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RESEARCH ARTICLE

Association of *Staphylococcus aureus, Streptococcus pyogenes* with the Severity of Psoriatic Iraqi Patients in Kerbala Province

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated disorder with cutaneous and systemic manifestations. The presence of a link between certain types of microorganisms and the onset of psoriasis and its worsening had been documented.

Aim of the study: The study aimed to study the association of *Staphylococcus aureus, Streptococcus pyogenes* with psoriasis and disease severity.

Method: A cross- sectional study was conducted. Thirty-one psoriatic patients have enrolled swabs (lesional skin, non-lesional skin swabs, nasal swabs, and throat swabs). The mean age of the patients was 21.13 ± 13.03. Out of 17 patients were females. The patients were classified according to PSAI score to mild, moderate, and severe cases. The time period for sample collection took about six months in an outpatient clinic, and the samples were examined in an external laboratory. All clinical details of patients were recorded according to the prepared questionnaire. The patients were divided by physician into three groups according to their PASI score: mild (PASI < 10), moderate (PASI 10–29), and severe (PASI > 30).

Swab sample: Four swabs were collected from each patient. One from throat, one from nasopharyngeal region, one from skin lesion and one from the non-lesional skin of the patients. Swabs from psoriatic lesions and non-lesional skin were used to determine *S. aureus* skin colonization. Throat and nasal swabs were used to determine carriage rates of *S. pyogenes* and *S. aureus* . The swabs were saved in a transport media until use. Bacterial culturing of the swabs was done on blood agar and pure colony for all bacteria types was prepared to be used in identification.

Result: Regarding *S. aureus* skin colonization, none of the lesional and non-lesional swabs revealed this bacteria's presence. And concerning throat and nasal carriage of this bacteria, three isolates were recovered from throat swabs (9.67%) and 4 isolates (12.9%) from a nasal swab. Psoriatic patients with positive *S. aureus* growth either in the throat or nasal swabs were fall within mild or moderate PASI. However, there is one person with severe PASI score harbor *S. aureus* in both nasal and throat swabs. Concerning Sex of psoriatic

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patients, three bacterial isolates (21.4%) were recovered from nasal swabs collected from male and three throat swab (17.6%) from female, from these, one female has both nasal and throat swabs. All of the recovered isolates from both nasal and throat swabs were collected from patients whom they have the psoriatic lesion for less than 5 years.

For *Streptococcus pyogenes*, none of the throat swabs revealed the presence of this bacteria. In order to explore the presence of previous infection with *S. pyogenes,* the ASO test was used. The mean level of ASO antibody test was (72.839 ± 66.748) and there was no presence of a significant difference in the mean serum ASO level between mild (66.1 ± 29.09), moderate (75.1 ± 14.15) and severe (78.4 ± 20.59) group of psoriasis (p =>0.715) However, the higher mean was seen in severe cases.

Bacterial identification: Colony morphology and culture characteristics was determined on blood agar and mannitol salt agar. Microscopic identification of bacteria was made by using gram staining technique. Biochemical tests was for identifying the isolated bacteria. Confirmation of identification with VITEK 2 compact system. The VITEK 2 automated system (BioMerieux, France) was for the identification of bacterial isolates. Pure culture colonies was used for bacterial identification.

INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory skin condition characterized by red, scaly plaques that most typically appear on the elbows, knees, scalp, and lower back, but it can affect any skin surface[1] regional, and country specific estimates of its prevalence. Design Systematic review and meta-analysis. Data sources Medline, Embase, Web of Science, SciELO, Korean Journal Databases, Russian Science Citation Index, WPRIM, SaudiMedLit, Informit, IndMed, and HERDIN were searched systematically from their inception dates to October 2019. Methods Studies were included if they reported on the incidence or prevalence of psoriasis in the general population. Incidence data were summarised descriptively, whereas bayesian hierarchical models were fitted to estimate the global, regional, and country specific prevalence of psoriasis. Results 41 164 records were identified and 168 studies met the inclusion criteria. In adults, the incidence of psoriasis varied from 30.3% person per years (95% confidence interval 26.6 to 34.1. The reported prevalence of this disorder ranged between 0.0 to 2.1% among children and 0.91 to 8.50% among adults, worldwide.[2]

Psoriasis has an unknown cause, the development of psoriasis is thought to be caused by a number of different factors, including genetic predisposition, environmental triggers, disruption of the skin barrier, immune dysfunction and infection, as well as microbial and complex cellular interactions. In patients with psoriasis, *Staphylococcus aureus* was found in the non-lesional skin of 27% of patients and in lesions of 46% of patients. Additionally, *S. aureus* was isolated from the skin of patients diagnosed with erythroderma.[3] Patients with psoriasis demonstrated a 4–5 fold greater risk of S. aureus colonization as compared to healthy controls. This study contrasts prior microbiome research, which reported a decreased incidence of *Staphylococcus* spp. in psoriasis patients. A subsequent investigation showed that a large proportion of *S. aureus* isolates from psoriasis patients secreted staphylococcal enterotoxins; curiously, those individuals had a much higher than the general population[4]. Patients with toxin-positive *S. aureus* in the skin had a higher Psoriasis PASI score than individuals with toxin-negative *S. aureus* in the skin or who do not have S. colonization of *S. aureus*. This has been demonstrated through clinical research[5,6].

Furthermore, for the first time, whole genome shotgun metagenomics was performed in psoriasis utilizing lesional and non-lesional samples from 28 different people. According to the data, psoriatic lesions had a greater concentration of *Staphylococcus* than non-lesional skin. Furthermore, they discovered four strains of *Staphylococcus epidermidis* that were detected in sick skin but not in healthy skin, and each of these strains had either an extracellular protein related with psoriasis or a virulence factor associated with Staphylococcus aureus. These findings highlight the necessity of adopting strain-resolving approaches to capture the strain-level variety that exists within the psoriasis-associated microbiome.[7]

Infections with *S pyogenes* are highly relevant among the environmental factors that contribute to the first onset of psoriasis or relapses in individuals predisposed to developing the condition. It's possible that more than one mechanism, like molecular mimicry or superantigens, are at play here. Many patients end up with a chronic streptococcal infection or colonization, which may be caused by the capacity of streptococci for intracellular uptake and persistence in epithelial cells. This phenomenon affects a significant number of patients[8]immune-mediated disorder with cutaneous and systemic manifestations. Genetic predisposition, environmental factors, and immune dysfunction all contribute to the pathogenesis of psoriasis with host-microbe interaction governing the progression of this disease. Emerging evidence has indicated that infection is an environmental trigger for psoriasis and plays multiple roles in its maintenance as evidenced by the frequent association between guttate psoriasis onset and acute streptococcal infection. Different infectious factors act on immune cells to produce inflammatory cytokines that can induce or aggravate psoriasis. In addition to bacterial infections, viral and fungal infections have also been shown to be strongly associated with the onset or exacerbation of psoriasis. Intervention of skin microbiota to treat psoriasis has become a hot research topic. In this review, we summarize the effects of different infectious factors (bacteria, viruses, and fungi. An infection with *S pyogenes* may play a role in the development and progression of psoriasis. IgA against *S. pyogenes* was found to be present in the plasma of patients suffering from plaque and guttate psoriasis, and its levels were found to be directly associated with a CLA+ T cell-dependent IL-17 response in an *ex vivo* model of the disease, which was found to correlate with clinical findings.[9] In addition

to this, many of these patients have elevated levels of IgG serum antibodies directed against *S pyogenes*.[10] Isolation of streptococcal organisms from the tonsils and measurement of antistreptococcal antibodies have provided supporting evidence for these observations, which have now been substantiated in several separate studies.[11]

The association of *S. aureus* and *S. pyogenes* and the disease severity were not documented among Iraqi psoriatic patients resident in Kerbala Province.

Of the Study

To Study the association of *S. aureus* infection and *S.pyogenes* with the disease severity among psoriatic patients. To determine whether *S. aureus* infection and *S. pyogenes* or carriage are associated with disease severity. To study the association of certain risk factor like sex, age, with the disease severity among psoriatic patients

RESULTS

The current study is a cross-sectional study which conducted to study the association of *S. aureus* infection and *S. pyogenes* and the severity of psoriatic disease in patients residing in Kerbala Province, Iraq. Thirty-one patients were included. Fourteen were males (45.2%) and 17 were females (54.8%). The patients' ages ranged from 2 to 49 years with a mean age of 21.13 ± 13.03. Ten patients had mild, 11 moderate, and 10 had severe PSAI scores. Four patients were smokers and one had diabetes mellitus, as shown in Table 1.

Table 1: Main features of the study population

Variation	*No.(%)*
Studied population	
Patient 31	
Sex	
Male	14 (45%)
Female	17 (55%)
Age-group (Years)	
≤15))	12 (38.7%)
(16–30)	12 (38.7%)
31–50))	7 (22.6%)
Hypertension	
Hypertensive	1 (3.33%)
Non-hypertensive	30 (96.77%)
Psoriasis score (PASI)	
<10	10(32.25%)
10–20	11(35.5%)
>30	10(32.25%)
Diabetes mellitus (DM)	
With DM	1 (3.33%)
Without DM	30 (96.77%)
Smoking	
Smokers	4 (12.9%)
Non-smokers	27 (87.1%)
Heart disease	
With Heart disease	0(0%)
Without Heart disease	31(100%)
Cancer	
With cancer	0(0%)
Without cancer	31(100%)
Psoriatic Arthritis (PsA)	
With PsA	0(0%)
Without PsA	31(100%)

Isolation and Identification of *S. aureus and S. pyogenes* in patients with psoriasis

A total of 124 clinical swabs samples were collected from patients with psoriasis during this study. For each patient, four swabs were taken.

Isolation of *Saureus* from Psoriatic Lesion and Normal skin

Out of 62 skin swabs was collected from patients (two swabs from each patient, one from psoriatic lesion and the other from normal skin area) and these swabs were used for isolation of the bacteria if present. None of these bacteria were recovered from the swab samples. Normal skin swabs revealed the presence of coagulase-negative staphylococci.

A systemic review documented that the proportion of patients with *S. aureus* on skin lesion of psoriasis varies from 0.03 to 0.64 in E of the 15 enrolled studies. While in non-lesional or healthy skin was examined in six studies and the prevalence of subjects with colonization varied between 0.06 and 0.55. The studies that included a control did not find a statistically significant difference in colonization between psoriatic patients and healthy controls.[12]

In another study, *S. aureus* has been found to be colonized in 60% of the psoriatic lesion and 60% of the bacterial isolates secrete toxins. Those patients with toxin-producing isolates have a higher psoriatic area with an increasing severity index score[8] immune-mediated disorder with cutaneous and systemic manifestations. Genetic predisposition, environmental factors, and immune dysfunction all contribute to the pathogenesis of psoriasis with host-microbe interaction governing the progression of this disease. Emerging evidence has indicated that infection is an environmental trigger for psoriasis and plays multiple roles in its maintenance as evidenced by the frequent association between guttate psoriasis onset and acute streptococcal infection. Different infectious factors act on immune cells to produce inflammatory cytokines that can induce or aggravate psoriasis. In addition to bacterial infections, viral and fungal infections have also been shown to be strongly associated with the onset or exacerbation of psoriasis. Intervention of skin microbiota to treat psoriasis has become a hot research topic. In this review, we summarize the effects of different infectious factors (bacteria, viruses, and fungi.

Furthermore, According to a systematic review, fourteen research out of twenty-one published prevalence estimates of Staphylococcal colonization in psoriatic skin lesions, including 11 comparative studies with one or more external comparison groups and three with internal (cross-body site)

comparisons. This research, the *Staphylococcal* colonization prevalence was found to be 36.6% on skin lesions of psoriatic patients and 5.1% in healthy people. Average Staphylococcal colonization risk was 4–5 times greater in psoriasis patients than in healthy people.[13]

Elfatoiki *et al*. discovered that *S. aureus* skin colonization was 3% in psoriatic skin lesions and 12.1% in non-psoriatic skin, and the author concluded that psoriasis might be a protective factor against *S. aureus* colonization.[14]

It has been shown that *S. aureus* can release a variety of exotoxins that can impact the immunological and inflammatory condition of psoriatic skin lesions. Atefi *et al.* discovered *S. aureus* in 47% of psoriasis patients and *S.* psoriatic patients have 25 times more aureus super antigens.[15] Additionally,[3] Isolated *S. aureus* from 60% of their psoriasis patients, 36% of whom produced toxin, and 12% of healthy controls, all of whom lacked *S.* Toxin from *S. aureus.*

Infected patients with *S. aureus have* worsened psoriatic lesions. This bacteria colonizes psoriatic lesions in 60% of psoriasis patients and secretes *Staphylococcal* enterotoxins and toxic shock syndrome toxin-1 in 60% of isolates (TSST-1). Patients with toxin-positive *S. aureus* in the skin have a greater psoriasis area and severity index score than those with toxin-negative *S. aureus* or no *S. aureus* colonization.[8]immune-mediated disorder with cutaneous and systemic manifestations. Genetic predisposition, environmental factors, and immune dysfunction all contribute to the pathogenesis of psoriasis with host-microbe interaction governing the progression of this disease. Emerging evidence has indicated that infection is an environmental trigger for psoriasis and plays multiple roles in its maintenance as evidenced by the frequent association between guttate psoriasis onset and acute streptococcal infection. Different infectious factors act on immune cells to produce inflammatory cytokines that can induce or aggravate psoriasis. In addition to bacterial infections, viral and fungal infections have also been shown to be strongly associated with the onset or exacerbation of psoriasis. Intervention of skin microbiota to treat psoriasis has become a hot research topic. In this review, we summarize the effects of different infectious factors (bacteria, viruses, and fungi

These differences in percentage of *S.aureus* skin colonization could be attributed to differences in study designs, sample size, geographical differences of patients involved in these studies, the immune system status of the patients, the genetic factors and also differences in environment.

The authors underlined the importance of antimicrobial peptides in human skin's innate immunity and the skin's capacity to fight bacterial infection in a research comparing *S. aureus* infection and antimicrobial peptides in patients with atopic dermatitis, psoriasis, and normal people. They said that Cathelicidins (LL-37) and defensins (HBD-2) from this antimicrobial peptides family contribute to host defense against *S. aureus.*[16]

Cathelicidins, defensins, dermcidin, ribonuclease 7 (RNase 7), psoriasin, lactoferrin, and other antimicrobial peptides (AMPs) are produced in human skin.[17] When danger is detected, the production of AMPs can either be constitutive or induced.[18] The AMPs not only have an antibacterial effect but also have an impact on the host cells. As a result, several names for AMPs have developed, such as "host defense peptides," "defensins," or "alarmins".[17] *In-vitro* at least, chemokines have been shown to inhibit the growth of bacteria, including *staphylococci.*[19]

Table 2: Numbers of *S. aureus* isolates recovered from different type of swab

Source of swab	Total No. of swab	No. of *S. aureus* isolates	
		Positive N (%)	Negative N (%)
Throat swab	31	3 (9.67)	28 (90.3)
Nasal swab	31	4 (12.9)	27 (87.09)
Lesion Swab	31	0 (0)	31 (100)
Skin swab	31	0	31 (100)

Nasal and throat carriage of *s aureus* in Psoriatic patients

Out of 62 swab samples, two swabs were collected from each patient (one from Nose and one from throat) for bacterial isolation. Three isolates were recovered from throat swabs (9.67%) and 4 isolates (12.9%) from nasal swab, (Table 2).

Elfatoiki *et al*. was found higher percentage of *S. aureus* nasal carriage (21%) in patients with psoriasis. Also, Boncompain *et al*. documented higher carriage rates in psoriatic patients (37.24%).[20,21]

Four studies documented the prevalence of nasal Staphylococcal colonization, 56, 44, 50, and 40%, in psoriatic patients and 8, 28, 34, and 42.1% in healthy individuals, respectively.[22-25] Compared to healthy controls, psoriasis patients had a 60% higher risk of nasal staphylococcal colonization[13-25].

Global variation may be seen in *S. aureus* nasal carriage among the general adult population. Carriage rates were shown to be between 24 and 25.2% in The Netherlands[26], between 27.3–27.6% in Norway[27], and 36.4% in Switzerland for Western Europe.[28] According to data from the National Health and Nutrition Examination Survey (NHANES) inside the USA, the prevalence was 30.4% between 2001 and 2004.[29] There are quite a few different carriage rates for South and Southeast Asia, including 9.1% in Indonesia,[30] 14.8% in Pakistan,[31] 23.4% in Malaysia,[32] 24.1% in Taiwan,[33] and 29.4% in India.[34]

In a Swiss study of risk factors for *S. aureus* throat carriage rate was 30.2% in community members of the population, whereas hospitalized patients and healthcare workers made up 18.4%.[28] In another study from Mexico, *S. aureus* was isolated more frequently from the throat (46.5%) than the nose (37.1%) from healthy people.[35]

According to a study by Kotpal *et al*., 35% of individuals had *S. aureus* found in their throat or anterior nares. *S aureus* was found in the throats of 4 out of 50 (8%) HIV-positive individuals and the nares of 22 (44%) HIV-positive individuals. None of the HIV-negative individuals had *S. aureus* in their throats, but 22% of them had it in their nasal cavity.[36]

Table 3: The differences in swab type of *S. aureus* between different psoriasis score (PASI) in the patient group

specimens types	*S. aureus* isolated from patients			
	Mild (<10)	Moderate (10-20)	Severe (>30)	Total (%)
Throat swab	1 (3.23%) a	1 (3.23%) a	1 (3.23%) a	3 (9.7)
Nasal swab	2 (6.45%) a	1 (3.23%) a	1(3.23%) a	4 (12.9)
Lesion Swab	0 a	0 a	0 a	0 (0)
Skin swab	0 (0%) a	0 (0%) a	0 (0%) a	0 (0)

Table 4: Distribution of *S. aureus* isolated in patients with psoriasis according to age groups

Age group (Year)	Patient		Total (N) (%)	*p*-value
	Positive for *S. aureus* (N)	Negative for *S. aureus* (N)		
≤ 15	3 (9.6%)	9 (29%)	12 (38.7)	
16–30	2 (6.5%)	10 (32.3%)	12 (38.7)	0.809
31–50	2 (6.5%)	5 (16.1%)	7 (22.6)	
Total	7 (22.6%)	24 (77.4)	31 (100)	

$p \leq 0.05$ was considered statistically significant

Association of *S. aurues* Carriage with Disease Severity and Site of the Skin Lesion

Psoriatic patients with positive *S. aureus* growth either in throat or nasal swabs were fall within mild or moderate PASI. However, one person with a severe PASI score harbor *S. aureus* in both nasal and throat swabs, as shown in Table 3. also, there were no significant differences between swab type of *S. aureus* among psoriatic patients bases on PASI scores.

Similarly, Boncompain *et al.,* nasal carriage of *S aureus* were more frequent in moderate and severe cases.[21]

Regarding the site of psoriatic lesion, five (71.4%) from 7 isolates were recovered from head region in patients with mild to moderate PASI score. Whereas, the other two isolates were recovered from the patient who had the lesion on all body sites and had a severe PASI score.

There is proof that the peptidoglycan of *S. aureus* suppresses apoptosis and influences keratinocyte growth.[37] Furthermore, staphylococcal antigens influence inflammatory mediators such as interleukin (IL)-22, which inhibits keratinocyte terminal differentiation.[38]

Association of *S. aureus* Carriage with Age and Sex

As shown in Tables 4 and 5, no significant differences were found between age group and the presence of *S. aureus* in patients with psoriasis (p=0.809). Regarding the sex of psoriatic patients, three bacterial isolates (21.4%) were recovered from nasal swabs collected from male and three throat swab (17.6%) from female, one female has both nasal and throat swabs (Table 6).

Boncompain *et al*., found that the nasal carriage of *S. aureus* was associated to male patients and with adult patients.[21]

Ungureanu *et al*., found that *S. aureus* nasal colonization in males (40.12%) had a slightly greater prevalence than in females.[39]

Greater population-based cross-sectional studies have revealed that men are more likely than women to carry *S. aureus* in their nasal passages.[40-42]

Nilsson and Ripa, revealed that no significant differences between men and women concerning isolation rates from the throat and anterior nares sites were found.[43]

Association of *S. aureus* carriage with Age of Psoriasis

All of the recovered isolates from both nasal (33.4%) and throat (25%) swabs were collected from patients whom they have the psoriatic lesion for less than 5 years.

Isolation of *S. pyogenes* from Throat Swabs

Out of 31 throat swabs taken from psoriatic patients was used for isolation of *S. pyogenes.* None of these swabs revealed the presence of this bacteria.

Antistreptolysin - O Serum Level in Psoriatic Patients

In order to explore the presence of the previous infection with *S. pyogenes,* the same blood samples taken from psoriatic patients were used for detecting H pylori antibody, wwereas used for determining the level of anti-streptolysin O antibody. The mean level of ASO antibody test was (72.839 ± 66.748).

Data from the this study reveal that were no present significant differences in the mean serum ASO level between mild (66.1 ± 29.09), moderate (75.1 ± 14.15) and severe (78.4 ± 20.59) group of psoriasis (p =0.715), as shown in Figure 1. Upon analysis of results, we found no correlation between serum ASO level and occurrence of psoriasis, and anti-streptolysin O titer (ASO) is still within normal range (>200 Ul/mL).

Rasi and Pour-Heidari, Cassandra *et al*., and Ibrahimbaş *et al*. all conducted studies demonstrating no discernible difference between the serum ASO levels of psoriasis patients and controls.[44-46]

According to Al-Hassnawi *et al.,* patients with chronic plaque-type psoriasis have significantly higher serum ASO levels than the healthy control group (*p-value* 0.05), and they

Table 5: Distribution of *S. aureus* isolated recovered from throat and nasal swab in patients with psoriasis according to sex

Gender	Throat swab			Nasal swab		
	Negative	Positive	Total	Negative	Positive	Total
Male (%)	14 (100)	0 (0)	14 (100)	11 (78.6)	3 (21.4)	14 (100)
Female (%)	14 (82.4)	3 (17.6)	17 (100)	16 (94.1)	1 (5.9)	17 (100)
Total (%)	28 (90.33)	3 (9.67)	31 (100)	27 (87.1)	4 (12.9)	31 (100)

Table 6: Distribution of *S. aureus* isolated recovered from throat and nasal swab in patients with psoriasis according to age of psoriasis

Age of psoriasis	Throat swab			Nasal swab		
	Negative (%)	Positive (%)	Total (%)	Negative (%)	Positive (%)	Total (%)
Less than 5 years	9 (75)	3 (25)	12 (100)	8 (66.7)	4 (33.4)	12 (100)
Less than 10 years	8 (100)	0 (0)	8 (100)	8 (100)	0 (0)	8 (100)
More than 10 years	11 (100)	0 (0)	11 (100)	11 (100)	0 (0)	11 (100)
Total	28 (90.33)	3 (9.67)	31 (100)	27 (87.1)	4 (12.9)	31 (100)

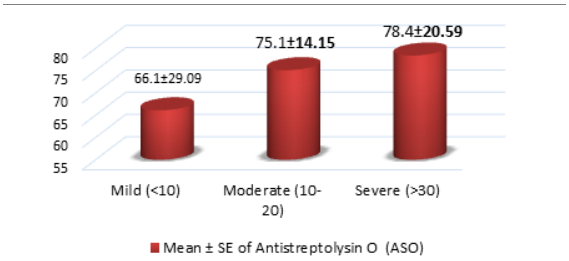

Figure 1: The differences in means of Antistreptolysin O (ASO) between different psoriasis scores (PASI) in the patient group

speculate that this may be related to the immunopathogenesis and/or susceptibility to this form of psoriasis.[47]

CONCLUSION

None of the psoriatic lesion and non-psoriatic skin swabs revealed the presence *S. aureus,* carriage rates was 9.67% in throat and 12.9% in the nasopharyngeal region. Positive *S. aureus* growth either from throat or nasal swabs fall within mild or moderate PASI. However, with severe PASI score, *S. aureus* was found in both nasal and throat swabs. There were no significant differences between age group and the presence of *S. aureus* in patients with psoriasis. None of the throat swabs revealed the presence of *S. pyogenes*. No significant difference in the mean serum ASO level between a mild, moderate and severe group of psoriasis, higher serum ASO level was seen with severe PASI score.

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