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Original Article

# Association between streptococcal throat infection and psoriasis in Bangladesh

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**Abstract**

***Objective*** To assess the association between streptococcal throat infection and psoriasis.

***Patients and methods*** Total one hundred fourteen (114) patients attending at outpatient and inpatient department of Dermatology and Venereology, Bangabandhu Sheikh Mujib Medical University, Dhaka were selected for our study. Patients clinically and histopathologically diagnosed as psoriasis were selected as case group and patients with skin diseases other than psoriasis were selected as control group.

***Results*** In our study, mean age was 30.73±14.69 years in cases and 26.47±12.64 years in controls. Majority of respondents were diagnosed as chronic plaque psoriasis (51%) among acute guttate psoriasis, guttate flare of chronic plaque psoriasis, deterioration of chronic plaque psoriasis and pustular psoriasis. 19 (30%) patients of psoriasis had sore throat clinically. ASO titer was raised in 65% psoriatics and 19.6% non-psoriatic controls. Majority of cases of guttate psoriasis (86.6%) had raised ASO titer versus 27.2% respective controls. Normal flora was the maximum finding in two groups and *Streptococcus pyogenes* was the second highest in frequency in both sides. In throat swab culture results of two groups, 15 (23.8%) psoriasis patients and 5 (9.8%) non-psoriatic controls were found positive for *S pyogenes*.

***Conclusion*** Streptococcal throat infection can cause provocation of psoriasis. So, psoriasis patients should be encouraged to report sore throat to their physician and early treatment of streptococcal throat infections might be beneficial in psoriasis.

***Key words***
Streptococcal infection, association, psoriasis.

## Introduction

Psoriasis is a common, chronic, inflammatory disease of the skin characterized by round, circumscribed, erythematous, dry, scaling plaques of varying sizes and covered by grayish white or silvery white, imbricated and lamellar scales, with corresponding microscopic (histological) skin alterations. It leads to considerable impairment of the quality of life of the affected patients.[1,2] Psoriasis is universal in occurrence. However its prevalence in different populations varies from 0.1% to 11.85% according to published reports.[3] The etiopathogenesis of the disease is still largely unknown but studies indicate that it is caused by an interaction of multiple genetic components and environmental factors including β-hemolytic streptococci.[4] There is now increasing evidence for the involvement of T-cell dysregulation in

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the development and maintenance of the psoriatic phenotype. It is known that pronounced parakeratosis is associated with the presence of neutrophils and that microabscesses of Munro are located exclusively within areas of parakeratosis.[3] Therefore, neutrophils are considered to play an important role in the development of psoriasis.[5] A high incidence of β-hemolytic streptococcal throat infections as the main trigger of first psoriasis exacerbation favours streptococcal antigens as causative agents that may induce cross reactive T cell responses against skin components. In this respect, psoriasis behaves like rheumatic fever, which follows upper respiratory tract infections with *Streptococcus pyogenes*.[6] As M protein is a major pathogenic surface antigen of streptococci (β-hemolytic streptococci), the cross-reactivity between streptococcal M protein surface antigens and human epidermis was investigated and proved.[7] There is a strong association between prior infection with *S pyogenes* (β-hemolytic streptococcus) and psoriasis, which was proved by a history of an acute sore throat 1 to 3 weeks before the eruption and bacteriological (culture of throat swabs) and serological (ASO titer) evidence of recent streptococcal infection.[8] Unfortunately, it is harder to eliminate the streptococcal carrier state than it is to treat an acute streptococcal infection.[9,10]

Currently there is no cure for psoriasis and treatment options produce variable response, in part, because disease pathogenesis is not completely understood.[11] Moreover, treatment of psoriasis and maintenance of its remission is still challenging. In Bangladesh several studies were done on psoriasis and no data about association between streptococcal throat infection (sore throat) and psoriasis are available so far. Considering the co-morbidities and sufferings of psoriasis patients, it might be justifiable to find out this treatable or modifiable event that provokes the initiation, exacerbation or maintenance of psoriatic disease process in order to control that one.

## Patients and methods

It was a cross-sectional study and carried out from February 2012 to October 2012 in the department of Dermatology and Venereology BSMMU, Shahbag, Dhaka. Patients with psoriasis attending in outpatient department were selected as cases and non-psoriatic patients as control. Consecutive type of non-probability sampling technique was followed in this study. Inclusion criteria were all types of psoriasis patients, diagnosed clinically and histopathologically (as case), patients of both sexes and all ages, patients who gave written consent and controls were non psoriasis patient (age matched within±5years with case). Patients on systemic anti-psoriatic drugs and/or systemic antibiotics currently and minimum within previous15 days from interviewing were excluded.

### *Study procedure*

63 recruited psoriasis patients were allocated to one of five groups: 1a, acute guttate psoriasis (no psoriasis present when eruption began) [n=15]; 1b, guttate flare of chronic psoriasis (n=14]; 2a, chronic plaque psoriasis (including scalp psoriasis) [n=26]; 2b, sudden exacerbation/ deterioration of chronic plaque psoriasis (n=7); 3, Other types of psoriasis (small plaque/ atypical guttate/ pustular/ inverse psoriasis) [n=1]. Similarly, 51 controls were distributed as, 11 for acute guttate psoriasis, 10 for guttate flare of chronic plaque psoriasis, 4 for deterioration of chronic plaque psoriasis, 25 for chronic plaque psoriasis and 1 for pustular psoriasis.

After selection, both case and control were asked to give detail personal and family history of skin disease, age of onset of 1st psoriasis, duration of present psoriatic skin lesion, current and within previous 15 days drug history (about systemic anti-psoriatic drugs and antibiotics), the presence or absence of sore throat, date of last attack of sore throat, frequency of sore throat per year and other infections; whether psoriatic lesions exacerbate/deteriorate suddenly after sore throat and any history of rheumatic fever or carditis, AGN (nephritis) and erythema nodosum. Clinical examination of skin, pharynx, tonsils and lymph nodes was also performed. Throat swab was taken and blood sample was collected from patients with psoriasis (case) and non-psoriatic controls. For histopathological confirmation of psoriasis we performed skin biopsy from lesional area of each psoriasis patient for histopathological examination.

Swabs were collected with sterile cotton swab (15 cm long) and venous blood was drawn with sterile syringe. Collected throat swabs were inoculated on blood agar media containing plate and incubated at 37ºC for 48 hours. The pathogenic organisms were identified by gross colony morphology and types of hemolysis (yellowish halo around the colony – beta hemolysis or turbid halo with greenish cast around colony – alpha hemolysis) on culture media. β-hemolysis indicated growth of *S pyogenes*. The level of anti-streptococcal antibody – like antistreptolysin "O" (ASO titer) was measured with BN ProSpec, SIEMENS, USA®. ASO titer ≥200 IU/ ml was considered as evidence of recent streptococcal infections.

### *Analysis of data*

Statistical analysis was done by using SPSS program (version 16). Test of association between bacteriological and serological findings and clinical presentation of psoriasis has been carried out by $\chi 2$ (chi square) test. Statistical significance was set at 5% level and confidence interval at 95% level.

## Results

63 patients were enrolled as psoriatic cases and 51 patients as control. Mean age was 30.73±14.69 years in cases and 26.47±12.64 in control ($P>0.05$). The age range of respondents was 57 years (8-65) in case and 56 years (4-60) in control group (**Table 1**). Majority of respondents were diagnosed as chronic plaque psoriasis 26 (41%) followed by acute guttate psoriasis 15 (24%), guttate flare of chronic plaque psoriasis 14 (22%), deterioration of chronic plaque psoriasis 7 (11%) and pustular psoriasis 1 (2%). 19 (30%) patients of psoriasis were present with sore throat.

Comparison of ASO titer in case and control groups is shown in **Table 2**. ASO titer was raised in 65% psoriaticss and 19.6% non-psoriatic controls ($P<0.05$). Majority of cases of guttate psoriasis (86.6%) had raised ASO titer versus 27.2% in respective controls. Chronic plaque psoriasis stood on next. There was significant difference in ASO titer status between two groups.

**Table 1** Distribution of respondents according to age.

| | *Cases* *N (%)* | *Controls* *N (%)* |
|---|---|---|
| Age groups (years) | | |
| 0-9 | 3 (4.8) | 3 (5.9) |
| 10-19 | 13 (20.3) | 9 (17.6) |
| 20-29 | 14 (22.2) | 22 (43.1) |
| 30-39 | 12 (19) | 9 (17.6) |
| 40-49 | 14 (22.2) | 4 (7.4) |
| 50-59 | 4 (6.3) | 3 (5.9) |
| >60 | 3 (4.8) | 1(2) |
| Mean age (years) | 30.73±14.69 | 26.47±12.64 |
| Min-max (years) | 8-65 | 4-60 |
| Range (years) | 57 | 56 |

*P* value obtained from $\chi^2$ test was non-significant.

**Table 2** Raised ASO titer in two groups.

| *Type of psoriasis* | *Case (n=63)* | | *Control (n=51)* | | |
|---|---|---|---|---|---|
| | *ASO titer raised (≥200 IU/ml)* | *ASO titer within normal limit (<200 IU/ml)* | *ASO titer raised (≥200 IU/ml)* | *ASO titer within normal limit (<200 IU/ml)* | *P value* |
| Acute guttate psoriasis | 13 (86.6%) | 2 | 3 (27.2%) | 8 | |
| Guttate flare of chronic plaque psoriasis | 11 (78.5%) | 3 | 2 (20%) | 8 | |
| Chronic plaque psoriasis | 12 (46.1%) | 14 | 4 (16%) | 21 | <0.05* |
| Deterioration of chronic plaque psoriasis | 5 (71.4%) | 2 | 1 (33.3%) | 3 | |
| Pustular psoriasis | 0 | 1 | 0 | 1 | |
| Total | 41 (65%) | 22 | 10 (19.6%) | 41 | |

*P* value obtained from $\chi^2$ test,* Significant

**Table 3** Results of throat swab culture in different types of psoriasis.

| *Type of psoriasis* | *Case (n=63)* *Culture result* | | | *Control (n=51)* *Culture result* | | |
|---|---|---|---|---|---|---|
| | *Streptococcus pyogenes* | *Normal flora* | *Staphylococcus/ Klebsiella spp* | *Streptococcus pyogenes* | *Normal flora* | *Staphylococcus / Klebsiella spp* |
| Acute guttate psoriasis | 5 (33.3%) | 10 (66.6%) | 0 | 1 (9.1) | 10 (90.9%) | 0 |
| Guttate flare of chronic plaque psoriasis | 3 (21.4%) | 11 (78.6%) | 0 | 1 (10%) | 9 (90%) | 0 |
| Chronic plaque psoriasis | 4 (15.3%) | 21 (80.8%) | 1 (3.8%) † | 2 (8%) | 23(92%) | 0 |
| Deterioration of chronic plaque psoriasis | 3 (42.8%) | 3 (42.8%) | 1 (14.2%) ‡ | 1 (25%) | 3 (75%) | 0 |
| Pustular psoriasis | 0 | 1 (100%) | 0 | 0 | 0 | 1 (100%) † |

† Klebsiella spp, ‡*Staphylococcus aureus*

**Table 4** *Streptococcus pyogenes* present in cases and controls.

| *Streptococcus pyogenes* | *Case (n=63) N (%)* | *Control (n=51) N (%)* |
|---|---|---|
| Present | 15 (23.8) | 5 (9.8) |
| Absent | 48 (76.2) | 46 (90.2) |

*P* value obtained from $\chi^2$ test, <0.05, significant

**Table 3** shows the throat swab culture results in different types of psoriasis and respective controls. The frequency of *S pyogenes*, normal flora, *Staphylococcus aureus* and *Klebsiella* species in two groups was shown in **Table 3**. Normal flora was the maximum findings in two groups and *S pyogenes* was the second highest in frequency in both sides. The frequency of normal flora was almost similar in two groups but visible difference was seen in case of *S pyogenes* (5/15) in psoriatic and non-psoriatic group.

**Table 4** shows that 15 (23.8%) psoriasis patients and 5 (9.8%) non-psoriatic controls were found positive for *S pyogenes*. The difference was significant in two groups (*P*<0.05).

## Discussion

63 cases were categorized into 5 types of psoriasis along with respective controls for comparison. Similar study was conducted by Telfar *et al*.[8] with 111 patients with psoriasis, where they found 60% cases as chronic plaque psoriasis, 30% as acute guttate psoriasis and 10% as others psoriasis. Clinically sore throat was present in 19 (30.15%) cases and number of attack (sore throat) per year was 5 times (mean) in cases and 3 times (mean) in controls. 66.6% patients gave a history of sore throat within 3 weeks period prior to the appearance of acute guttate psoriasis (AGP) and 64.2% patients gave a history of sore throat within 4 weeks period prior to the appearance of guttate flare of chronic plaque psoriasis. In two different studies the history of sore throat preceding the onset of acute guttate psoriasis within 3 weeks period was 77.1% and 39%.[12,8] Results of our study stood between these two results.

In our study, 65% psoriasis patients were detected with raised ASO titer whereas it was raised in 19.6% control patients ($p$<0.05). Serologic evidence of recent streptococcal infections (raised ASO titer) was found in 18 (56%) of 32 patients with 31% had a history of sore throat 1 to 3 weeks before the appearance of rash and 17 (85%) of 20 patients with AGP (acute guttate psoriasis) in studies reported by Mukherjee *et al*.[13] In a cross sectional study with 111 patients, ASO titer was raised 43% in total, 58% in AGP and 26% in guttate exacerbation of chronic plaque psoriasis.[8] Naqqash *et al*.[12] found ASO titer raised 60% in AGP versus 6.6% in control. In a prospective study, Gudjohnson *et al*.[4] described ASO titer status raised 10 times than their controls in chronic plaque psoriasis. All these studies showed significant differences ($p$<0.05) with their respective controls. The outcome of above mentioned studies were similar and also supportive for our findings.

In throat swab culture of all psoriasis patients, we isolated *S pyogenes* 15 (23.8%), normal flora in 46 (73%), *S aureus* 1 (1.6%) and *Klebsiella* spp. 1 (1.59%) and in controls *S pyogenes* in 5 (9.8%), normal flora in 45 (88.2%) and *Klebsiella* spp. in 1 (2%), respectively. *S pyogenes* was found 5 (33.3%) case of AGP versus 1 (9.1%) in their controls, 10 (21.21%) in chronic plaque psoriasis versus 4 (10.2%) in controls. In total *S pyogenes* was isolated from 15 (23.8%) psoriatic patients and 5 (9.8%) controls ($p$<0.05). *S pyogenes* was isolated from 26% in AGP, 13% in guttate flare of chronic plaque psoriasis, 14% in chronic plaque psoriasis and 6% in total controls in a study on 111 psoriasis patients.[8] In another study by Naqqash *et al*.[12] *S pyogenes* was isolated in 34% cases of chronic plaque psoriasis and 97% in AGP. Gudjohnson *et al.*[4] showed *S pyogenes* positive in throat swab culture was 9.1% in chronic plaque psoriasis versus 0.9% in controls ($p$<0.05). Keeping these studies in mind, and the strong association documented in our study, between acute guttate psoriasis, guttate flare of chronic plaque psoriasis and chronic plaque psoriasis in terms of evidence of recent streptococcal throat infections (raised ASO titer and positive throat swab culture), it is important to search for and eliminate microbial infections in the treatment of psoriasis.

## Conclusion

It can be concluded that *S pyogenes* (group A β-hemolytic streptococcus) throat infections can cause provocation of guttate psoriasis, guttate flare of chronic plaque psoriasis and as a whole exacerbation and maintenance of chronic plaque psoriasis.

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