

The Comparison of *Candida* spp. Colonization on Psoriasis Vulgaris Patient and Control

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ABSTRACT

Background: Psoriasis is an inflammatory, chronic, and recurring skin disease that affects patient's quality of life. Environment, drugs, and infections in the form of antigen and toxin such as *Candida* spp. are assumed responsible for triggering and exacerbating psoriasis. **Purpose:** To compare the prevalence of skin colonization of *Candida* spp. on psoriasis vulgaris patient and control. **Methods:** This study was observational analytic research involving 33 psoriasis vulgaris patients and 33 healthy individuals. They were matched up based on sex, age, and lesion location. Swab of skin lesions on the Koebner area of both groups was cultured on CHROMagar media. *Candida* spp. was later identified and the colonization was calculated. **Result:** *Candida* spp. was found on 51.5% of lesions in psoriasis vulgaris patients and 21.2% in the control group with a significant difference of $p < 0.05$. The average colony number of *Candida* spp. in psoriasis vulgaris group was lower than the control group with no significant difference of $p > 0.05$. The predominant *Candida* spp. in the psoriasis vulgaris and control groups was *Candida krusei*, which were 35.5% and 85.6%, respectively. No significant difference between sex, age, period of complaints, age onset, degree of severity (Psoriasis Area and Severity Index), and therapy history, with positive culture of *Candida* spp. **Conclusion:** There was no difference in the number of *Candida* spp. colonization in the psoriasis vulgaris group and the control group. However, the culture positivity of *Candida* spp. in the psoriasis vulgaris patients was higher than in the control group.

Keywords: *Candida* spp., CHROMagar, Psoriasis vulgaris.

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BACKGROUND

Psoriasis is an inflammatory, chronic, recurrent skin disease characterized by red plaques, unsharply marginated, and covered with thick squama. It is caused by uncontrolled proliferation and differentiation of keratinocytes in the epidermis.^{1,2} This disease is associated with inflammatory dysregulation of the immune system (immunological processes mediated), genetic, and environmental. Its prevalence ranges from 2%–4% worldwide. Psoriasis vulgaris or plaque type psoriasis is the classic and the most common form, accounting for 85–90% of all psoriasis cases.^{2,3} Several factors including environment, infections (bacterial, fungal, and viral), individual stress, and drug exposure can provoke and exacerbate psoriasis.^{4,5} Psoriasis, which often recurs and persists in a chronic state, has a negative effect on the patient's quality of life in terms of self confidence, psychological burden (depression), and financial burden.⁶

Over the past few years, studies have suggested that microbial dysbiosis of the skin and mucosa can increase immune response in susceptible hosts, inducing a persistent inflammatory process associated with autoimmune disorders. Some bacteria (*Staphylococcus pyogenes*, *Staphylococcus aureus*), fungi (*Malassezia* and *Candida*), and viruses (*Papillomavirus*, *Retrovirus*) act as superantigens that activate non specific T cells thus initiating, exacerbating, and maintaining a chronic inflammatory process in skin diseases thus disrupting the patient's quality of life.⁴ In the amino acid sequence, *Candida albicans* (*C. albicans*) have an Int1 protein cell wall that shows a part that can directly bound to the class II major histocompatibility complex (MHC) so that it has a superantigen like effect. This superantigen like effect will induce non specific T lymphocytes without going through the classical antigen process and release excessive proinflammatory cytokines, causing cell damage.⁷

Previous studies showed that *Candida* colonization was found more on the oral mucosa, skin, and gastrointestinal tract of psoriasis patients than healthy people.⁸ In 2014, Sarvtin *et al.* reported that the number of colonization of *C. albicans* was 15% in psoriasis vulgaris patients and 4 in healthy persons ($p < 0.05$).⁹ Also, in 2015, Pezeshkpoor *et al.* in 2015 reported higher *C. albicans* colonies in oral of psoriasis patients (46%) than in healthy persons (18%) and on the skin (2%) than healthy persons (0%) with a significant difference ($p < 0.05$).¹⁰ Underlying mechanisms of the association between *Candida* spp. colonization and the severity of psoriasis are not fully understood. However, the number of *Candida* tends to progress along with changes in a compromised immune system. Therefore, in conditions of chronic inflammatory skin disorders such as atopic dermatitis and psoriasis, the prevalence of *Candida* spp. colonization would increase. Moreover, the pharmacological intervention for psoriasis patients is in the form of corticosteroid and immunosuppressive agents such as methotrexate, inhibitor tumor necrosis factor- α (TNF- α), and interleukin-17 (IL-17) could affect the response of the antifungal immune.^{1,11} As a result, the risk of *Candida* spp. superficial infection will also increase in those suffering psoriasis.¹² This was based on previous research, comparing the colonization of *Candida* spp. between psoriasis vulgaris patients and control. Such condition may act as a triggering factor and exacerbation of psoriasis.

METHODS

This was observational analytic research aimed to determine the differences between *Candida* spp. colonization on psoriasis vulgaris patients and healthy persons. We used a pair of samples, involving 35 psoriasis vulgaris patients and 35 healthy individuals. This research took place at the Dermatology and Venereology Outpatient Clinic, Dr. Soetomo General Academic Teaching Hospital Surabaya from January–March 2020. Psoriasis vulgaris patients, aged 18–65 years, diagnosed through clinical and histopathological examinations and signed informed consent. The control group consisted of healthy individuals in which age and sex matched with psoriasis vulgaris patients. The rejection criteria were patients taking oral antifungals in the last 1 month or topical antifungals in the last 2 weeks, pregnant, and diagnosed with diabetes mellitus, malignancy, or Human Immunodeficiency Virus (HIV)/ acquired immune deficiency syndrome (AIDS). We used consecutive sampling, which means selecting subjects per the acceptance criteria.

Demographic data and medical conditions of the sample included age, sex, length of the complaint, age of onset, severity, and medication history. Severity of the disease is measured based on the Psoriasis Area and Severity Index (PASI) score. The data collected from anamnesis, physical examination, skin specimens of psoriatic lesion from psoriasis patient and healthy skin from a control subject, specimens were growth the culture medium, and *Candida* colonies were counted. The culture medium used was CHROMagar *Candida*. Skin specimens were collected using a skin swab of 3 x 3 cm² in the Koebner area with a sterile cotton swab soaked in 1 ml of 0.9% normal saline solution, both in psoriasis vulgaris patients and healthy individuals. Swab results were put into the culture medium and allowed *Candida* to grow for 36–48 hours at 37°C. The number of colonies was counted and the *Candida* spp. identification through color changes produced by colony growth. The *Candida* culture examination was carried out at the Microbiology Laboratory of Dr. Soetomo General Academic Teaching Hospital Surabaya. The collected data were analyzed using the Statistical Package for Social Science (SPSS) software release version 17. Comparative tests were carried out using the chi square test and the t test, while the Shapiro Wilk test was used for the normality test. Ethical clearance was obtained from the Ethics Committee of Dr. Soetomo General Academic Teaching Hospital Surabaya before this research was conducted, Number 1652/KEPK/XI/2019.

RESULT

Thirty-three patients were diagnosed with psoriasis vulgaris clinically and histopathologically. Each group consisted of 20 males (60.6%) and 13 females (39.4%) with a ratio of 1.5 : 1. There were 11 individuals (33.3%) in the 45–55 years age group and 4 individuals (12.1%) in the 18–25 years age group ($p = 1.000$). The mean age of each group was 43.33 + 11.73 years. The statistical test results showed a homogeneous distribution in sex and age between the psoriasis vulgaris group and the control group (Table 1).

We found more *Candida* spp. positivity in females (53.8%) than males (50.0%). The statistical test results showed a difference between *Candida* spp. culture between both sexes ($p > 0.05$). We found 100% *Candida* spp. positivity in the 18–25 and 55–65 years age group of the psoriasis vulgaris group. However, there was no significant difference in culture positivity based on age in the psoriasis vulgaris group ($p = 0.711$) (Table 2).

Table 1. The distribution of sex and age on the psoriasis vulgaris and control groups

Variables	Psoriasis vulgaris (n = 33) (%)	Control (n = 33) (%)	Quantity (n = 66) (%)	p-value
Sex				1.000
- Male	20 (60.6)	20 (60.6)	40 (60.6)	
- Female	13 (39.4)	13 (39.4)	26 (39.4)	
Age (years)				0.959
- 18–25	4 (12.1)	4 (12.1)	8 (12.1)	
- 26–35	5 (15.2)	5 (15.2)	10 (15.2)	
- 36–45	7 (21.2)	7 (21.2)	14 (21.2)	
- 46–55	11 (33.3)	10 (30.3)	21 (31.8)	
- 56–65	6 (18.2)	7 (21.2)	13 (19.7)	

Table 2. The distribution of *Candida* spp. culture positivity proportion on psoriasis vulgaris group based on sex and age

Variables	Psoriasis vulgaris		Quantity n = 33 (%)	p-value
	<i>Candida</i> spp. Culture positive (n = 17) (%)	<i>Candida</i> spp. Culture negative (n = 16) (%)		
Sex				1.000
- Male	10 (50.0)	10 (50.0)	20 (60.6)	
- Female	7 (53.8)	6 (46.2)	13 (39.4)	
Age (years)				0.711
- 18–25 (Late adolescent)	4 (100)	0	4 (12.1)	
- 26–35 (Early adult)	2 (40.0)	3 (60.0)	5 (15.2)	
- 36–45 (Late adult)	2 (28.6)	5 (71.4)	7 (21.2)	
- 46–55 (Early elderly)	3 (27.3)	8 (72.7)	11 (33.3)	
- 56–65 (Late elderly)	6 (100)	0	6 (18.2)	

The highest prevalence of illness duration in psoriasis vulgaris patients was > 10 years (51.5%) and the mean duration of complaints in psoriasis vulgaris patients was 11.73 ± 8.73 . The results of *Candida* spp. positivity based on the illness duration were found for < 5 years (65.2%), 5–10 years (65.2%), and > 10 years (41.2%). However, statistically, there was no significant difference ($p = 0.274$). In this research, the age of disease onset in the psoriasis vulgaris group was mostly < 30 years (54.5%) and the mean age of onset was 31.15 ± 10.93 . The results of *Candida* spp. culture positivity on age of onset were ≥ 30 years (53.3%) and < 30 years (50%) but not statistically significant ($p = 1.000$) (Table 3).

We found 20 patients (60.6%) get into the most severe category with PASI > 10 and the average PASI score in psoriasis vulgaris patients was 13.19 ± 8.71 . Ten psoriasis vulgaris patients (50%) with severe severity (PASI > 10) had positive culture results for *Candida* spp., while 10 patients (50%) had negative culture results. In moderate (PASI 5–10) and mild (PASI < 5) severity, 5 patients (83.3%) and 2 patients (28.6%) had positive cultures for *Candida* spp., respectively. However, there was no significant

difference in the degrees of severity ($p = 0.789$) (Table 3). Positive culture result means positive for *Candida* spp. based on the treatment history, all of the methotrexate, topical corticosteroids, and narrowband-ultraviolet B (NB-UVB) phototherapy did not have a significant statistical difference ($p > 0.05$) (Table 4).

Of the 33 psoriasis vulgaris patients, positive cultures of *Candida* spp. were found in 51.5% of psoriasis vulgaris patients and only 21.2% positive cultures were found in the control group with statistically significant differences ($p = 0.021$) (Table 5). The Odds Ratio of *Candida* spp. colonization on the skin of psoriasis vulgaris patients and the control group was 3.94. The mean colony count of *Candida* spp. in the psoriasis vulgaris group was smaller than the control group, which was 6.71 ± 18.850 and 10.29 ± 9.759 , respectively. The Mann-Whitney test result showed that the mean value of *Candida* spp. of the two groups did not differ significantly ($p = 0.226$) (Table 5).

Of the 25 study subjects with *Candida* colony growth, the identification of *C. albicans* was found in 5 psoriasis vulgaris patients 29.4%, *C. krusei* 35.3%, *C. glabrata* 5.8%, the remaining was a mixture of two

types of *Candida*, namely *C. albicans* and *C. krusei* 23.5%, *C. krusei* and *C. tropicalis* 5.8%. In the control group there were no *C. albicans*, but only 85.7% *C. krusei*, 14.3% *C. albicans*, and 14.3% *C. krusei* (Table 6).

Table 3. The distribution of *Candida* spp. culture positivity proportion on psoriasis vulgaris group based on the duration of psoriasis, onset of psoriasis, and PASI score

Variables	Psoriasis vulgaris		Quantity n = 33 (%)	p-value
	<i>Candida</i> spp. Culture positive (n = 17) (%)	<i>Candida</i> spp. Culture negative (n = 16) (%)		
Psoriasis duration				0.253
- < 5 years	5 (65.2)	3 (37.5)	8 (24.2)	
- 5–10 years	5 (65.2)	3 (37.5)	8 (24.2)	
- > 10 years	7 (41.2)	10 (58.8)	17 (51.5)	
Onset of psoriasis				1.000
- < 30 years	9 (50.0)	9 (50.0)	18 (54.5)	
- ≥ 30 years	8 (53.3)	7 (46.7)	15 (45.5)	
Psoriasis severity (PASI Score)				0.789
- Mild (PASI < 5)	2 (28.6)	5 (71.4)	7 (21.2)	
- Moderate (PASI 5–10)	5 (83.3)	1 (16.7)	6 (18.2)	
- High (PASI > 10)	10 (50.0)	10 (50.0)	20 (60.6)	

PASI=Psoriasis Area and Severity Index

Table 4. The distribution of *Candida* spp. culture positivity proportion on psoriasis vulgaris group based on treatment modality

Variables	Psoriasis vulgaris		Quantity n = 33 (%)	p-value
	<i>Candida</i> spp. Culture positive (n = 17) (%)	<i>Candida</i> spp. Culture negative (n = 16) (%)		
Treatment modality				
• Systemic Methotrexate				0.708
- Yes	11 (47.8)	12 (52.2)	23 (69.7)	
- No	6 (60.0)	4 (40.0)	10 (30.3)	
• Topical Corticosteroid				1.000
- Yes	16 (51.6)	15 (48.4)	31 (93.9)	
- No	1 (50.0)	1 (50.0)	2 (6.1)	
• NB-UVB Phototherapy				0.199
- Yes	8 (40.0)	12 (60.0)	20 (60.6)	
- No	9 (69.2)	4 (30.8)	13 (39.4)	

NB-UVB=Narrowband-ultraviolet B

Table 5. Distribution of culture proportion and *Candida* spp. on psoriasis vulgaris and control groups

<i>Candida</i> spp. culture result	Psoriasis vulgaris (n = 33)	Control (n = 33)	p-value	OR
Positive	17 (51.5)	7 (21.2)	0.021	3.94
Negative	16 (48.5)	26 (78.8)		

OR=Odd ratio

Table 6. Distribution of *Candida* spp. on psoriasis vulgaris and control groups with positive culture

<i>Candida</i> spp.	Psoriasis vulgaris (n = 17) (%)	Control (n = 7) (%)
<i>C. albicans</i>	5 (29.4)	0
<i>C. krusei</i>	6 (35.3)	6 (85.7)
<i>C. glabrata</i>	1 (5.8)	0
<i>C. albicans</i> + <i>C. krusei</i>	4 (23.5)	1 (14.3)
<i>C. krusei</i> + <i>C. tropicalis</i>	1 (5.8)	0

DISCUSSION

This research found that there were more male patients (60.6%) than female patients (39.4%) with a ratio of 1.5 : 1. This is in accordance with the study of Ogawa *et al.* in 2017 in Japan, reporting that 71.5% of the participants were males and 28.5% were females.¹³ In 2018, Chiu *et al.* reported the 1: 1.4 sex ratio of males and females.¹⁴ Most of the patients were under the 46–55 years age group, which was 11 people (33.3%). The range of age in the psoriasis vulgaris group was 43.33 ± 11.96 years and 43.48 ± 11.80 years in the control group. In 2016, Chularojanamontri *et al.* reported that the mean age of the psoriasis vulgaris group was 46.0 ± 14.6 years and 45.5 ± 14.6 years in the control group.¹⁵ Ogawa *et al.* reported an increase in the distribution of psoriasis incidence persons aged 40–49 years.¹³

In this research, we found higher *Candida* spp. positive culture result in female psoriasis vulgaris patients than in the male with insignificant difference ($p = 1.000$). In 2017, Rafat *et al.* reported that 51.2% *Candida* spp. colonization in male participants and 48.7% female participants, with statistically insignificant difference based on sex.¹⁶ Furthermore, Somerville stated that *Candida* colonization is more common in females than males.¹⁷ Multiple factors, including sociodemographic patterns, economic levels, and lifestyles might affect the research results.

Based on age, the *Candida* spp. positive culture results were mostly found in the 18–25 years age group 100% and 56–65 years age group 100%, with no significant difference statistically ($p = 0.711$). Lee *et al.* reported that trans epidermal water loss (TEWL) increased in psoriasis vulgaris patients and psoriasis vulgaris patients had drier skin compared to the control.¹⁸ This indicates that the skin condition of the psoriasis vulgaris group was drier than healthy individuals. In 2017, Rafat *et al.* reported that *Candida* colonization found the most in the > 60 years age group (51.2%).¹⁶ The higher prevalence of *Candida* isolation in elderly might due to a drier skin that corresponds to pH elevation, resulting in a more alkaline skin. This decreases the ability of an impaired immune system in this age group. The low pH condition of the skin helps

to stabilize the skin's normal flora and adhere to the skin, while an alkaline environment promotes the spread and growth of pathogenic bacteria from the skin.

Psoriasis vulgaris is a chronic and residual inflammatory skin disease. The mean duration of complaints experienced by the psoriasis vulgaris group was 11.73 ± 8.73 , with the highest prevalence of complaint length was > 10 years as much as 51.5%. Positivity results of *Candida* spp. culture in psoriasis vulgaris patients were found mostly in patients with a duration of complaint < 5 years and 5–10 years (65.2%) compared to > 10 years (41.2%), but the difference between the duration of complaint in three groups was not significant ($p = 0.253$). Two studies found that oral *Candida* colonization was found more common in patients with a complaint duration of ≥ 10 years 32% and 69.4% compared to < 10 years, which were 26% and 68.6%, respectively. Also, no significant difference between the two groups ($p = 0.65$ and $p = 0.93$).^{15,19} Positivity culture of *Candida* spp. based on the age onset, mostly were ≥ 30 years (53.3%) than that of < 30 years ($p = 1.000$). Research by Bedair *et al.* found the medium of *Candida* in the mouth was more common at late onset ≥ 30 years compared to early onset < 30 years with a significant statistical difference ($p = 0.002$).¹⁹ Based on the available data, the latter observation could not be explained.

In this research, *Candida* spp. was mostly found on psoriasis vulgaris patients with moderate severity (PASI 5–10) (83.3%), compared to mild and high degrees of severity with no statistically significant difference ($p = 0.789$). Some studies have not found stronger evidence of *Candida* colonization in the oral cavity, intestine, and skin, with psoriasis ($p > 0.05$).^{9,20} Study by Sarvtin *et al.* on anti *Candida* antibodies detection reported no significant relationship between PASI score, carriage of *Candida* spp., and antibody levels in patients with psoriasis.⁹ Psoriasis Area and Severity Index was not an adequate parameter to describe the characteristic of psoriasis vulgaris patients. This led to a suggestion that the severity of psoriasis vulgaris, as measured by the PASI score, only reflects the current status of the disease. Psoriasis vulgaris is a chronic inflammatory process with acute

attacks. Therefore, the degree of severity as measured by the PASI score may not be related to the positivity of the *Candida* culture, as suspected, due to one of the causes and triggers of exacerbation of psoriasis vulgaris.

Eleven patients (47.8%) who received systemic methotrexate monotherapy had positive culture results for *Candida* spp. ($p = 0.708$), while 16 patients (51.6%) who received topical corticosteroid therapy had positive culture results for *Candida* spp. ($p = 1.000$). Eight patients (40.0%) who received NB-UVB phototherapy had positive culture results ($p = 0.199$). There was no statistically significant difference in culture positivity of *Candida* spp. based on the three types of therapy received by the patients. Several studies suggest an increase in *Candida* colonization occurs in patients with psoriasis vulgaris and atopic dermatitis. In addition to increased colonization, the risk of superficial *Candida* infections increases for psoriasis vulgaris patients. This relates to the treatment received by the patient, namely corticosteroids or new immunosuppressive agents such as tumor necrosis factor-alpha (TNF- α) or interleukin-17 (IL-17) inhibitors, because these pharmacological interventions specifically affect the antifungal immune response.¹¹ Methotrexate and topical corticosteroid therapy suppress IL-22 and IL-17, respectively, so as not to induce β -defensin production by epithelial cells. Beta-defensin is a cationic, amphiphilic antimicrobial peptide of 12-50 amino acids that acts as an antifungal that restricts the overgrowth of *Candida* colonization.¹²

Positive culture results for *Candida* spp. were found in 51.5% of the psoriasis vulgaris patients, whereas only 29.2% of the control group had positive culture results for *Candida* spp. with a significant difference of ($p = 0.021$). Sarvatin *et al.* found a positive medium of *Candida* spp. which was 15% on the skin in the psoriasis vulgaris group and 4% in the control group.⁹ In 2015, Pezeshkpoor *et al.* reported that *Candida* spp. culture positivity was low in skin swab, which was only 2%, compared to the culture positivity of saliva swab, which had high positive culture results in the psoriasis vulgaris group (46%) and 18% positive culture results in the control group.²¹

This can be caused by differences in sampling criteria. In our research, we involved patients who received either methotrexate or topical steroid therapy. Psoriasis occurs with increased activation and increased maturation of dendritic cells and keratinocytes induced by increased interferon (IFN)- γ and TNF- α as well as increased hyperproliferation and abnormal differentiation of keratinocytes induced by increased IL-23, IL-17, and IL-22 immune responses. The systemic immunosuppressive agent used to treat

psoriasis has the key immunological inhibition in lowering IL-22 levels and corticosteroids suppress IL-17 production. The suppression of IL-17 and IL-22 has an important role in the phagocytosis mechanism of *Candida* spp., especially in *C. albicans*. Besides, a highly humid environment and a higher temperature can increase *Candida* proliferation, thereby changing its nature to become a pathogen.¹²

This research also showed the odds ratio result of 3.94, where the risk of *Candida* cultures positivity on the skin of psoriasis vulgaris patients was 3.94 times greater than that of the control group. The metaanalysis study was based on several studies found that colonization of *Candida* spp. was not significant between psoriasis and control groups with odds ratio of 1.14, ($p = 0.001$).²¹ Pezeshkpoor *et al.* reported that the incidence of *Candida* in the saliva of psoriasis vulgaris patients was 2.5 times higher than that of the healthy group.¹⁰ A study by Henseler on the medical records of 44,695 dermatology patients concluded that psoriasis patients had a 1.3–1.6 higher relative risk of developing mucocutaneous candidiasis than the healthy group.²² A study on the medical records of 3,006 psoriasis patients and 1,808 atopic dermatitis patients in Kiel, Germany found that *Candida* skin infections were found in psoriasis patients more frequently than in controls. However, there was no correlation between *Candida* skin infections and detected psoriasis activity.²³

Although only a few previous studies have presented data on the colonization of *Candida* spp. in psoriasis vulgaris patients in comparison to the control group, they still show that the prevalence of colonization of *Candida* spp. in psoriasis vulgaris patients was higher than in controls. This may explain that *Candida* spp. colonization is not the only factor that may trigger and exacerbate psoriasis vulgaris but rather related to the ability of *Candida* spp. to become superantigen that can directly bound to the class II MCH, induce nonspecific T lymphocytes (variable β on TCR or T Cell Receptors (TCR) which act as antigen receptors) without antigen presenting cell (APC) intermediaries and release excess proinflammatory cytokines, triggering and aggravating the psoriasis vulgaris conditions.

This study found that *C. non albicans* was predominantly found both in psoriasis and control groups. This study also in line with a study by Ovicina-kortovic who found that the majority of *C. non albicans* composition of psoriasis vulgaris patient group and psoriasis was 18.3%.²⁴ Within the last two decades, the incidence of non *albicans* species has been increasing, especially in immunocompromised subjects.²⁵ Moreover, other factors such as prematurity, wide spectrum antibiotic consumption, and elderly were

suspected to be related to an increased incidence in non *albicans* species.²⁶

In conclusion, there was no difference in the number of *Candida* spp. colonization in the psoriasis vulgaris group and the control group. However, the culture positivity for *Candida* spp. on the skin of psoriasis vulgaris patients was higher than the control group.

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