ABO Blood Group System and Periodontal Disease Indices: A Cross-Sectional Study in Greek Adults

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Received date: 03 July 2021; Accepted date: 02 August 2021; Published date: 08 August 2021


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Abstract

Introduction: Periodontal disease (PD) development has been associated with the presence of causative microorganisms, host immunity and risk factors, whereas the types of periodontal diseases are characterized by interactions between host and bacteria. Moreover, ABO blood groups are the most investigated erythrocyte antigen system. However, a small number of researches have been focused on the possible associations between ABO blood groups and periodontal diseases.

Methods: A cross-sectional, epidemiological study was carried out on 854 individuals, 404 males and 450 females, aged 45 to 77. The study sample was interviewed and underwent an oral and dental clinical examination. The assessment of the possible associations between several indices of PD, such as Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL) and Bleeding on Probing (BOP) as dependent variables and ABO blood groups A, B, AB, and O as independent ones was carried out by using a multiple regression analysis model.

Results: Individuals with blood group A [OR= 2.94, 95% CI = 1.27-3.96] and B [OR= 2.66, 95% CI = 1.11-3.87] were significantly associated with the risk of developing deeper periodontal pockets (PPD) and worse values of attachment loss (CAL) [OR= 2.42, 95% CI= 1.37-3.85] and [OR= 2.31,95% CI=1.25-3.68], respectively. However, no significant associations were recorded between ABO blood groups and BOP [OR= 1.04, 95% CI= 0.92-1.18].

Conclusion: A significant association was revealed between A and B blood groups and deeper periodontal pockets and worse attachment loss, whereas no associations were observed between ABO blood groups and bleeding of probing.

Keywords
ABO Blood Group System, Periodontitis, Genetic Factors, Risk Factor, Adults

Introduction

Periodontal disease (PD) is characterized by a multifactorial etiology and affects a large population worldwide. Dental plaque accumulation is the main etiologic factor; however genetic factors seem to play an important role in PD pathogenesis [1].
Consequently, it would be interesting to focus on the influence of genetic factors in PD patients and to investigate the possible association between those and PD. One of those factors is the ABO blood group and it would be important to investigate if the antigens of the ABO blood group have somehow increased the susceptibility to the PD. The ABO blood groups system discovered decades ago [2] and its antigens are biochemical indices that are expressed in several cell types including erythrocytes, gastrointestinal cells, lung epithelial cells, mucosa cells, plasma and other body fluids [3].

Between the presence or absence of the ABO blood group antigens and several diseases and disorders an association has been recorded, whereas those antigens also can be acted as receptors for infectious agents. An increasing number of researchers have recorded that the ABO blood group is involved in several disorders and pathological condition as it has already mentioned and the possible link between ABO blood group and susceptibility to chronic disease as an example of genetic basis for family predisposition, has also been investigated [4]. To be more specific an association between inherited human ABO blood group antigens with diseases such as coronary heart disease [5], ischemic stroke [6,7] and several types of malignancies [2,8-15] has been recorded.

However, a small number of studies have investigated the relationship and the incidence of oral and dental diseases such as PD and the possible association with ABO blood group. Significant associations have been recorded between ABO blood group system and several oral diseases such as dental caries [16] salivary gland tumours [17], and oral cancer [18]. Moreover, significant associations have been found between ABO blood group and PD [16,19-25]. On the contrary, few studies have shown no significant associations between ABO blood group and PD [26-28]. The mentioned controversial findings could be attributed to the geographic diversity in the population groups.

The purpose of the present study was to investigate the possible association of some PD indices with different ABO blood groups in a sample of Greek adults. Such an investigation may be being helpful to better understand the risk factors of PD and to predict the effective methods for its prevention and treatment.

Methodology

Study Design and Study Population Sample:

A cross-sectional, epidemiological study was carried out between 2019 and 2020. The study size was estimated considering the PD prevalence determined by Hyman et al. [29], with 90% confidence interval and relative precision 20.0%, whereas the age group was based on the World Health Organization (WHO) recommendations [30,31] for assessing disease prevalence. This procedure led to a study sample of 854 individuals [29]. The current investigation was carried out on 404 males and 450 females, aged 45 to 77. Participants, were out-patients of a dental and two private medical practices, completed a health questionnaire and underwent an oral clinical examination.

Individuals Selection Criteria:

Participants should have at least 20 natural teeth excluding the 3rd molars and remaining roots in order to be included in the study [32].

The criteria of established periodontitis [33], which referred to at least 2 teeth with CAL ≥ 6mm and more than one site with PPD ≥ 5mm was the main inclusion criterion for the participants. Individuals suffering from cardiovascular diseases, diabetes mellitus, acute infections, liver cirrhosis, malignancies, rheumatoid arthritis, immunosuppressed individuals because of recent transplantation or haematological malignancies and those who received medical treatment for the mentioned diseases, and general glucocorticoids were excluded from the study protocol.

They also excluded individuals who underwent scaling and root planning or surgical periodontal treatment within 6 months before their examination or those with prescription of anti-inflammatory or systemic antibiotics or other systemic drugs within the past 6 weeks. Those criteria were applied in an attempt to eliminate or avoid as much possible potential confounding influences on the study indices examined.
Clinical Study

Groups selection was based on the friendly and collegial environment in an effort to control potential confounders such as socioeconomic level, smoking, etc.

Oral and Dental Clinical Examination:

A well-trained and calibrated dental surgeon carried out the dental and oral examination at the mentioned practices. The Periodontal examination assessed probing pocket depth (PPD), clinical attachment loss (CAL) and bleeding on probing (BOP), and was measured by a William’s 12 PCP probe (PCP 10-SE, Hu-Friedy Mfg. Co. Inc. Chicago, IL, USA) at six sites (facial, lingual, disto-facial, mesio-facial, disto-lingual and mesio-lingual).

The presence of PPD was coded as [34]: - score 0: moderate periodontal pockets, 4.0-6.0 mm and - score 1: advanced periodontal pockets, >6.0 mm.

The severity of CAL coded as [35]: - score 0: mild, 1.0-2.0 mm of attachment loss, and - score 1: moderate/severe, ≥ 3.0 mm of attachment loss. PPD and CAL measurements concerned the immediate full millimeter, whereas the presence/absence of BOP was coded as - score 0: absence of BOP, and - score 1: presence of BOP and deemed positive if it occurred within 15 seconds of probing.

Research Questionnaire:

Individuals filled in a self-administered questionnaire that included variables such as age, gender, smoking status (active, former / no-smokers), socio-economic and educational level and data regarding their general medical history with reference to ABO blood group, medication, several chronic systemic diseases/disorders and the frequency of their dental follow-up. For establishment of the intra-examiner variance a randomly chosen sample of 171 (20%) individuals were re-examined clinically by the same dentist after 3 weeks, and no differences were recorded between the 1st and the 2nd clinical assessment (Cohen’s Kappa= 0.96).

During this time period no oral hygiene instructions were given to the participants.

Ethical Consideration:

The present cross-sectional study was not reviewed and approved by authorized committees (Ministry of Health, etc.), as in Greece only experimental studies must be approved by those Authorities. An informed consent form was obtained by the individuals who agreed to participate in the current study.

Statistical Analysis:

For each individual, the worst values of PPD and CAL on six sites per tooth and the presence/absence of BOP were recorded and coded as dichotomous variables. Current and former smokers were coded as 1, individuals with a high socio-economic (income/monthly ≥ 1,000 €) and educational (graduated from University/College) level were coded as 0, male’s participants were coded as 1, and individuals that reported a regular dental follow-up were coded as 1. Age groups distribution was coded as 0, 1, 2 and 3 for ages 45-49, 50-59, 60-69 and 70+ respectively.

Univariate analysis was carried out to test the relationship between the independent variables examined and the ABO blood group, separately, by using $x^2$ test.

Multivariate regression analysis was carried out to model the associations between the dependent PD variables, and independent ones that were determined by the enter method. Adjusted Odds Ratios (OR’s) and 95% (Confidence Interval) CI were also calculated. Finally, the independent variables were included to stepwise method in order to estimate gradually the variables that showed significant associations with the dependent ones.

Statistical analysis was performed using the statistical package of SPSS ver.19.0. A p-value of less than 5% ($p < 0.05$) was considered significant for all statistical test conducted.

Results

Table-1 presents univariate analysis of the examined variables. Age of participants was statistically significantly associated with PPD and BOP, low socioeconomic level was statistically significantly
Table-1: Univariate analysis of cases and controls regarding each independent variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>PPD (mm)</th>
<th>CAL (mm)</th>
<th>BOP (+/-)</th>
<th>BOP (+/-)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5.0 (no) (%)</td>
<td>&gt;5.0 (no) (%)</td>
<td>&lt;3.0 (no) (%)</td>
<td>&gt;3.0 (no) (%)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>198 (55.0)</td>
<td>252 (51.0)</td>
<td>215 (53.3)</td>
<td>235 (52.0)</td>
</tr>
<tr>
<td>Males</td>
<td>162 (45.0)</td>
<td>242 (49.0)</td>
<td>187 (46.5)</td>
<td>217 (48.0)</td>
</tr>
<tr>
<td></td>
<td>P = 0.249</td>
<td>OR = 1.174</td>
<td>P = 0.663</td>
<td>OR = 1.062</td>
</tr>
<tr>
<td></td>
<td>95% CI = 0.89-1.54</td>
<td>95% CI = 0.81-1.39</td>
<td>95% CI = 0.75-1.32</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>45-49</td>
<td>83 (23.1)</td>
<td>169 (34.2)</td>
<td>116 (28.9)</td>
<td>136 (30.0)</td>
</tr>
<tr>
<td>50-59</td>
<td>108 (30.0)</td>
<td>130 (26.3)</td>
<td>107 (26.6)</td>
<td>131 (29.0)</td>
</tr>
<tr>
<td>60-69</td>
<td>127 (35.3)</td>
<td>137 (27.7)</td>
<td>126 (31.3)</td>
<td>138 (30.5)</td>
</tr>
<tr>
<td>70+</td>
<td>42 (11.7)</td>
<td>58 (11.7)</td>
<td>53 (13.2)</td>
<td>47 (10.5)</td>
</tr>
<tr>
<td></td>
<td>P = 0.003*</td>
<td>P = 0.574</td>
<td>P = 0.000*</td>
<td></td>
</tr>
<tr>
<td>Socio-economic level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>182 (50.6)</td>
<td>268 (54.3)</td>
<td>207 (51.5)</td>
<td>243 (53.8)</td>
</tr>
<tr>
<td>Low</td>
<td>178 (49.4)</td>
<td>226 (45.7)</td>
<td>195 (48.5)</td>
<td>209 (46.2)</td>
</tr>
<tr>
<td></td>
<td>P = 0.285</td>
<td>OR = 0.862</td>
<td>P = 0.507</td>
<td>OR = 0.913</td>
</tr>
<tr>
<td></td>
<td>95% CI = 0.66-1.13</td>
<td>95% CI = 0.70-1.20</td>
<td>95% CI = 1.75-3.15</td>
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<tr>
<td>Educational level</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>203 (56.4)</td>
<td>247 (50.0)</td>
<td>238 (59.2)</td>
<td>212 (46.9)</td>
</tr>
<tr>
<td>Low</td>
<td>157 (43.6)</td>
<td>247 (50.0)</td>
<td>164 (40.8)</td>
<td>240 (53.1)</td>
</tr>
<tr>
<td></td>
<td>P = 0.065</td>
<td>OR = 1.293</td>
<td>P = 0.000*</td>
<td>OR = 1.643</td>
</tr>
<tr>
<td></td>
<td>95% CI = 0.98-1.70</td>
<td>95% CI = 1.25-2.16</td>
<td>95% CI = 2.45-4.51</td>
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</tr>
<tr>
<td>Smoking</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>216 (60.0)</td>
<td>234 (47.4)</td>
<td>195 (48.5)</td>
<td>255 (56.4)</td>
</tr>
<tr>
<td>Yes</td>
<td>144 (40.0)</td>
<td>260 (52.6)</td>
<td>207 (51.5)</td>
<td>197 (43.6)</td>
</tr>
<tr>
<td></td>
<td>P = 0.000*</td>
<td>OR = 1.667</td>
<td>P = 0.021*</td>
<td>OR = 0.728</td>
</tr>
<tr>
<td></td>
<td>95% CI = 1.27-2.19</td>
<td>95% CI = 0.56-0.95</td>
<td>95% CI = 3.91-7.48</td>
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</tr>
<tr>
<td>Dental follow-up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annual</td>
<td>258 (71.7)</td>
<td>192 (38.9)</td>
<td>214 (53.2)</td>
<td>236 (52.2)</td>
</tr>
<tr>
<td>2 times/year</td>
<td>102 (28.3)</td>
<td>302 (61.1)</td>
<td>188 (46.8)</td>
<td>188 (46.8)</td>
</tr>
<tr>
<td>&lt;2 times or no/year</td>
<td>P = 0.000*</td>
<td>OR = 3.979</td>
<td>P = 0.765</td>
<td>OR = 1.042</td>
</tr>
<tr>
<td></td>
<td>95% CI = 2.97-5.33</td>
<td>95% CI = 0.80-1.36</td>
<td>95% CI = 1.44-2.57</td>
<td></td>
</tr>
</tbody>
</table>

*p-value: statistically significant
Clinical Study

associated with BOP, low educational level was statistically significantly associated with CAL and BOP, smoking was statistically significantly associated with PPD, CAL and BOP, irregular dental follow-up was statistically significantly associated with PPD and BOP and, ABO blood group was also statistically significantly associated with PPD, CAL and BOP. Table-1 also presents unadjusted OR's and 95% CI.

Table-2 also presents adjusted OR's with 95% CI. Table-4 shows that no one of the ABO blood groups were significantly associated with BOP index.

Discussion

Male gender, advanced age and low socio-economic status (SES) have been found to be significantly associated with PD, according to previous reports, and many of those have revealed more serious periodontal destruction among males compared to females [36-38]. Those differences may be explained by the facts that females usually are more aesthetically conscious,

<table>
<thead>
<tr>
<th>Step 4a</th>
<th>Variables in the Equation</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% C.I.for EXP(B)</th>
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<td>Lower</td>
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<tr>
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<td>.219</td>
<td>.172</td>
<td>.347</td>
<td>1</td>
<td>.023*</td>
<td>1.976</td>
<td>1.171</td>
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<td>educ_level</td>
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<td>-1.546</td>
<td>.180</td>
<td>73,660</td>
<td>1</td>
<td>.000*</td>
<td>4.694</td>
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<tr>
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<td>.176</td>
<td>1,52,428</td>
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<td>.000*</td>
<td>8.736</td>
<td>6.193</td>
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<td>ABO_bloodgroup (O)</td>
<td></td>
<td>11,422</td>
<td>3</td>
<td>.955</td>
<td>1</td>
<td>.000</td>
<td>.112</td>
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<td>ABO_bloodgroup(A)</td>
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<td>.661</td>
<td>.216</td>
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<td>.002*</td>
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<td>.145</td>
<td>.246</td>
<td>.348</td>
<td>1</td>
<td>.045*</td>
<td>2.656</td>
<td>1.114</td>
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<td>ABO_bloodgroup(AB)</td>
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<td>.098</td>
<td>.272</td>
<td>.130</td>
<td>1</td>
<td>.718</td>
<td>.647</td>
<td>1.882</td>
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<td>Constant</td>
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<td>.243</td>
<td>80,900</td>
<td>1</td>
<td>.000</td>
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a. Variable(s) entered on step 4a: smok_stat, educ_level, dent_followup, ABO_bloodgroup

<table>
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<th>Step 4a</th>
<th>Variables in the Equation</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% C.I.for EXP(B)</th>
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<td>Lower</td>
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<tr>
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<td></td>
<td>.182</td>
<td>.170</td>
<td>6,812</td>
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<td>.009*</td>
<td>1.834</td>
<td>1.328</td>
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<tr>
<td>educ_level</td>
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<td>5,012</td>
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<td>.025*</td>
<td>1.373</td>
<td>1.040</td>
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<tr>
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<td>.145</td>
<td>10,801</td>
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<td>.001*</td>
<td>1.611</td>
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<td>ABO_bloodgroup (O)</td>
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<td>11,422</td>
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<td>.955</td>
<td>1</td>
<td>.000</td>
<td></td>
<td></td>
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<td>ABO_bloodgroup(A)</td>
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<td>.521</td>
<td>.212</td>
<td>7,123</td>
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<td>.253</td>
<td>.398</td>
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<td>.031*</td>
<td>2.311</td>
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<td>ABO_bloodgroup(AB)</td>
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<td>.188</td>
<td>.281</td>
<td>.231</td>
<td>1</td>
<td>.628</td>
<td>1.207</td>
<td>.597</td>
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<td>Constant</td>
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<td>2.271</td>
<td>.238</td>
<td>78,321</td>
<td>1</td>
<td>.000</td>
<td>.138</td>
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</tr>
</tbody>
</table>

a. Variable(s) entered on step 4a: smok_stat, educ_level, dent_followup, ABO_bloodgroup
males have poorer oral hygiene practices than females, access to dental care is different between both genders [39] and also it could be attributed to the ignorance of oral hygiene, which is usually observed among males [40]. However, gender is a demographic variable, which may interfere with the effects of other risk factors and it might also be acting as a confounder.

PD affects approximately up to half of adults over 50 years of age, one-third of adults over 30 years of age [41] and it has also been estimated that affects 30%-35% of dentate US adults [42]. Advancing age is a known PD risk factor [43], although this association could be attributed to the cumulative periodontal breakdown over time than to an age-related, intrinsic deficiency that contributes to susceptibility to PD [1].

Previous studies have recorded significant associations between socio-economic status (SES) and PD severity [44-48]. However, few studies have shown a weak association between SES and periodontitis after adjustment for oral hygiene and smoking [1,49]. Similar association has been recorded with other socio-economic parameters such as income and educational level and could be attributed to the close relation between income, educational level, and occupation [50]. Higher SES individuals wish to have better periodontal status and this is in accordance with the general belief that those individuals have healthier behaviours than individuals with lower SES [51].

Jiang et al. [52] found that risks of oral diseases increase in lower educational or academic training patients, or lack health insurance access. A higher prevalence of PD among individuals with lower educational level than among the ones with higher educational level has been observed [47,49]. Moreover, worse PD indices in individuals lacking education, or with basic primary education has also been recorded [53]. Similar studies have revealed that low educational level was significantly associated with CAL severity [36,45,47,48], whereas individuals with lower school educational levels were 3 times more susceptible to suffer from PD than those with higher educational level [48]. Individuals who have higher educational level, and live under more favourable conditions, show better health conditions than the ones who have lower educational level and live under less favourable conditions [54]. It is supposed that high-educated individuals take care of their own oral

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### Table 4: Associations between ABO blood group and BOP

<table>
<thead>
<tr>
<th>Variables in the Equation</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% C.I. for EXP(B)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lower</td>
</tr>
<tr>
<td>Step 1*</td>
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<td></td>
<td></td>
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<tr>
<td>gender</td>
<td>0.017</td>
<td>0.155</td>
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<td>1</td>
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<tr>
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<td>1.093</td>
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<td>0.108</td>
<td>1.287</td>
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\* Variable(s) entered on step 1: gender, age, smok_stat, educ_level, socio_econ_l, dent_followup, ABO_bloodgroup
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hygiene more than low-educated ones [55], who had difficulties with their access to social health services [53].

No significant associations were recorded among the mentioned risk factors and PD indices examined in the current research after performing of the multivariate logistic regression model, except for the educational level, as low educational level was significantly associated with the worse values of PPD, CAL and BOP.

The relationship between smoking and periodontal health has been investigated and a large number of epidemiological, clinical and in vitro studies have provided strong evidence that smoking negatively affects periodontal health and suggest mechanisms by which this may occur [46,56,57]. Many epidemiological studies have revealed that smoking is a crucial risk factor for PD [36,44,58-63].

Various mechanisms have been proposed to explain the role of smoking in PD pathogenesis and progression. This is might be related to the crucial role of smoking by affecting host immune responses [60]. However, to date it remains unclear the mechanism which explain how smoking may affect PD. It is possible that genetic susceptibility and genetic polymorphisms may explain the mentioned influence [1]. Cigarette smoking affects the inflammatory and immune responses, as well as the microvasculature [64], causes an essential destructive effect on the periodontal tissues and contributes to PD progression. It seems that cigarette smoking modifies the host’s response to the bacteria in dental plaque [65]. In the present study, a significant association between smoking and PD indices was recorded.

Lack of a regular dental follow-up was significantly associated with worse values of PD indices examined findings that were in agreement with the findings of a previous study [44]. Only one study recorded different findings [66]. As it has already mentioned individuals with a higher SES wish to have better periodontal health, have healthier oral behaviours and lifestyles than those with a lower SES [51]. Tooth brushing is essential for periodontal health maintenance as can reduce dental plaque accumulation and in turn can prevent gingivitis and periodontitis [67].

The aim of the current report was to investigate the possible association between ABO blood groups and PD indices. Only few studies have investigated the relationship between ABO blood group and oral and dental diseases.

Decades ago, Suk [68] suggested that ABO blood groups had an increased effect on the risk for the development of oral diseases, whereas an association between the patient’s susceptibility to dental caries and his ABO blood group was also observed [69]. In another early research [70] a significant association between M.N. blood groups and dental caries history was recorded. The outcomes of the current research showed that A and B blood groups were significantly associated with PD indices, such as pocket depth and attachment loss which are related to the criteria of established periodontitis. More specific, the current research showed that individuals with A and B blood groups had worse values of pocket depth (PPD) ≥ 5.00 mm and CAL ≥ 6.00mm, compared with those with O and AB blood groups, whereas no associations were observed between ABO blood groups and BOP. Those outcomes were in agreement with previous studies [25,71].

Vivek et al. [23] in a recent article found that individual’s blood group O had a greater trend for periodontitis, whereas similar studies [21,72] recorded that blood group O may act as a predictive factor for PD development. However, the evidence of such a role requires multicentre collaborative studies that should include diverse population groups from multiple geographic regions and should explore whether there is any genetic basis for that relationship.

Similar researches showed that the mean CAL and the mean proportion of sites with CAL ≥ 3.00mm were greatest among patients with blood group B [24,73], and a significant association between increased incidence of Aggressive Periodontitis (AP) and blood group B, whereas in blood group O found reduced incidence of AP [20]. Moreover, some investigators demonstrated that patients with blood group B had a higher risk of developing periodontitis [25,74,75], and
greater pocket depth [73].

Few studies showed no significant associations between individuals with PD and ABO blood group [26-28,76], whereas in a study by Mortazavi et al. [77] was recorded that periodontitis did not show any relationship with blood groups despite the fact that the most frequent blood group with periodontitis was O.

On the contrary, Gawrzewska [78] found that individuals with blood group O had greater severity of PD, whereas individuals with blood group A had greater resistance to PD. Similarly, in a recent study was recorded a higher percentage of blood type A in patients with gingivitis and a higher percentage of blood type O in patients with periodontitis [16].

Kundu et al. [79] recorded that patients with AP most frequently had group AB (60%) or group O (40%) blood type. Similar articles revealed that periodontitis was more common among patients with blood group O [19,23], and in a recent one was recorded that blood group O individuals were at greater risk for developing Chronic Periodontitis (CP) irrespective of its severity, followed by those with blood group A, B, and AB [80].

A superior predisposition for periodontitis was also recorded in individuals with blood group O [81]. Nevertheless, the outcomes were controversial as were based on the type of disease and could be attributed to geographical diversity between populations. Microorganisms are involved in PD pathogenesis, however its progression is associated with host’s risk factors, observation that shows its multifactorial nature [82]. Systemic, local, genetic and environmental factors are involved in causing PD, however the primary factor is bacterial plaque accumulation [83].

Some individuals are at relatively high risk for developing PD as its clinical spectrum is wide. In high risk individuals, host’s factors seem to play an important role in susceptibility to periodontitis and this risk may be partly under genetic control [84]. However, in case such an association between ABO blood groups and PD can be established, it can be concluded that the presence of particular ABO blood group antigen has somehow increased the susceptibility to the disease. The association between ABO blood group and their susceptibility to chronic disease as an example of genetic basis for family predisposition has been suggested decades ago [4]. Genetic influences such as blood group antigens may act as a risk factor that affects the development and severity of chronic periodontitis [80]. The presence or absence of certain antigens has been associated with various diseases and disorders and these antigens also act as receptors for infectious agents associated with PD. Hellstrom et al. [85] found that carbohydrates act as receptors for Porphyromonas gingivalis and these carbohydrate receptors constitute the ABO antigens.

The antigens in the tissues are related to the ABO blood group, and the tissue expression is dependent on the individual’s secretor status. Secretor status refers to the secretion of blood group antigens ABO (H), that may influence the development of systemic oral diseases in the oral epithelium [85]. Differential secretion of blood group antigens ABO (H) in the tissues may be a factor influencing the development of systemic oral diseases [86]. Campi et al. [86] and Demir et al. [16] observed that different ABO blood groups may show significant differences in the rates of colonization of a number of periodontal pathogens that are the main etiologic factors of PD.

A major limitation of the present study is that clear data interpretation was not possible, as a reference group comprising periodontally healthy individuals was not assessed (case-control design). Moreover, another disadvantage of such studies is the presence of selection biases and their retrospective design. The most important problem with this type of study is the differentiation between cause and effect from simple association.

It is very difficult to elaborate a hypothesis on why individuals with particular ABO blood group are found at an increased frequency in healthy, gingivitis, and periodontitis groups, and also in various grades of periodontal involvement. However, occurrence of gingivitis and periodontitis is the result of many factors and the probable genetic influence
demonstrates a small rate of multifactorial etiology of periodontitis. Since most of those studies were carried out on a small group of individuals, until universal epidemiological studies will be available, the decision as to whether a particular blood group has a particular immunity or susceptibility should be postponed. Further, long-term studies with larger sample size are required to confirm this conclusion and investigate the biological plausibility to explain this association.

Conclusions

A significant association was recorded between A and B blood groups and deeper periodontal pockets and worse attachment loss, whereas no associations were observed between ABO blood groups and bleeding of probing.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author has no conflicts of interest to declare.

References


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[68] Suk V. Über die beziehung zwischen gesunden Zähnen und den Zerfall und die Pflege der Zähne bei den weissen. Spisy lék Fak Masaryk Univ. 1930;125.


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