of the penis. Burning sensation during urination or sex		Clotrimazole cream, Miconazole cream

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Table 2. Some biological drugs for the treatment of psoriasis in adults

Drug	Biological composition	Therapeutic target	Candidiasis risk
Adalimumab	Monoclonal IgG1 antibody	TNF	Increase
Infliximab	Monoclonal IgG1 antibody	TNF	Increase
Etanercept	Recombinant TNF-α receptor	TNF	Increase
Certolizumab pegol	IgG_1antibody Fab' fragment	TNF	Increase
Secukinumab	Monoclonal IgG1 antibody	IL-17A	Increase
Ixekizumab	Monoclonal IgG_4 antibody	IL-17A	Increase
Brodalumab	Monoclonal IgG2 antibody	IL-17A receptor	Increase
Ustekinumab	Monoclonal IgG_1 antibody	IL-12/IL-23p40	little increase
Guselkumab	Monoclonal IgG1 antibody	IL-23p19	low
Tildrakizumab	Monoclonal IgG_1 antibody	IL-23p19	Infrequent
Risankizumab	Monoclonal IgG1 antibody	IL-23p19	No

TNF= Tumor necrosis factor; IL= Interlukine

Discussion

Psoriasis is a chronic inflammatory disease with long-term treatment [48]. Some of these drugs can increase the risk of *Candida* infection in these patients [49]. Several biological therapies are available that target various inflammatory cytokines; these include tumor necrosis factor (TNF) antagonists, IL-17 inhibitors, IL-12/23p40 inhibitors, and IL-23p19 inhibitors. An increase in the risk of *Candida* infection has been seen in the treatment with IL-17 inhibitors [48]. As an important factor in innate and adaptive immunity, IL-17 plays a fundamental role in the defense against yeast and other microorganisms at mucosal and cutaneous interfaces, so its inhibition can increase candidiasis [48, 50]. It is mentioned that the use of IL-23p19 inhibitors has a lesser role in increasing the risk of candidiasis in psoriatic patients [48]. *Candida* infections in patients with psoriasis often occur in areas that are not easily visible and are often asymptomatic; so, dermatologists should always inquire about oral or genital discomfort to identify candidiasis [33]. There is no requirement to screen patients for *Candida* infections before initiating therapy with biologic drugs [51]. *Candida* infection is suspected in patients with psoriasis, it is best to confirm the diagnosis by culture or molecular diagnosis to rule out non-*albicans* species of *Candida*, as some of them are intrinsically resistant to azoles [50]. Isolation of *Candida* alone does not necessarily indicate disease and must be accompanied by clinical signs and symptoms [33]. The majority of *Candida* infections in patients with psoriasis are localized and mild to moderate in severity; therefore, these infections cannot be a reason to discontinue treatment by biological drugs [50]. In these patients, candidiasis can be definitively treated with antifungal drugs [33]. The initial choice of treatment usually depends on the severity of the disease and whether the patient is pregnant [51]. In uncomplicated mild cases, topical antifungal treatment is preferable to systemic treatment. In cases of moderate to severe infection or if the disease is resistant to topical treatment, systemic antifungal therapy may be necessary [50]. Current guidelines state that

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systemic azoles (itraconazole, fluconazole, posaconazole, and isavuconazole) should be avoided in pregnant women, especially those in the first trimester, because of the possibility of birth defects associated with these drugs [52]. *C. albicans* is the most common species and empiric therapy should aim to target this species [51]. If candidiasis persists despite antifungal therapy, (re) confirmation of *Candida* infection, species determination, and susceptibility testing is recommended [50]. Relapse is not uncommon and subsequent treatment recommendations remain unchanged, although a longer course is sometimes used [51].

Conclusion

Increased *Candida* colonization and candidiasis have been confirmed in patients with psoriasis due to the nature of the disease and the use of some biological drugs. It is necessary to check patients for candidiasis during the treatment. Diagnosis of candidiasis is based on clinical and laboratory symptoms. Anti-IL-23p19 agents appear to have an acceptable safety profile in adults with moderate to severe psoriasis. An increase in *candida* colonization and candidiasis can aggravate psoriasis. More studies are needed to investigate the exact mechanism of this phenomenon. In candidiasis, topical therapy is generally effective and safe. Azoles are mostly used to treat Candida infections.

Conflict of Interest

The authors declare that there is no conflict of interest.

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