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ORIGINAL ARTICLE

Association of Elevated ASO Titer with Plaque and Guttate Psoriasis

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ABSTRACT

Objectives: To determine the association of elevated ASO titer with plaque and guttate psoriasis.
Study Design: Case control study.
Place and Duration: Department of Dermatology, Shaheed Mohtrama Benazir Bhutto Medical University (SMBBMU), Larkana during from 22nd January 2018 to 21st July 2018
Materials & Methods: A total of 30 patients with plaque or guttate psoriasis and 30 controls, 20 to 60 years of age of either gender were included. Patients taking systemic anti-psoriatic drugs, CRF and erythroderma were excluded. After this throat swabs were taken and pathogenic organism was identified by gross colony morphology and types of hemolysis on culture media. β-hemolysis indicated growth of Streptococcus pyogens. Antistreptolysin O titer (ASO titer) was measured and titer >200 IU/ml was taken as elevated.
Results: Mean age was 40.92 ± 10.62 years. Out of 60 patients, 40 (66.67%) were males and 20 (33.33%) were females with male to female ratio of 2:1. Frequency of elevated ASO titer in patients with plaque and guttate psoriasis was found in 13 (43.33%) and in 04 (13.33%) controls with p-value of 0.014 and odds ratio of 2.97.
Conclusion: This study concluded that concluded that there is positive association of elevated ASO titer with plaque and guttate psoriasis.
Keywords: Psoriasis, Throat Infection, ASO Titer

INTRODUCTION

Psoriasis is an immune-mediated, chronic, inflammatory skin disease including red, scaly patches, papules, and plaques, which usually itch. The causes of psoriasis are not fully understood. It is not purely a skin disorder and can have a negative impact on many organ systems. Psoriasis has been associated with an increased risk of certain cancers, cardiovascular disease, and other immune-mediated disorders such as Crohn's disease and ulcerative colitis. It is generally considered agenetic disease, thought to be triggered or influenced by environmental factors [1]. The skin lesions seen in psoriasis may vary in severity from minor localized patches to complete body coverage [2, 3]. It usually affects approximately 2% of the world's population [4]. The five main types of psoriasis are plaque, guttate, inverse, pustular, and erythrodermic. The most common form is plaque psoriasis, which appears as sharply demarcated, erythematous areas covered with silvery-white scale [5]. Although not life-threatening or contagious, psoriasis substantially affects health-related quality of life (HRQoL) and has negative psychological and social implications [6]. The pathogenesis of psoriasis is quite complex, but there is compelling evidence that overproduction of proinflammatory cytokines by T cells and keratinocytes, including tumor necrosis factor (TNF), plays a very important role [7-8]. TNF-α is found in increased concentrations in the joints and skin of patients with rheumatoid arthritis, psoriatic arthritis and psoriasis [9].

There is evidence that an immunologic mechanism is involved in the triggering of psoriasis by Streptococcal infections. Stimulation of T cells by Streptococcal superantigen causes release of immune cytokines such as IL-2, which are important in the pathogenesis of psoriasis [10, 11]. There is a strong association between prior infection with streptococcus pyogenes (b-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 [12, 13]. In a case control study, elevated ASO titer was seen in 86.6% patients with guttate psoriasis versus 27.2% in control group. Similarly, elevated ASO titer was seen in 46.1% patients with plaque psoriasis versus 16.0% in control group [14]. The purpose of the present study was to determine the association of elevated ASO titer with plaque and guttate psoriasis.

MATERIALS AND METHODS

The study design was Case-control study and study setting was at Department of Dermatology, Shaheed Mohtrama Benazir Bhutto Medical University (SMBBMU) Larkana. The study duration was 22nd January 2018 to 21st July 2018. The calculated sample size was 60 i.e. 30 in each group with 5% margin of error, 80% power of study, taking elevated ASO titer as 46.1% in patients with plaque psoriasis versus 16.0% in control group.[14] Non-probability, consecutive sampling was the technique adopted. After permission from the ethical review committee, total number of 60 patients presented to Department of Dermatology, Shaheed Mohtrama Benazir Bhutto Medical University Larkana, fulfilling the Inclusion criteria were selected. Informed written consent was taken from each patient. All patients with psoriasis were taken as case group and non-psoriatic patients with matched ages and gender were taken as control group. After this throat swabs were taken and pathogenic organism was identified by gross colony morphology and types of hemolysis on culture media. β-hemolysis indicated growth of Streptococcus pyogens. Antistreptolysin O titer (ASO titer) was measured and titer >200 IU/ml was taken as elevated. All this data was recorded on a specially designed proforma. Statistical analysis was performed using SPSS version 20. Results were presented as mean and standard deviation for quantitative variables i.e. age and duration of disease. Frequency and percentage were calculated for qualitative variable like family h/o psoriasis (yes/no), place of living (rural/urban), occupation (domestic/office/filed), type of psoriasis (plaque/guttate) and elevated ASO titer (yes/no). The elevated ASO titer of the two study groups were compared for difference. Chi Square was applied to compare the frequency of elevated ASO titer. P value ≤0.05 was considered as significant. Odds ratio was also calculated to see the strength of association between elevated ASO titer and psoriasis and OR >1 was considered as significant. Effect modifiers like age, duration of disease, family h/o psoriasis (yes/no), place of living (rural/urban), occupation (domestic/office/filed) and type of psoriasis (plaque/guttate) were controlled through stratification and post-stratification chi square was applied to see their effect on elevated ASO titer.

RESULTS

Age range in this study was from 20 to 60 years with mean age of 40.92 ± 10.62 years. The mean age of women in group A was

41.07 ± 11.65 years and in group B was 40.77 ± 9.69 years . Out of 60 patients, 40 (66.67%) were males and 20 (33.33%) were females with male to female ratio of 2:1. Mean duration of disease was 4.50 ± 1.47 months. Distribution of family h/o psoriasis, place of living, occupation and type of psoriasis is shown in Table I and II respectively.

Frequency of elevated ASO titer in patients with plaque and guttate psoriasis was found in 13 (43.33%) and in 04 (13.33%) controls with p-value of 0.014 and odds ratio of 2.97as shown in Table III which showed positive association between elevated ASO titer with plaque and guttate psoriasis.

Stratification of elevated ASO titer with respect to age groups , gender duration of diseaseis in both groups is shown in Table IV . Stratification of elevated ASO titer with respect to family h/o psoriasis and place of livings is shown in Table V. Stratification of elevated ASO titer with respect to occupation and type of psoriasis is shown in Table VI

Table 1: Distribution of family h/o psoriasis (n=60) and Place of living for both groups (n=60).

Case / Control Group	Domestic		Place of living (n=60).	
	Yes	No	Rural	Urban
Case group No. of patients (n=30)	13 (43.33%)	17 (56.67%)	14 (46.67%)	16 (53.33%)
Control group No. of patients (n=30)	13 (43.33%)	17 (56.67%)	16 (53.33%)	14 (46.67%)
Total (n=60)	26(43.33%)	34(56.67%)	30(50%)	30(50%)

Table 2: Distribution of occupation and type of psoriasis for both groups (n=60).

Occupation / type of psoriasis	Case group (n=30)		Control group (n=30)		Total (n=60)	
	No. of patients	%age	No. of patients	%age	No. of patients	%age
Domestic	17	56.67	11	36.67	28	46.67
Office	08	26.67	08	26.67	16	53.33
Field	05	16.67	11	36.67	16	53.33
Plague	15	50.0	17	56.67	32	53.33
Guttate	15	50.0	13	43.33	28	46.67

Table 3: Comparison of frequency of elevated ASO titer between patients with plaque and guttate psoriasis versus controls (n=60).

		Case group (n=30)		Control group (n=30)	
		No. of Patients	%age	No. of Patients	%age
Elevated Aso Titer	Yes	13	43.33	04	13.33
	No	17	56.67	26	86.67

- P value is 0.014 which is statistically significant.
- Odds ratio is 2.97 which is significant.

Table 4: Stratification of elevated ASO titer with respect to age groups ,gender and duration

Age (years)/ gender and duration	Case group (n=30)		Control group (n=30)		p-value	OR
	Elevated ASO titer		Elevated ASO titer			
	Yes	No	Yes	No		
20-40	07	07	02	10	0.087	5.00
41-60	06	10	02	16	0.085	4.80
Male	08	11	03	18	0.058	4.36
Female	05	06	01	08	0.120	6.67
≤5	11	11	03	17	0.022	5.67
>5	02	06	01	10	0.365	3.33

Table 5: Stratification of elevated ASO titer with respect to family h/o psoriasis and place of living.

Family h/o psoriasis/ place of living	Case group (n=30)		Control group (n=30)		p-value	OR
	Elevated ASO titer		Elevated ASO titer			
	Yes	No	Yes	No		
Yes	06	07	01	12	0.048	10.3
No	07	10	03	14	0.141	3.27
Rural	06	08	03	13	0.156	3.25
Urban	07	09	01	13	0.045	10.1

Table 6: Stratification of elevated ASO titer with respect to occupation and type of psoriasis..

Occupation/ type of psoriasis	Case group (n=30)		Control group (n=30)		p-value	OR
	Elevated ASO titer		Elevated ASO titer			
	Yes	No	Yes	No		
Domestic	08	09	00	11	0.047	20.6
Office	03	05	01	07	0.268	4.20
Field	02	03	03	08	0.613	1.78
Plaque	08	07	03	14	0.041	5.33
Guttate	05	10	01	12	0.128	6.00

DISCUSSION

Psoriasis is universal in occurrence. However its prevalence in different populations varies from 0.1% to 11.85% according to published reports [15]. 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 [16].

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 [17]. 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 [18]. 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 [19]. Unfortunately, it is harder to eliminate the streptococcal carrier state than it is to treat an acute streptococcal infection [20, 21].

This study was conducted to determine the association of elevated ASO titer with plaque and guttate psoriasis. In this study, frequency of elevated ASO titer in patients with plaque and guttate psoriasis was found in 13 (43.33%) and in 04 (13.33%) controls with p-value of 0.014 and odds ratio of 2.97.In a case control study, elevated ASO titer was seen in 86.6% patients with guttate psoriasis versus 27.2% in control group. Similarly, elevated ASO titer was seen in 46.1% patients with plaque psoriasis versus 16.0% in control group [14].

In this study, chronic plaque psoriasis patients were shown to have significantly higher ASO level compared to the control group ($P < 0.05$). These findings are consistent with many previous studies [22]. All these studies showed significant differences ($P < 0.05$) with their respective controls. In a previous study, association between streptococcal infections and psoriasis was suggested in 18(56%) of the 32 patients, 31% had a history of sore throat 1 to 3 weeks before the appearance of rash and 17(85%) of the 20 patients with acute guttate psoriasis (AGP) [22].

Increased levels of antibodies to streptolysin O were also found in the serum of patients in response to infection with haemolytic streptococcus groups A, C or G. Streptococcal infection usually leads to eruptive guttate psoriasis, and deterioration of other clinical forms of psoriasis [23]. Another study found ASO titre raised in 60% of AGP patients versus 6.6% in healthy control group [24]. Another prospective cohort study described ASO serum level increased 10 times compared with their controls in chronic plaque psoriasis [25].

In a retrospective cohort, about 30% of patients with chronic psoriasis stated that they had noted worsening of their disease in association with sore throat [26]. An exacerbation of chronic plaque psoriasis only if streptococci were isolated 4 days or later after the onset of sore throat was observed [27]. In addition, some studies proposed that the psoriasis patients with high ASO titres

had guttate psoriasis more frequently compared with patients with normal ASO titres [28].

CONCLUSION

This study concluded that there is positive association of elevated ASO titer with plaque and guttate psoriasis. So, we recommend that there should be early identification and management of sore throat infections in order to prevent the incidence of psoriasis as well as reducing the progression of psoriasis.

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