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Original Article

Genotypic Correlation of ABO Blood Groups with 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: To elucidate the genotypic 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 1,200 participants selected through convenience sampling based on predefined eligibility criteria and voluntary informed consent.

Materials and Methods: Subjects were stratified into six distinct groups according to their specific blood group genotypes. To maintain the integrity of the study, allocation concealment was rigorously implemented using sequentially numbered, opaque, sealed envelopes, after which various periodontal parameters were investigated.

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

Results: Blood group I^AI^B has primarily been associated with periodontal health, while blood group I^AI^A and I^Ai were mainly associated with gingivitis, and blood group I^BI^B, I^Bi and *ii* were associated with periodontitis.

Conclusion: This research identifies the ABO blood group system as a critical immunogenetic determinant of periodontal health. Our findings demonstrate a clear genotypic–phenotypic correlation: The I^B allele increases vulnerability to pathogenic dysbiosis, whereas the I^AI^B genotype provides protective glycan shielding. These results establish ABO status as a robust, non-invasive biomarker for risk stratification, supporting a shift from reactive treatment toward a model of Precision Periodontics.

Key words: ABO blood group, Genetics, Gingivitis, 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,

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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 under-explored. This is particularly evident within the Indian populace, whose unique genomic landscape necessitates localized immunogenetic research. To date, investigations have primarily focused on serological phenotypes; to the best of our knowledge, no study has yet evaluated the genotypic correlation between the ABO locus and periodontal destruction. The objective of this study was to elucidate the genotypic association of ABO blood group systems with periodontal health and disease, and to assess its potential role as genetic susceptibility markers for periodontal disease.

MATERIALS AND METHODS

The study was conducted on 1200 patients (600 males and 600 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). Written informed consent was obtained from all the participating members in the study after a thorough explanation of the procedure and risks involved; it was also made clear that participation in the study was voluntary.

Participants reporting to the Outpatient Department of the Department of Periodontics aged 14–65 years and who knew 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 past 6 months, with a history of smoking or tobacco chewing, pregnant or lactating women, and those 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 design, consisting of a convenience sample of 1200 patients who fulfilled the eligibility criteria and were willing to participate in the study. The participants were allocated to 6 groups based on their blood group genotype, and allocation concealment was done using opaque sealed envelopes. Group: 1: $I^A\ I^A$ (AA), Group: 2: $I^A\ i$ (AO), Group: 3: $I^B\ I^B$ (BB), Group: 4: $I^B\ i$ (BO), Group: 5: $I^A\ I^B$ (AB), and Group: 6: $i\ i$ (OO). Clinical parameters such as plaque index, gingival index, Russell'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, Statistical Package for the Social Sciences (Version 16, IBM Analytics) and Systat 8.0. Chi-square tests were used to test the significance of the prevalence of blood groups.

RESULTS

The study was conducted on 1200 patients (600 males and 600 females) with a 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, Institute of Dental Sciences Jammu, J&K.

Out of this population, 831 (69.25%) were patients with periodontitis, while 243 (20.25%) patients suffered from gingivitis and 126 (10.5%) were healthy individuals [Table 1 and Figure 1].

Regarding periodontal status, Group 5 exhibited the highest prevalence of healthy individuals (8.917%), followed by Group 1 (1.583%) [Table 1 and Figure 2]. In terms of disease progression, mild gingivitis was most frequent

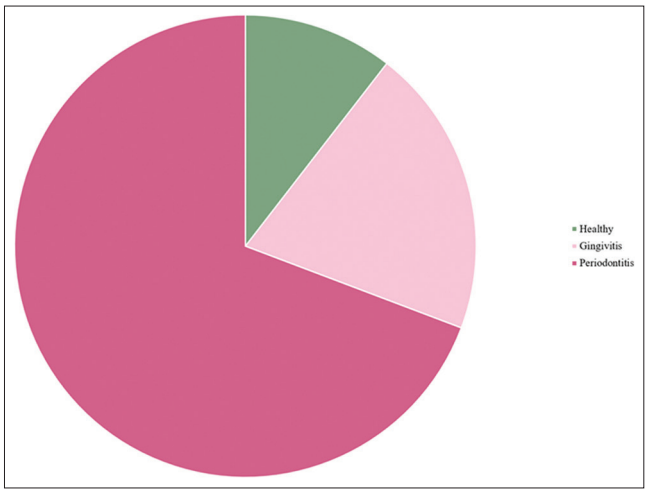

Figure 1: Distribution of healthy, gingivitis and periodontitis patients in the study

Table 1: Frequency distribution of blood group in healthy, gingivitis and periodontitis patients

Groups	Periodontal status			
	Healthy (%)	Gingivitis (%)	Periodontits (%)	*P*-value
Group 1	19 (1.583)	147 (12.25)	34 (2.833)	<0.001
Group 2	0 (0)	12 (1)	188 (15.67)	
Group 3	0	0 (0)	200 (16.67)	
Group 4	0	5 (0.417)	195 (16.25)	
Group 5	107 (8.917)	72 (6)	21 (1.75)	
Group 6	0	7 (0.583)	193 (16.08)	
Total	126 (10.5)	243 (20.25)	831 (69.25)	

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

Groups	Gingivitis			
	Mild (%)	Moderate (%)	Severe (%)	*P*-value
Group 1	49 (4.083)	96 (8)	2 (0.167)	<0.001
Group 2	6 (0.5)	6 (0.5)	0 (0)	
Group 3	0 (0)	0 (0)	0 (0)	
Group 4	2 (0.167)	3 (0.25)	0 (0)	
Group 5	58 (4.83)	14 (1.167)	0 (0)	
Group 6	3 (0.25)	3 (0.25)	1 (0.083)	
Total	118 (9.833)	122 (10.167)	3 (0.25)	

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 (%)	*P*-value
Group 1	34 (2.83)	0 (0)	0 (0)	0 (0)	<0.001
Group 2	177 (14.75)	11 (0.92)	0 (0)	0 (0)	
Group 3	39 (3.25)	82 (6.83)	48 (4)	31 (2.58)	
Group 4	110 (9.17)	76 (6.33)	5 (0.417)	4 (0.33)	
Group 5	21 (1.75)	0 (0)	0 (0)	0 (0)	
Group 6	168 (14)	18 (1.5)	7 (0.58)	0 (0)	
Total	549	187	60	35	

in Group 5 (4.83%) and Group 1 (4.083%). However, moderate gingivitis was notably higher in Group 1 (8%) compared to Group 5 (1.167%). Severe gingivitis remained rare across all cohorts, with the highest incidence observed in Group 1 (0.167%), followed by Group 6 (0.083%) [Table 2 and Figure 3].

The prevalence of periodontitis by Grade is detailed in Table 3 and Figure 4. Grade I periodontitis was most frequent in Group 2 (14.75%), followed by Group 6 (14%) and Group 4 (9.17%). However, a shift toward higher disease severity was observed in Group 3, which exhibited the highest prevalence of Grade II (6.83%), Grade III (4%), and Grade IV (2.58%) periodontitis. Following Group 3, Group 4 consistently showed the next highest

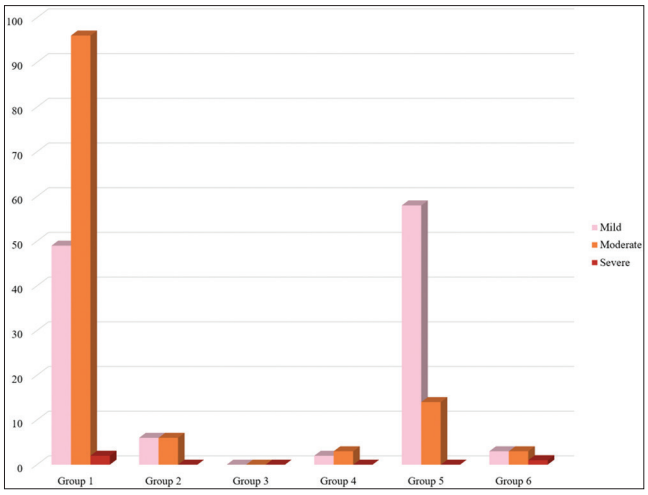

Figure 2: Distribution of gingivitis among all the groups in the study.

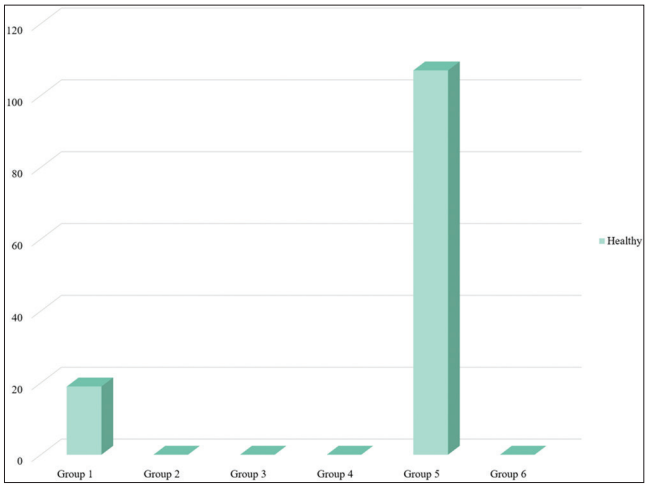

Figure 3: Distribution of periodontal health among all the groups in the study.

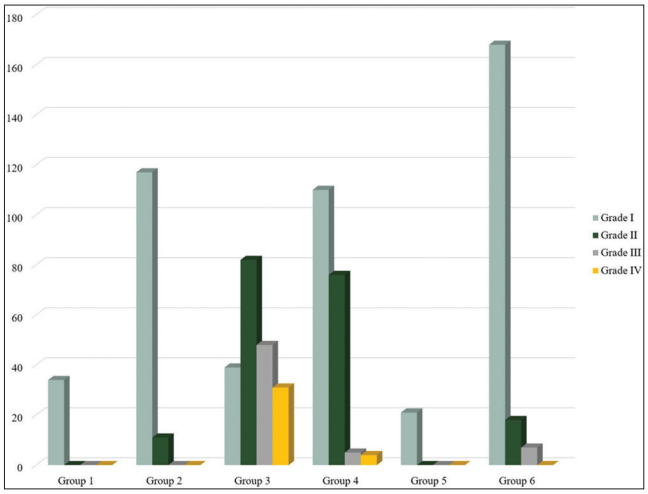

Figure 4: Distribution of periodontitis among all the groups in the study.

rates for Grade II (6.33%) and Grade IV (0.33%) disease, while Group 6 presented the second-highest prevalence for Grade III (0.58%).

DISCUSSION

Periodontal diseases, ranging from gingivitis to chronic periodontitis, represent a major public health challenge. Given their high global prevalence, these conditions are of significant concern not only for oral health but also for 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 immunohomeostasis, 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 (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 c- Jun N-terminal Kinase (JKN) and Inhibitor of nuclear factor kappa- B kinase (INK) β pathways and drives atherogenesis through endothelial cell activation and oxidative stress. The observation that Group 5 (8.917%) and Group 1 (1.583%), as shown in Table 1 and Figure 2, exhibited the most favorable periodontal parameters suggests a potential genetic protective phenotype linked to the ABO-H histo-blood group system. 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 5 (AB phenotype) 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 5 (4.83%), followed by Group 1 (4.083%), showed the highest number of gingivitis, as shown in Table 2 and

Figure 3. Similar observations have been made by Demir *et al.*[12] and Koregol *et al.*[13] who conclude 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. Genetic and epistatic factors: As mentioned earlier regarding Group 5 and the ABH substances is scientifically linked to the *FUT2* gene, which determines an individual's secretor status. In secretors, ABH antigens are found in saliva; these soluble antigens act as decoys that bind to bacterial lectins, preventing the bacteria from attaching to the actual gingival tissue.[16] In addition, the non-secretors lack these salivary decoys and often exhibit lower levels of salivary Secretory IgA, which significantly increases the risk for chronic inflammatory infiltration and periodontitis.
3. 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 NF-kB activation in gingival fibroblasts.[17]

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.[18] In the present study, it was observed that the highest number of patients having periodontitis belonged to Group 3, followed by Group 4 and Group 6, as shown in Table 3, and illustrated in Figure 4, with the highest number of grade IV patients in Groups 3 and 4. Similar observations have been made in the studies conducted by Ghamdi[19] and Arowojol*u et al.*,[20] 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:[21]

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 such as *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 pro-inflammatory cytokines such as IL-1β in the gingival crevicular fluid.

Key Messages: The high prevalence of periodontal disease in India, affecting approximately 45% of the adult population, demands a transition from traditional curative models to precision-based preventive dentistry. Integrating ABO blood group assessment into routine clinical screening offers a non-invasive, cost-effective framework for early risk stratification.

CONCLUSION

Based on the clinical parameters analyzed in the study, a distinct hierarchy of periodontal susceptibility emerges, stratified by the genotype. The severity of periodontal destruction is more severe in Groups 3, 4, and 6 (I^BI^B, I^Bi, ii), while Groups 1 and 2 (I^AI^A, I^Ai) are associated with gingivitis and Group 5 (I^AI^B). 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.

REFERENCES

1. Gautam A, Mittal N, Singh TB, Srivastava R, Verma PK. Correlation of ABO blood group phenotype and rhesus factor with periodontal disease: An observational study. Contemp Clin Dent 2017;8:253-8.
2. Anup P, Siddhartha V, Girish S, Keshava A, Sameer Z, Vishwajeet K. ABO and Rh blood group system and periodontal disease - a prevalence study. J Adv Med Med Res 2016;16:1-6.
3. Tonetti MS, Jepsen S, Jin L, Otomo-Corgel J. Impact of the global burden of periodontal diseases on health, nutrition and wellbeing of mankind: A call for global action. J Clin Periodontol 2017;44:456-62.
4. Landsteiner K. On the knowledge of the anti-fermentative, lytic and agglutinating effects of blood serum and lymph. Cent J Bacteriol Parasitol Infect Dis 1990;27:357-62.
5. Kinane DF, Bartold PM. Clinical relevance of the host responses of periodontitis. Periodontol 2000 2007;43:278-93.
6. Roberts JA. Blood groups and susceptibility to disease: A review. Br J Prev Soc Med 1957;11: 107-25.
7. Offenbacher S. Periodontal diseases: Pathogenesis. Ann Periodontol 1996;1: 821-78.
8. Shaju JP, Zade RM, Das M. Prevalence of periodontitis in the Indian population: A literature review. J Indian Soc Periodontol 2011;15:29-34.
9. Grisi DC, Salvador SL, Figueiredo LC, Souza1 SL, Novaes AB, Grisi MF. Effect of a controlled-release chlorhexidine chip on clinical and microbiological parameters of periodontal syndrome. J Clin Periodontol 2002;29:875-81.
10. Gawrzewska B. ABO, Rh and MN blood and ABH group factors in saliva related to periodontal diseases. Czas Stomatol 1975;28(10):1007-14.
11. Vivek S, Jain J, Simon SP, Battur H, Supreetha S, Haridas R. Association of ABO blood group and Rh factor with periodontal disease in a population of Virajpet, Karnataka: A cross-sectional study. J Int Oral Health 2013; 5(4):30-4.
12. Demira T, Tezela A, Orbaka R, Eltasa A, Karaa C, Kavrut F. The effect of ABO blood types on periodontal status. Eur J Dent 2007;1:139-43.
13. Koregol AC, Raghavendra M, Nainegali S, Kalburgi N, Varma S. ABO blood groups and Rhesus factor: an exploring link to periodontal diseases. Indian J Dent Res. 2010; 21(3):364-8.
14. Clark L, Dean A, Mitchell A, Torgerson DJ. Envelope use and reporting in randomised controlled trials: A guide for researchers. Res Methods Med Health Sci 2020;2:2-11.
15. Hujoel PP, Cunha-Cruz J, Loesche WJ, Robertson PB. Personal oral hygiene and chronic periodontitis: A systematic review. Periodontol 2000 2005;37:29-34.
16. Eid HA. Effects of rhesus factor (Rh) and blood group type on gingival health status. Egypt Dent J 2011;57:1-6.
17. Dhingra K, Vandana KL. Indices for measuring periodontitis: A literature review. Int Dent J 2011;61:76-84.
18. Arowojolu MO, Dosmu EB, Adingbola TS. The relationship between juvenile and non-juvenile periodontitis, ABO blood groups and haemoglobin types. Afr J Med Med Sci 2002;31:249-52.
19. Ghamdi AS. Association between ABO blood groups and severity of chronic periodontitis. J King Abdulaziz Univ Med Sci 2009; 16:31-41.
20. Arowojolu MO, Dosmu EB, Adingbola TS. The relationship between juvenile and non-juvenile periodontitis, ABO blood groups and haemoglobin types. Afr J Med Med Sci 2002; 31:249-52.
21. Rheu GB, Ji S, Ryu JJ, Lee JB, Shin C, Lee JY, *et al*. Risk assessment for clinical attachment loss of periodontal tissue in Korean adults. J Adv Prosthodont 2011;3:25-32.

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