Original Article:

Relationship of Serum High Sensitivity C - reactive Protein Level with Severity of the Disease in Patients with Psoriasis According to Psoriasis Area and Severity Index

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Abstract

Background: Psoriasis is an inflammatory disease of the skin of multifactorial aetiology. Psoriasis has been considered mainly to be a Th1-driven autoimmune disease defined by a cytokine pattern consisting of IFN-ß, tumour necrosis factor-alpha (TNF-a), IL-1, IL-2, IL-3, IL-6, IL-8, epidermal growth factor and transforming growth factor-alpha (TGF-α). However recent findings have revealed a potential role for IL-23 and Th17 responses in the pathogenesis of psoriasis. C-reactive protein(CRP) is important for psoriasis due to its relation with cytokines that are responsible for skin inflammation. **Objectives:** This study measured the serum hsCRP level in patients with psoriasis and assessed its relationship with the severity of the disease. **Materials and Method:** This cross-sectional study included 52 psoriatic patients, diagnosed in the Department of Dermatology & Venereology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh The study was conducted in two years period, from July 2018 to June 2020 and purposive type of sampling technique was applied to collect the sample from the study population.. Disease severity was measured by the Psoriasis Area Severity Index (PASI). **Results:** The mean serum hsCRP level was 6.29±6.8 and the mean Psoriasis Area and Severity Index (PASI) was 10.07±5.88. The mean hsCRP in mild psoriasis (PASI≤10) was 1.82±1.32 and in moderate to severe psoriasis (PASI>10) was 11.51±6.93. There was a significant positive correlation between serum hsCRP level and PASI ($r=0.795$, $p<0.001$). **Conclusion:** The present study revealed a positive correlation between the disease activity of psoriasis and with patient's serum hsCRP concentration. So, serum hsCRP can be considered a marker of disease activity.

Keywords: C reactive protein, Psoriasis, BSMMU, PASI

Introduction

Psoriasis is a complex chronic inflammatory systemic disease, with environmental and genetic components, that affects the skin, nails and occasionally the joints, with periods of exacerbation and remission.[1] Psoriasis is considered a global problem with a prevalence ranging from 0.5 to 11.4 percent.[2] It poses a significant physical, mental and social burden.[3] Quality of life of the patient, in general, is often significantly impaired.[4] Significant disfigurement, discomfort, disability and loss of productivity are important challenges for the patients. There is also a significant cost to mental well-being, such as higher rates of depression, leading to a negative impact on individuals and society.[5] Social exclusion, discrimination and stigma are psychologically devastating for individuals suffering from psoriasis and their families.[6] Psoriasis can occur at any age, and is most common in the age group 50–69.[7] Psoriasis involves the skin and nails and is associated with several comorbidities. Skin lesions are localized or generalized, mostly symmetrical, sharply demarcated, red papules and plaques, and usually covered with white or silver scales. Between 1.3% and 34.7% of individuals with psoriasis develop chronic,

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inflammatory arthritis (psoriatic arthritis) that leads to joint deformations and disability.[8] Between 4.2% and 69% of all patients suffering from psoriasis develop nail changes.[9] Individuals with psoriasis are reported to be at increased risk of developing other serious clinical conditions such as cardiovascular and other non-communicable diseases (NCDs).[10] Psoriasis was first described as a disease that primarily affects epidermal keratinocyte proliferation and secondary cutaneous inflammatory infiltration. In the last decade, it has been evident that psoriasis is a systemic, immune-mediated, inflammatory disease primarily involving Th1 cells. Cytokines of the Th1 pathway (interferon-Gamma, interleukin 2, interleukin 12, and TNF-alpha) predominate in psoriatic plaques. It is widely accepted that an unknown stimulus activates dendritic antigen-presenting cells. The activated antigen-presenting cells then activate helper T cells which lead to the subsequent release of a cascade of inflammatory cytokines. This cascade results in the recruitment and activation of other cell types such as endothelial cells and neutrophils, and the production of chemokines and growth factors. Eventually, that leads to hyperproliferation of keratinocytes. A chronic inflammatory state that ensures and leads to the formation of psoriatic skin lesions.[11] There is increased mitotic activity of the basal cell layer which results in rapid epidermal cell turnover with the 28-day normal epidermal cell cycle reduced by 3 to 5 days. The stimulus for the increased rate of keratinization and even the site of the initial pathologic changes remains controversial.[12] This is reflected clinically by profuse scaling, histologically by a greatly thickened epidermis with increased mitotic activity and by the presence of immature nucleated cells in the horny layer and under the electron microscope by reduced production of intracellular filaments and granules seen within normal keratinization and biochemically by increased synthesis and degradation of nucleoproteins.[13] While analyzing the triggering factors, it was seen that mental stress was the most important triggering factor seen in 48% of patients.[14] Psychological factors can trigger the onset or exacerbation of disease.15 The stress reaction in the patients is mediated by a hypothalamic, pituitary-adrenal relationship with immunologic effects. The commonest factor for the onset of the disease is the environment in which a person has been living and working for a longer period of time and the attitude of a person towards such environment. The other commonest triggering factor was found to be drug intake, which was seen in 36% of patients. The sore throat was found to be a triggering factor in 36% of cases. Trauma as a trigger was found in 22% of cases.[14] Any form of trauma results in psoriasis appearing in the traumatized areas known as koebner's phenomenon[16] Outbreaks often correlate with environmental triggers, often linked to nutritional deficiencies and poor eating habits.[17] It is of important and prevalent skin disease developing due to increasing epidermal cell multiplication. The disease may be intensified by different factors such that the lesions may extend, erythroderma(affecting more than 90% of body skin) may develop and the patient may be hospitalized.[18] Considering available reasons for psoriasis, the main reason is not known but several factors such as family records and accompanying some Human Leukocytes Antigens (HLA) have been mentioned.[19]

C-reactive protein (CRP) was first described in 1930 by Tillet and Francis, named after its ability to precipitate and interact with phosphorylcholine residues of the C polysaccharide derived from teichoic acid within the cellular wall of Streptococcus pneumoniae, as well as its ability to precipitate with calcium ions.[20] CRP is a highly sensitive systemic marker of inflammation and tissue damage. The acute-phase response involves the nonspecific physiological and biochemical responses of endothermic animals to most forms of tissue damage, infection, inflammation, and malignant neoplasia. Plasma CRP is produced exclusively by hepatocytes, predominantly under transcriptional control by the cytokine IL-6, although other sites of local CRP synthesis and possibly secretion have been suggested. De novo hepatic synthesis starts very rapidly after a single stimulus, with serum concentrations rising above 5 mg/l by about 6 hours and peaking around 48 hours. The plasma half-life of CRP is about 19 hours and is constant under all conditions of health and disease, so the sole determinant of circulating CRP concentration is the synthesis rate which thus directly reflects the intensity of the pathological processes stimulating CRP production. When the stimulus for increased production completely ceases, the circulating CRP concentration falls rapidly, at almost the rate of plasma CRP clearance. The circulating value of CRP reflects ongoing inflammation and/or tissue damage much more accurately than other laboratory parameters of the acute-phase response, such as plasma viscosity and the erythrocyte sedimentation rate. The CRP concentration is thus a very useful nonspecific biochemical marker of inflammation, measurement of which contributes importantly to screening for organic disease, monitoring of the response to treatment of inflammation and infection, and detection of intercurrent infection in immunocompromised individuals, and in the few specific diseases characterized by modest or absent acute-phase responses.[21] Psoriasis development depends on skin infiltration of Th1/Th17 cells that stimulate

macrophages and dermal dendritic cells to release mediators that sustain inflammation and cause abnormal keratinocyte proliferation. The mediators of the Th17 immune system include IL-1, IL-6, IL-23 and transforming growth factor (TGF)-β. Elevated C-reactive protein levels result from the interaction between pro-inflammatory cytokines, namely IL-6, TNF-alpha and IL-1. The increased magnitude of CRP seems to be related to the extent of tissue injury and inflammation severity in the active stage of psoriasis.[22] C-reactive protein has special importance for psoriasis due to its relation with cytokines responsible for skin inflammation.[23] Conventional CRP assays can not detect low levels of rise in CRP. The high-sensitivity C-reactive protein (hsCRP) assay is a quantitative analysis of very low levels of CRP in blood (<10mg/L). The high-sensitivity CRP (hsCRP) test accurately measures low levels of C-reactive protein to identify low but persistent levels of inflammation 1. Though several clinical tools for assessing the severity of psoriasis are available, no relevant biochemical marker is commonly recommended for patients with psoriasis. Thus in recent years, a biochemical marker was always thought to be important to find out, which could be the representative of severity of the disease. Analyzing the importance of C-reactive protein in psoriasis, the latter can offer new hope for the physician both in assessing the severity of the disease and also in the global treatment of psoriasis.

Materials & Methods

It was a Cross-Sectional type of observational study. The study was conducted in the Department of Dermatology & Venereology and the Department of Microbiology at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, conducted two years period, from July 2018 To June 2020. Study subjects were the individuals, who were diagnosed with psoriasis vulgaris clinically, with or without the supporting evidence of histopathology. A purposive type of sampling technique was applied to collect the sample from the study population during the study period.

Study Procedure

This present study included 52 psoriatic patients. Patients attending the outpatient and inpatient Department of Dermatology and Venereology, Bangabandhu Sheikh Mujib Medical University; who were diagnosed with cases of psoriasis vulgaris by clinical with or without histopathological confirmation by an expert dermatologist, were informed about the objectives of this study. Related information was collected Disease severity of every patient was measured by using the severity assessment tool namely, the Psoriasis Area and Severity Index (PASI). The score of PASI usually varies between 0 and 72. A PASI score≤ 10 was classified as mild disease, whilst a score of >10 was considered to be moderate to severe disease. Patient's own palm was used according to the checklist based on inclusion and exclusion criteria from them. All aseptic precautions five (5) ml of venous blood were drawn into a vacutainer tube containing no anticoagulant from the median antecubital vein of each patient. Serum concentrations of hsCRP were measured in the Department of Microbiology, Bangabandhu Sheikh Mujib Medical University, Dhaka. Serum hsCRP was analyzed by a nephelometric method using an automated analyzer: BN ProSpec, SIEMENS. After getting the report, the values of the biochemical variable were documented in the data collection sheet.

Result

The mean serum hsCRP level was 6.29±6.8 and the mean Psoriasis Area and Severity Index (PASI) was 10.07±5.88. The mean hsCRP in mild psoriasis (PASI≤10) was 1.82±1.32 and in moderate to severe psoriasis (PASI>10) was 11.51±6.93. There was a significant positive correlation between serum hsCRP level and PASI (r=0.795, p<0.001).

Table: I Distribution of the study population by PASI score (n=52)

PASI score	Frequency	Percentage (%)	PASI Mean±SD
≤10 (Mild disease)	28	53.8	5.41±2.07
>10 (Moderate to severe disease	24	46.2	15.51±3.7.7
Total	52	100.0	10.07±5.88

Table-I It was observed that the majority (53.8%) of patients belonged to PASI score ≤10 (Mild disease) and nearly half (46.2%) of patients belonged to PASI score >10(Moderate to severe disease). The mean PASI score was found 10.07±5.88

Table-II: Association of age with severity of psoriasis (n=52)

Age group (years)	Severity of psoriasis		p-value
	Mild disease (n=28) No. (%)	Moderate to severe disease (n=24) No. (%)	
18-20	3(10.7)	1(4.2)	0.341[ns]
21-30	12(42.9)	8(33.3)	
31-40	8(28.6)	4(16.7)	
41-50	2(7.1)	3(12.5)	
51-60	3(10.7)	7(29.2)	
>60	0(0.0)	1(4.2)	
Total	28(100.0)	24(100.0)	

Data were expressed as frequency and percentage (%)
Chi-square test was done, ns= not significant

Table-II shows the association of age with the severity of psoriasis in study populations. It was observed that statistically insignificant (p=0.341) association between the age of patients with the severity of the disease.

Table III : Association of serum hsCRP with severity of disease according to PASI (n=52)

hsCRP (mg/L)	Severity of psoriasis		Total	p-value
	Mild disease (PASI≤10) (n=28) No. (%)	Moderate to severe disease (PASI>10) (n=24) No. (%)	(n=52) Mean±SD	
Normal (3.0 mg/L)	22(78.6)	0(0.0)		
Elevated (>3.0 mg/L)	6(21.4)	24(100.0)		
Mean±SD	1.82±1.32	11.51±6.93	6.29±6.81	<0.001*
Range (min- max)	(0.20 – 4.65)	(4.62 –25.5)	(0.20-25.5)	
Data wereexpressed as mean±SD Unpaired student t-test was done, *significant				

Table III , shows that hsCRP was strongly associated with the severity of psoriasis according to PASI (P10) was 11.51±6.93 ranging from 4.62 mg/L –25.5 mg/L

Table-IV: Relation of demographic variables with severity of psoriasis. (n=52)

Variables	Severity of psoriasis		p-value
	Mild disease (n=28) No. (%)	Moderate to severe disease (n=24) No. (%)	
Age (years)	32.71±10.60	39.58±14.64	0.056^{ns}
Age of onset (years)	29.64±10.29	36.38±13.96	0.051^{ns}
Duration of disease (years)	3.42±3.06	3.70±3.34	0.748^{ns}

Data were expressed as mean±SD
Unpaired student t-test was done, ns= not significant

Table-IV shows the relationship between demographic variables and with severity of psoriasis and found no significant relationship among these demographic variables.

Table : V Correlation coefficients of severity of psoriasis (PASI) with age, onset of disease, duration of disease and hsCR

Variables	PASI	
	Correlation coefficient (r)	p-value
Age (years	+.306	0.027*
Age of onset	+.296	0.033*
Duration of disease	+.107	0.451
hsCRP	+.795	<0.001*
Pearson's correlation coefficient test was done, *significant		

Table V: It was observed that PASI, the most commonly used clinical assessment tool for disease severity, showed a significant strong positive correlation with hsCRP (r=0.795, p<0.001) and a significant weak positive correlation with age (r=0.306, p=0.027), age of onset (r=0.296, p=0.033). However, PASI showed a weak insignificant positive correlation with duration of disease (r=+.107, p=0.451).

Discussion

This cross-sectional study was carried out to measure serum hsCRP levels in patients with psoriasis and to find out its relationship with the severity of the disease. In this study, the majority (38.5%) of patients belonged to 21-30 years. The mean age was found 35.9±12.9 years with a range from 18 to 70 years. Adrian et al. (2005)25 reported a mean age of 35±15.5 years in their study population. Male predominance was observed in this study with a male-female ratio of 3.1: 1. Globally this ratio is considered to be 1:1 (WHO global report on psoriasis, 2016)26. A study done by Sikder et al. (2017)27, on psoriatic patients at Bangabandhu Sheikh Mujib Medical University, Dhaka, included 30 patients whose age range was 15-67 years, with a mean of 35.8±16.9 years.27 Male-female ratio in that study was 2:1. Jain k et al. (2017) observed a male-to-female ratio of 1.92:1 in their study.1 The current study showed a slightly higher male-female ratio in comparison to other studies. Various factors can be considered reasonable for this difference in the sex ratio in this current 52 study. Social aspects such as the way men and women perceive their health, their different social roles and levels of tolerability could be considered as some of the important determinants for accessibility to health care facilities. Sometimes cultural and religious barriers impose major stigma explaining why in Bangladesh, women are not privileged enough to seek medical help whenever needed. For these issues, males were thought to be enrolled more in number in this study in comparison to the female patients. The mean age of onset of psoriasis was 32.75±12.46 years with a range from 13 to 57 years. The mean duration of the disease was 3.55±3.16 years with a range from 0.20 to 11.0 years. Jain K et al. (2017)1 reported the mean age of onset of their study subjects as 32±14.10 years.1 This finding is almost similar to the current study. In the current study, it was observed that PASI, the most commonly used

clinical assessment tool for disease severity, showed a significant weak positive correlation with age (r=0.306, p=0.027), insignificant weak positive correlation with duration of disease (r=+.107, p=0.451). El-Komy et al. (2020) in their study found that PASI correlated positively with patients' age and duration of psoriasis. 27 In another study by Cakmur&Dervis, (2015) found a positive correlation between disease duration and the severity of psoriasis (PASI). These findings are similar to the current study. In this study, it was observed that the majority (53.8%) of the patients had mid psoriasis (PASI ≤10) and nearly half (46.2%) of the patients had moderate to severe psoriasis (PASI >10). The severity of psoriasis was determined by the PASI score. A PASI score ≤ 10 was considered mild psoriasis and >10 was considered a moderate to severe form of psoriasis 29. The mean PASI score was found 10.07±5.88. Vadakayil et al. (2018) observed that the mean PASI score in 53 their study patients was 18.15 ± 12.208. Kumari and Kumar, (2018) found the mean PASI score of psoriatic patients was 15.58±6.51. 30-31 The mean PASI in patients with psoriasis was 10.36±10.36.32 In another study, Gisondi et al. (2012) found the mean PASI in patients with psoriasis was 11.1 ± 10.4.33 So, most of the study showed that mean PASI corresponds to the current study. In this study, the mean hsCRP in mild disease (PASI≤10) was 1.82±1.32 ranging from 0.20 to 4.65 and in moderate to severe disease (PASI>10) was 11.51±6.93 ranging from 4.62 to 25.5. Out of 52 patients, the disease was mild (PASI ≤10) in 28 patients and moderate to severe (PASI > 10) in 24 patients. When comparing the mean PASI in each severity group (mild, moderate to severe) to the mean hsCRP in that group, the results were statistically highly significant. It was also observed that the mean hsCRP was higher in the group with maximum severity of psoriasis. Mean hsCRP levels of patients with moderate to severe psoriasis in studies by Keerthana et al. (2016) and Agravatt et al. (2013) were 7.42±3.26 mg/l and 6.26 ±3.84 mg/l, respectively, with a highly significant correlation with PASI.23,34 In another study conducted by Murari (2017) found that the mean hsCRP levels of patients with moderate to severe psoriasis were 10.64± 2.23 with a highly significant correlation with PASI.22 These findings are nearly similar to the current study. The present study found a significant strong positive correlation between hsCRP and PASI (r=0.795, p< 0.003). Uaratanawong et al. (2016) in their study observed findings similar to the current 54 studies. 35Gupta et al. (2019) in their study also found a significant correlation of hsCRP with PASI score (r=0.48, p<0.001).36 This present study considered this fact as the explanation of this observation.

Conclusion

A statistically significant positive correlation was found between the PASI score and serum hs-CRP level. A statistically significant weak positive correlation was found between PASI score and age. A statistically significant weak positive correlation was found between PASI score and age of onset. A statistically insignificant weak positive correlation was found between the PASI score and the duration of the disease.

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