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Original Article

ABO Blood Groups, Periodontal Health and Disease

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Abstract

Context: Periodontal disease remains a significant public health challenge in India, characterized by high morbidity and substantial out-of-pocket expenditure. This study investigated the ABO blood group system as a primary genetic determinant of periodontal health. Aims: The aim of the study was to elucidate the associations of ABO blood group systems with periodontal health and disease, and to assess their potential role as genetic susceptibility markers for periodontal disease.

Settings and Design: The study employed a double-blind, parallel-group design featuring a sample of 800 participants selected through convenience sampling based on predefined eligibility criteria and voluntary informed consent. Methods and Material: Subjects were stratified into eight distinct groups according to their gender and the blood group genotypes.

Statistical analysis used: Data were analyzed using the statistical software, namely, the Statistical Package for the Social Sciences (Version 16, IBM Analytics), and Systat 8.0 analysis of variance was used to test the significance of the prevalence of blood groups.

Results: Blood group AB has primarily been associated with periodontal health, while blood Group A was majorly associated with gingivitis, and blood Groups B and O were associated with periodontitis.

Conclusion: This research identifies the ABO blood group system as a critical immunogenetic determinant of periodontal health.

Key words: ABO blood group, Gingivitis, Human genetics, Periodontal health, Periodontitis

INTRODUCTION

Periodontal disease is a highly prevalent, multifactorial condition that represents a significant global health burden. According to the World Health Organization, severe periodontitis affects approximately 19% of the global adult population,[1] accounting for more than one billion cases worldwide. This heterogeneous group of inflammatory infections is driven by a complex interplay between dysbiotic dental plaque biofilms and the host's immune-inflammatory response, which, if left untreated, leads to the progressive destruction of the connective tissue attachment and supporting alveolar bone. While microbial biofilms are the primary initiators of the disease, the extent and severity of tissue destruction are governed by the magnitude of the host response. This biological reaction is significantly modulated by systemic conditions[2] and genetic predisposition.[3] During recent years, attention has turned toward specific genetic markers that may influence the inflammatory landscape. Among these, the ABO blood group antigens have emerged as potential determinants of individual risk, possibly dictating the severity of periodontal destruction and the host's overall vulnerability to the disease. The 1901 discovery of the ABO blood group system by Karl Landsteiner marked a transformative milestone in transfusion medicine and human genetics.[4] Over a century later, these antigens remain a cornerstone of modern healthcare, extending beyond hemocompatibility to serve as critical biomarkers in the pathogenesis of various systemic and inflammatory diseases. While the role of ABO antigens is well-documented in other systemic conditions, their specific relationship with periodontal disease remains underexplored. This is particularly evident within the Indian populace, whose unique genomic landscape necessitates localized immunogenetic research. The objective of this study was to elucidate the association of ABO blood group systems with periodontal health

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and disease, and to assess its potential role as genetic susceptibility markers for periodontal disease.

MATERIALS AND METHODS

The study was conducted on 800 patients (400 males and 400 females) from August 2023 to May 2025. The patients required for the study were recruited from the outpatient department of the Department of Periodontics in our institute. The study protocol was reviewed and approved by the Institutional Ethics Committee and Review Board (approval number: IDS/IEC/2023/PERIO/01) and registered on Clinical Trials Registry India (Registration number: CTRI/2023/03/050183). A written informed consent was obtained from all the participating members in the study after thorough explanation of the procedure and risks involved; also, it was made clear that participation in the study was voluntary. Participants reporting to the outpatient department of the Department of Periodontics, having an age group 14–65 years, and who know the blood group of their parents as per their medical record, were included in the study. While the participants having systemic disease, having any local or systemic antimicrobial and anti-inflammatory therapy for the last 6 months, with the history of smoking or tobacco chewing, pregnant or lactating women, and the having undergone periodontal therapy other than standard prophylaxis during the previous 6 months, were excluded from the study.

The study design was a double-blinded parallel group, consisting of a convenience sample of 800 patients who fulfilled the eligibility criteria and were willing to participate in the study. The participants were allocated to eight groups based on their blood group genotype, and allocation concealment was done using opaque sealed envelopes. Group: 1: A (Male), Group: 2: A (Female), Group: 3: B (Male), Group: 4: B (Female), Group: 5: AB (Male), Group: 6: AB (Female), Group: 7: O (Male), Group: 8: O (Female). Clinical parameters such as plaque index, gingival index, Russel's periodontal index, and clinical attachment level were used to check the status of periodontal health, gingivitis, and periodontitis.

Statistical Analysis

Data were analyzed using the statistical software, namely, the Statistical Package for the Social Sciences (Version 16, IBM Analytics) and Systat 8.0. Analysis of variance was used to test the significance of the prevalence of blood groups.

RESULTS

The study was conducted on 800 patients (600 males and 600 females) with the mean age of 35 ± 05 years from August 2023 to May 2025. The patients required for the study were recruited from the outpatient department of the Department of Periodontics of our institute. Out of this population, 522 (65.25%) were patients having periodontitis, while 132 (16.50%) patients suffered from gingivitis, and 146 (18.25%) were healthy patients [Table 1 and Figure 1].

Regarding periodontal status, Group 6 exhibited the highest prevalence of healthy patients (9.0%), followed by Group 5 (8.125%) [Table 1 and Figure 2]. In terms of disease progression, mild gingivitis was most frequent in Group 2 (2.125%) and Group 6 (1.875%). However, moderate gingivitis was also notably higher in Group 2 (2.875%), followed by Group 1 (2.75%). Severe gingivitis remained rare across all cohorts, with the highest incidence observed in Group 1 (0.125%) and Group 6 (0.125%) [Table 2 and Figure 2].

The prevalence of periodontitis by Grade is detailed in Table 3 and Figure 3. Grade I periodontitis was most severe in Group 7 (10.675%), followed by Group 8 (10.375%) and Group 1 (6.75%). However, a shift toward higher disease severity was observed in Group 3, which exhibited the highest prevalence of

Table 1: Frequency distribution of blood group in periodontal health and periodontal disease in patients

Groups	Periodontal status		Significance (*P*)
	Periodontal health (%)	Periodontal disease (%)	
Group 1	6 (0.75)	94 (11.75)	<0.001
Group 2	3 (0.375)	97 (12.125)	
Group 3	0 (0)	100 (100)	
Group 4	0 (0)	100 (100)	
Group 5	65 (8.125)	35 (4.375)	
Group 6	72 (9.00)	28 (3.5)	
Group 7	0 (0)	100 (100)	
Group 8	0 (0)	100 (100)	
Total	146 (18.25)	654 (81.75)	

Table 2: Frequency distribution of blood group in mild, moderate and severe gingivitis patients

Groups	Gingivitis			
	Mild (%)	Moderate (%)	Severe (%)	Significance (*P*)
Group 1	14 (1.75)	22 (2.75)	1 (0.125)	<0.001
Group 2	17 (2.125)	23 (2.875)	0 (0)	
Group 3	2 (0.25)	1 (0.125)	0 (0)	
Group 4	1 (0.125)	2 (0.25)	0 (0)	
Group 5	13 (1.625)	10 (1.25)	0 (0)	
Group 6	15 (1.875)	4 (0.5)	0 (0)	
Group 7	0 (0)	2 (0.25)	0 (0)	
Group 8	3 (0.375)	1 (0.125)	1 (0.125)	
Total	65 (8.125)	65 (8.125)	2 (0.25)	

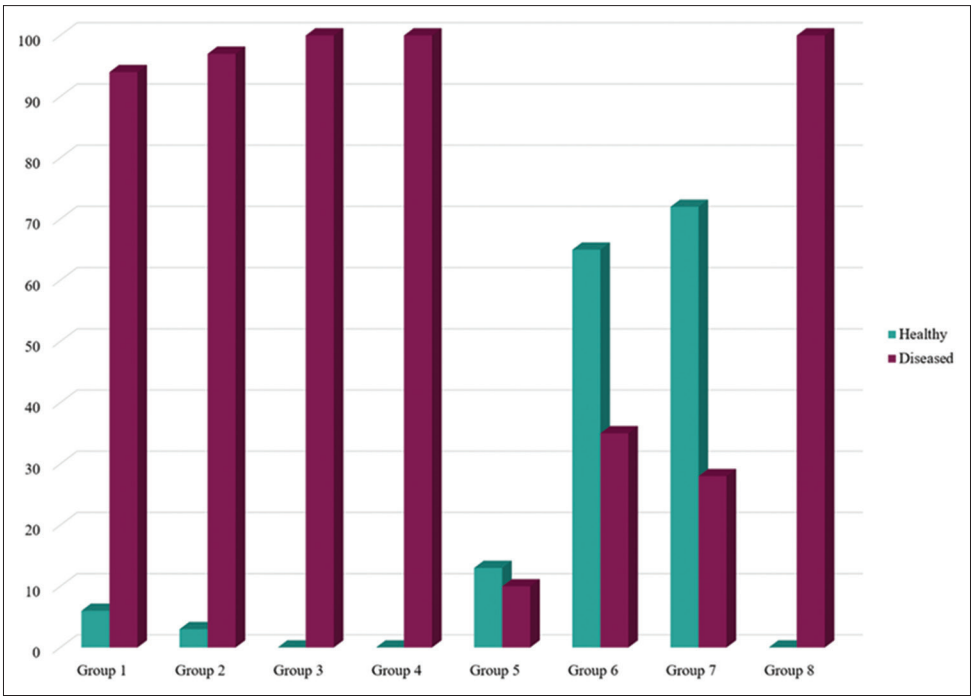

Figure 1: Frequency distribution of blood group in periodontal health and periodontal disease in patients

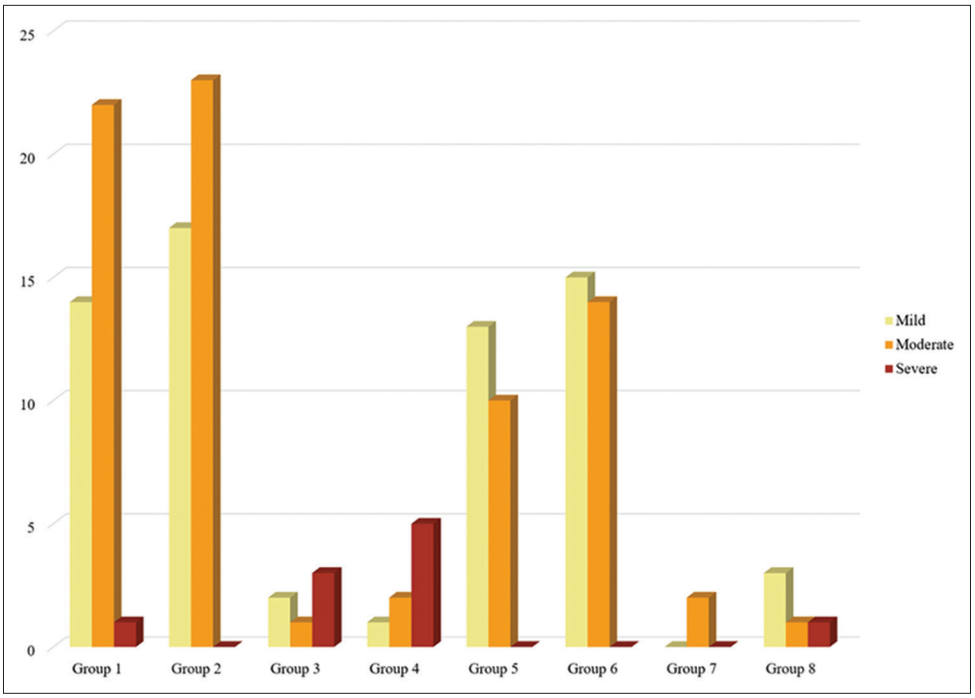

Figure 2: Frequency distribution of blood group in mild, moderate and severe gingivitis patients

Grade II (5.25%), Grade III (1.875%), and second highest prevalence of Grade IV (1.125%) periodontitis. Following Group 3, Group 4 consistently showed the next highest rates for Grade II (4.00%) and Grade III (1.5%) disease, while Group 6 presented the highest prevalence for Grade IV (1.25%).

DISCUSSION

Periodontal diseases, ranging from gingivitis to chronic periodontitis, represent a major public health challenge. Given their high global prevalence, these conditions are of a significant concern not only for oral health but also for

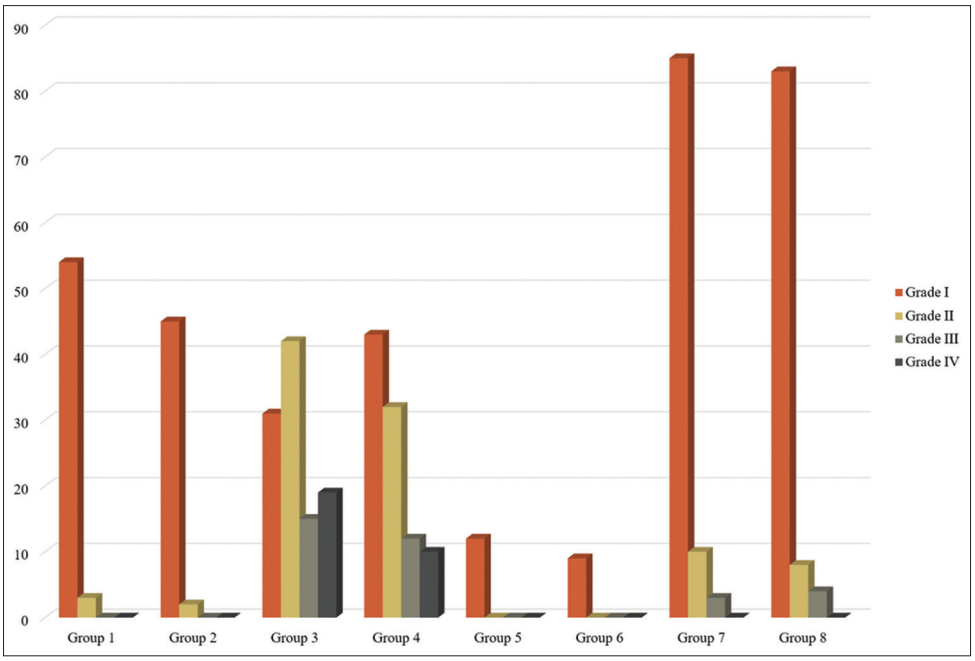

Figure 3: Frequency distribution of blood group in Grade I, Grade II, Grade III, and Grade IV periodontitis patients

Table 3: Frequency distribution of blood group in Grade I, Grade II, Grade III, and Grade IV periodontitis patients

Groups	Periodontitis				
	Grade I (%)	Grade II (%)	Grade III (%)	Grade IV (%)	Significance (*P*)
Group 1	54 (6.75)	3 (0.375)	0 (0)	0 (0)	<0.001
Group 2	45 (5.625)	2 (0.25)	0 (0)	0 (0)	
Group 3	31 (3.875)	42 (5.25)	15 (1.875)	9 (1.125)	
Group 4	43 (5.375)	32 (4.0)	12 (1.5)	10 (1.25)	
Group 5	12 (1.5)	0 (0)	0 (0)	0 (0)	
Group 6	9 (1.125)	0 (0)	0 (0)	0 (0)	
Group 7	85 (10.625)	10 (1.25)	3 (0.375)	0 (0)	
Group 8	83 (10.375)	8 (1.0)	4 (0.5)	0 (0)	
Total	362 (45.25)	97 (12.125)	34 (4.25)	19 (2.375)	

their established links to severe systemic complications. The global burden of periodontal disease remains substantial, with severe forms affecting roughly 11% of the population – a figure that has remained high despite advances in dental care. According to recent Global Burden of Disease estimates, severe periodontitis is the sixth most frequent condition globally.[3]

The prevailing paradigm regarding the pathogenesis of periodontitis has undergone a significant shift, moving beyond a strictly infectious etiology toward a model of host-mediated destruction. While the presence of a dysbiotic microbial community remains a prerequisite for disease initiation, the subsequent progression and clinical severity are fundamentally governed by host-derived inflammatory responses. Consequently, periodontal diseases are increasingly classified as ecogenetic disorders, emphasizing a complex multifactorial interplay between microbial challenges, genetic susceptibility, and environmental modifiers.[5] Genetic polymorphisms serve as critical determinants in the clinical heterogeneity of periodontitis, acting as either risk or protective factors that modulate the host's inflammatory landscape. These genetic traits do not merely influence immune reactivity but actively reshape the oral ecological niche and the trajectory of tissue degradation.[6]

Emerging evidence suggests a significant correlation between periodontal susceptibility and innate hematological markers, specifically the ABO blood group system. These erythrocyte antigens, which are also expressed in salivary secretions and periodontal tissues, may function as specific receptors for periopathogens, thereby facilitating differential bacterial colonization and influencing the rate of disease progression. Offenbacher[7] and Roberts[6] established the ABO blood group as an essential marker for investigating the genetic basis of familial disease predisposition. The American Academy of Periodontology (AAP) defines periodontal health as a state of clinical immune homeostasis, characterized by the absence of inflammatory biomarkers and the preservation of the structural integrity of the periodontium on either an intact or stable reduced architecture. Beyond mechanical dentition stability, the periodontium functions as a semi-permeable immunologic rheostat. When the subgingival dysbiosis triggers a shift from resolution to chronic inflammation, the periodontium acts as a systemic reservoir for senescence-associated secretory phenotypes, including interleukin-1β

(IL-1β), tumor necrosis factor-alpha (TNF-α), and IL-6. These mediators, along with translocated pathobionts, fuel a systemic inflammatory burden that precipitates insulin resistance via the JNK/IKK β pathways and drives atherogenesis through endothelial cell activation and oxidative stress. At a molecular level, the I^A and I^B alleles compete for the H-substance substrate; the resulting glycosylation patterns on epithelial cell surfaces and in salivary secretions may dictate the selective adhesion of primary colonizers. The dominance of healthy parameters in these cohorts suggests that the specific carbohydrate moieties present in the AB/H secretor status may interfere with the lock-and-key attachment of keystone pathogens like *Porphyromonas gingivalis*, thereby maintaining the subgingival niche in a state of symbiosis rather than dysbiosis. Furthermore, the observed periodontal resilience in Group 6 may be attributed to the unique stereochemical configuration of the H-substance terminal sugars. In the AB blood group, the presence of both N-acetylgalactosamine (A-antigen) and D-galactose (B-antigen) on the oligosaccharide chains of the sulcular epithelium and salivary mucins creates a complex, heterogeneous glycan shield. This phenotypic complexity likely induces competitive inhibition or steric hindrance against the fimbrial adhesins of keystone pathogens such as *P. gingivalis* and *Prevotella*.[8] Thus, the AB phenotype may function as a biological decoy,[9] effectively neutralizing bacterial attachment and maintaining the shallow pockets and less bleeding on probing (BOP) observed in this study's healthiest cohorts. This correlation, aligning with the findings of Gawrzewska[10] and Vivek *et al*.,[11] may be elucidated through the lens of Mendelian co-dominance and epistatic modulation.

Gingivitis is defined as a reversible inflammation of the gums, characterized by redness, swelling, and bleeding, caused primarily by dental plaque, with clinical signs including blunted papillae and absence of attachment/bone loss, requiring at least 10% BOP for diagnosis on an intact periodontium. In the present study, patients belonging to Group 2 (5.00%) followed by Group 1 (4.625%) showed the highest number of gingivitis, as shown in Table 2 and Figure 2. Similar observations have been made by Demir *et al*.[12] and Koregol *et al*.[13] who concluded that the patients with gingivitis were more in blood group A. This could be explained by the following three mechanistic domains:

1. Lectin-Mediated Adhesion and Selective Colonization: The Blood Group A antigen is characterized by a terminal N-acetylgalactosamine (GalNAc) sugar moiety, by selective adhesion where periopathogens such as *P. gingivalis* and *Prevotella intermedia*, express surface lectins and fimbriae that specifically bind to carbohydrate receptors. The GalNAc residue in Group A serves as a high-affinity docking site, facilitating faster colonization of the subgingival niche.[14] Furthermore, maturation of virulence factors, such as gingipains in *P. gingivalis*, involves complex glycosylation processes that may be modulated by the host's carbohydrate environment.[15]
2. Immunomodulation and cytokine profiles: Blood group antigens are not merely passive erythrocytic markers; they function as active immunomodulators that dictate the magnitude of the host inflammatory response. This is evidenced by cytokine regulation, where individuals with Blood Group A often demonstrate elevated levels of (IL-1β, IL-6, and TNF-α) even when plaque indices are standardized.[1] Furthermore, research suggests that the A-antigen phenotype is associated with a heightened innate immune sensitivity to the lipopolysaccharides (LPS) of periodontal pathobionts, leading to more pronounced clinical manifestations such as increased BOP and edema, suggesting that the A-antigen may lower the threshold for nuclear factor kappa B activation in gingival fibroblasts.[16]

The AAP defines periodontitis as a chronic, multifactorial inflammatory disease caused by dysbiotic plaque biofilms, leading to the progressive destruction of the tooth's supporting structures, characterized by clinical attachment loss, periodontal pocketing, and radiographic bone loss.[17] In the present study, it was observed that the highest number of patients having periodontitis belonged to Group 7, followed by Group 3 and 4, and Group 8, as shown in Table 3, and illustrated in Figure 3, with the highest number of Grade IV patients in to Group 3 and 4. Similar observations have been made in the studies conducted by Ghamdi[18] and Arowojolu *et al*.,[19] who concluded that patients with blood Group B are more likely to have periodontitis. The possible mechanism for this could be due to the following reasons:[20]

1. Molecular mimicry and immune evasion: Several periodontal pathogens, most notably certain strains of *P. gingivalis* produce LPS that contain galactose-rich O-antigen side chains, which allows the subgingival biofilm to reach a higher microbial load and mature into a more virulent state before a robust inflammatory response is triggered, leading to more extensive bone loss.
2. The glycobiology of the niche: The B-antigen serves as a specific substrate within the subgingival environment. The D-galactose moiety acts as a receptor for specific bacterial lectins, facilitating the anchoring of the bridge species, like *Fusobacterium nucleatum,* which are essential for the architectural stability of the pathogenic biofilm. The presence of soluble B-antigens in the saliva acts as a competitive inhibitor (decoy).
3. Reduced molecular mimicry and efficient clearance: The innate immune system in Group O individuals

may recognize and clear periodontal pathogens more effectively, leading to a lower systemic inflammatory load, reflected in lower concentrations of proinflammatory cytokines such as IL-1β in the gingival crevicular fluid.

CONCLUSION

Based on the clinical parameters analysed in the study, a distinct hierarchy of periodontal susceptibility emerges, stratified by the genotype. The severity of periodontal destruction is more in Groups 7, 3, and 4 and 8 (blood group O and B), while Groups 2 and 1 (blood group A) were associated with gingivitis, and Groups 6 and 7 (blood group AB) were associated with periodontal health. This pattern suggests a synergistic interaction between the *IB* allele, which may exacerbate the inflammatory responses. Furthermore, the transition from homozygous to heterozygous states across the A and B alleles appears to provide a quantitative reduction in the disease severity, pointing toward a gene-dosage effect in periodontal pathogens.

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