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Determination of Prevalence of Periodontitis Among Sickle Cell Anemia Patients - A Cross Sectional Analytical Study

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Introduction

Sickle cell anemia (SCA) is a hereditary autosomal recessive disorder. It is caused by the replacement of glutamic acid by valine in position 6 at the N-terminus of the beta chain of globin. This genetic alteration results in hemoglobin S formation. [1] The pathophysiology of sickle cell anemia is result from polymerization of hemoglobin S in red blood cell under hypoxic conditions, which result in Vaso-occlusion (VOC). [2]

It is also hypothesized that IL-1β, IL-6 gene polymorphism in SCA individuals results in to increased production of those cytokines which enhance the chances of VOC. [3] Researches have indicated that prevalence of IL-1β, IL-6 gene polymorphism renders the individuals susceptible to periodontitis which is a chronic inflammatory disease. [4] In the light of this information, it is logical to expect SCA patients to be prone for periodontitis.

The hypothesis of the study is that there is a relationship between these two chronic inflammatory diseases i.e. SCA & Periodontitis, which share common pathogenesis of chronic inflammation. Thus, this study was conducted with the aim to investigate the prevalence of periodontal disease among SCA patients.

Materials & Methods

The present study included a total of 1050 subjects. Group I comprised of Sickle cell anaemia patients (n=350) randomly selected after screening from SCA belt of MP i.e. Mandla, district; Group II comprised of

periodontitis patients (n=350) randomly selected from the Out Patient Department of Periodontology, Sri Aurobindo College of Dentistry, Indore. Group III comprised of systemically healthy individuals (n=350) who were included in control group. Our study is a cross-sectional analytical study, conducted in accordance with the guidelines of the Declaration of Helsinki (1975), as revised in 2013. Study was approved by the Institutional Ethical Committee and was carried out over a period of one year, from April 2021 to August 2022. All participants signed a WHO-informed written consent form after the details of research were explained to them.

The inclusion criteria were patients within the age range of 18-65 years, comprising both genders, sickle cell anaemia patients in steady state & with at least one relative having sickle trait, periodontitis patients who had not undergone periodontal treatment in the past 6 months, periodontitis & healthy volunteers who are systemically healthy. Exclusion criteria included patients with any systemic disease, sickle cell anaemia patients who had received blood transfusion within 3 months prior to the study, pregnant and lactating females and patient unwilling to participate in study.

All patients underwent a comprehensive periodontal examination, which included assessment of the Plaque Index (PI; Silness & Loe, 1964), Gingival Index (GI; Loe & Silness, 1963), and Modified Sulcular Bleeding Index (mSBI; Mombelli et al., 1987). Probing pocket depth (PPD) and clinical attachment loss (CAL) were measured using a periodontal probe (UNC 15, Hu-Freidy, United States) at six standard sites on each tooth, excluding third molars. Demographic, medical, and dental history were recorded prior to the clinical assessment.

Statistical Analysis

The data was collected and entered in MS excel 2010, statistical analysis was performed using SPSS 26 version. Intergroup comparison was done using ANOVA test (one way). P value <0.05, considered as statistically significant result and if p value > 0.05, then it is statistically insignificant result.

Results

Table 1: Demographic data of the patients of all the three groups

Parameters		SCA Group (n=350)	Periodontitis group (n=350)	Healthy group (n=350)
Mean age (years)		26.8 ± 7.38	41.2 ±, 8.71	26.6 ± 6.9
Gender	Male (%)	191(54.6%)	214(61.1%)	228(65.1%)
	Female (%)	159(45.4)	136(38.9%)	122(34.9)

Table 2: Intergroup comparative analysis of periodontal parameter in all 3 groups

Variable	Group	N	Mean ± SD	Mann-Whitney U	p-value
PI	SCA	350	1.1 ± 0.58	33129	0.001
	Periodontitis	350	1.6 ± 0.27		(HS)
	SCA	350	1.1 ± 0.58	26889	0.001
	Healthy	350	0.5 ± 0.41		(HS)

	Periodontitis Healthy	350 350	1.6 ± 0.27 0.5 ± 0.41	5747	0.001 (HS)
GI	SCA Periodontitis	350 350	0.6 ± 0.39 1.6 ± 0.33	3368.5	0.001 (HS)
	SCA Healthy	350 350	0.6 ± 0.39 0.3 ± 0.38	34139	0.001 (HS)
	Periodontitis Healthy	350 350	1.6 ± 0.33 0.3 ± 0.38	2338.5	0.001 (HS)
mSBI	SCA Periodontitis	350 350	0.7 ± 0.55 2.2 ± 0.38	3040	0.001 (HS)
	SCA Healthy	350 350	0.7 ± 0.55 0.6 ± 0.66	52535	0.001 (HS)
	Periodontitis Healthy	350 350	2.2 ± 0.38 0.6 ± 0.66	5052.5	0.001 (HS)
PPD	SCA Periodontitis	350 350	3.6 ± 0.53 5.9 ± 0.96	0	0.001 (HS)
	SCA Healthy	350 350	3.6 ± 0.53 3.2 ± 0.71	42587	0.001 (HS)
	Periodontitis Healthy	350 350	5.9 ± 0.96 3.2 ± 0.71	0	0.001 (HS)
CAL	SCA Periodontitis	350 350	3.2 ± 0.70 4.0 ± 0.95	34960	0.001 (HS)
	SCA Healthy	350 350	3.2 ± 0.70 0.2 ± 0.38	0	0.001 (HS)
	Periodontitis Healthy	350 350	4.0 ± 0.95 0.2 ± 0.38	0	0.001 (HS)

Table 1 shows the demographic data of all the groups. The mean age of the SCA group patients is 28.2±9.08 years, periodontitis group is 41.3±7.64, & healthy group is 28.0±7.96. The number of male patients in SCA group was 191, periodontitis group was 214 & healthy group was 228. The number of female patients in SCA group was 159, periodontitis group was 214 & healthy group was 228.

Table 2 shows the intergroup comparisons of all the periodontal parameters in all the group.

The plaque index is highest in periodontitis patients (1.6 ± 0.27) followed by SCA patients (1.1 ± 0.58) & healthy group (0.5 ± 0.41) the differences being statistically highly significant (p-0.001). The gingival index is highest in periodontitis patients (1.6 ± 0.33) followed by SCA patients (0.6 ± 0.39) & healthy group (0.3 ± 0.38) differences being statistically highly significant (p-0.001). The mean mSBI is highest in periodontitis patients (2.2 ± 0.38) followed by SCA patients (0.7 ± 0.55) & healthy group (0.6 ± 0.66) differences being statistically highly significant (p-0.001).

The PPD is highest in periodontitis patients (5.9 ± 0.96) followed by SCA patients (3.6 ± 0.53) & healthy volunteers (3.2 ± 0.71) which is statistically highly significant (p-0.001). The CAL is highest in periodontitis patients (4.0 ± 0.95) followed by SCA patients (3.2 ± 0.70) & healthy group (0.2 ± 0.38) which is statistically highly significant (p-0.001). Overall, all the periodontal parameters is highest in periodontitis group followed by SCA & healthy individuals the differences being statistically highly significant (p-0.001).

Discussion

The present study aims to determine the prevalence of periodontitis among SCA patients. We observed that all the periodontal parameters i.e. PI, GI, mSBI, PPD & CAL were significantly higher in SCA patients as compared to healthy individuals.

The results of present study were consistent with previous studies done by Abid et al. & Mehra et al. They examined the periodontal health in SCA patients and observed higher scores of PI, GI which indicate that periodontal health is affected in SCA patients. [5,6] Similarly, Guzeldemir observed higher plaque & gingival indices in SCD patients than healthy individual. [7] The higher plaque scores observed can be attributed to inadequate oral hygiene practices among SCA patients, as oral health is often not a priority for patients with SCD. It further leads to increased gingival inflammation which is clinically evident as increased BOP. The heightened plaque scores & gingival inflammation were responsible for increased PPD & CAL scores. Therefore, increased PI, GI & mSBI due to plaque accumulation & poor oral hygiene in SCA patients may suggest severity of periodontal disease in SCD.

SCD is a genetic condition which is hereditary, autosomal recessive disorder. This chronic disorder is ought to result from polymerization of hemoglobin S in red blood cells (RBCs) under low oxygen tension which results in sickling. The sickled erythrocytes have an increased tendency for adhesion to vascular endothelium resulting in vaso-occlusion. Along with vaso-occlusion other factors such as upregulation of inflammasome pathway gene expression increases propensity to bacterial infections.

The upregulated pathway results in expression of toll like receptor-4 (TLR-4) & increased production of pro-inflammatory cytokine IL-6 & IL-8. It is hypothesized that increased level of IL-6 & IL-8 are responsible for poor clinical outcome of SCD. A combination of vaso-occlusion and the cascade of pro-inflammatory events triggers activation of neutrophils, monocytes & platelets. This further leads to opsonization, complement pathway and antibody production. Thus, the propensity to persistent gingival infection, poor oral hygiene & increased load of bacteria along with underlying pathophysiological factors that exacerbate inflammatory process in an immuno-compromised host are thought to be responsible for the development of periodontitis in SCA patients.

The results of present study are suggestive of a possible link between SCD & periodontitis and henceforth, it can be established that SCA patients are more susceptible to periodontal diseases. The plausibility of this finding may be related to the pathophysiology of periodontitis and SCD as both are chronic inflammatory disorder.

In conclusion, there is an increased prevalence of periodontitis in SCA patients. However, further research is needed to establish a plausible association between SCD & periodontitis due to the extensive range of clinical complications observed in SCD population. As it is a matter of concern since occurrence of periodontitis in SCA patients will further worsen the quality of their life. Consequently, prioritizing preventive dental care is imperative & enhancing quality of life for patients with SCA.

The limitations of the present study are, the study population was taken from a restricted area and more multicenter, cross-sectional studies on different population groups are required to provide more evidence of correlation between SCA & periodontitis. Hence more studies are required to validate our findings.

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