Evaluation of the Relationship between Periodontal Parameters and Liver Cirrhosis

Seyed Ali Banihashemrad1* • Abbas Shirdel2 • Samira Pakro3

1Assistant Professor, Department of Periodontics, School of Dentistry and Dental Research Center, Mashhad University of Medical Sciences, Mashhad, Iran
2Associate Professor, Department of Internal Medicine, Ghaem Hospital, Mashhad, Iran
3Dentist, Private Practice, Mashhad, Iran
*Corresponding author; E-mail: banihashemrad@yahoo.com

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Abstract

Background and aims. Liver cirrhosis and other chronic liver diseases are sometimes detected with oral manifestations, and in recent years, cirrhosis has been recognized as a potential predisposing condition for periodontal disease. The purpose of this study was to determine the periodontal parameters in cirrhotic patients.

Materials and methods. In this study, twenty hospitalized patients with liver cirrhosis (case group) and 20 healthy individuals (control group) were selected and matched by age, gender, oral hygiene, and smoking habit. All subjects were non-alcoholic. The diagnosis of liver cirrhosis was confirmed histologically, and no other systemic diseases were present. Periodontal indices including probing depth (PPD), clinical attachment level (CAL), bleeding points index (BPI), and plaque index (PI) were recorded in both groups. The data were analyzed using Mann-Whitney and t-test.

Results. Mean CAL in case and control groups were 5.05 ± 0.33 mm and 4.90 ± 0.32 mm, respectively (P > 0.05). Mean PPD was 4.37 ± 0.20 mm in case and 4.28 ± 0.18 mm in control group (P > 0.05). Mean BPI was found to be 21.1% and 19.9% in the case and control group, respectively (P > 0.05). Mean PI was 21.55 in case and 19.45 in control subjects (P > 0.05).

Conclusion. According to the results of this study, it could be concluded there are no significant differences between cirrhotic and healthy subjects in terms of periodontal disease parameters.

Key words: Cirrhosis, clinical attachment level, plaque index, probing depth.

Introduction

Periodontal disease is a microbial infection caused by gram-negative bacteria. Bacterial infections are frequent complications in patients with cirrhosis, especially in liver cirrhosis. A potential source of infection may be dental foci.1 Liver cirrhosis and other chronic liver diseases sometimes are detected with oral manifestations. In recent years, cirrhosis has been recognized as a potential reservoir for periodontal disease.2 However, the effect of cirrhotic liver disease, liver transplantation, and immuno-
suppressive therapy on the periodontium is yet unclear.\textsuperscript{3}

The aim of this study was to evaluate the relationship between periodontal parameters and liver cirrhosis in cirrhotic patients and healthy individuals.

**Materials and Methods**

In this study, 20 hospitalized patients with liver cirrhosis (case group) were selected randomly. The diagnosis of liver cirrhosis was confirmed histologically, and no other systemic diseases were present. The control group consisted of 20 healthy subjects referred to Faculty of Dentistry, Mashhad University of Medical Sciences, Mashhad, Iran, at the same time and did not have any sign and symptoms of others diseases. The two groups were matched by age, gender, oral hygiene, and smoking habit. All subjects were non-alcoholic. After recording personal information of the patients, periodontal indices including probing pocket depth (PPD), clinical attachment level (CAL), bleeding points index (BPI),\textsuperscript{4} plaque index (PI),\textsuperscript{5} and gingival index (GI)\textsuperscript{6} were determined in both groups.

Data were analyzed using Mann-Whitney and $t$-test.

**Results**

The mean ages of patients in case and control groups were $43 \pm 2$ and $42 \pm 2$ years, respectively. 85\% of the participants in the case group compared to 75\% of the control group were male. The number of smokers in the case group was 3 (15\%); while there were 5 (25\%) smokers in the control group. 90\% of patients in the case group compared to 75\% of the control group had poor oral hygiene. The data of evaluated criteria are presented in Table 1.

Mean CAL was $5.05 \pm 0.33$ mm in the case and $4.90 \pm 0.32$ mm in the control group ($P = 0.575$). Mean PPD in the case group and control groups were $4.37 \pm 0.20$ mm and $4.28 \pm 0.18$ mm, respectively ($P = 0.744$) (Figure 1). Mean BPI of case and control groups were found to be 21.1 and 19.9, respectively ($P = 0.733$). Mean GI was 20.80 in the case group and 20.20 in the control group ($P = 0.86$). Mean PI in the case group was 21.55 and in the control group 19.45 ($P = 0.744$) (Figure 2). There were no significant differences between the two groups in the evaluated periodontal indices.

**Table 1. The data of assessed variables in case and control groups**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Age (years)</th>
<th>PPD (mm)</th>
<th>CAL (mm)</th>
<th>GI</th>
<th>BPI (%)</th>
<th>PI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case</td>
<td>Mean: 43.70 4.37 5.05 2.30 6.57 7.88</td>
<td>Median: 46 4.24 4.49 2.45 1 1</td>
<td>Mode: 50 4 4 4 1 1</td>
<td>S.D: 10.98 0.89 1.52 1.67 12.24 10.68</td>
<td>Min: 28 2 2.83 0 0 0</td>
<td>Max: 65 6 9.29 4 45.83 39.97</td>
</tr>
<tr>
<td>Control</td>
<td>Mean: 42.60 4.28 4.90 2.21 9.99 5.91</td>
<td>Median: 42.50 4.20 4.73 2.37 1 1</td>
<td>Mode: 32 2.20 2.70 4 1 1</td>
<td>S.D: 10.09 0.84 1.54 1.70 22.57 7.67</td>
<td>Min: 28 2 2.70 0 0 0.35</td>
<td>Max: 61 6 8.98 4 94.73 26.78</td>
</tr>
</tbody>
</table>

**Discussion**

Although a link between periodontitis and inflammatory systemic diseases is supported by some studies and seems biologically plausible, it still remains a is controversial topic.\textsuperscript{7} Now evidence suggests that periodontitis, once established, provides a biological burden of endotoxin and inflammatory cytokines, which serve to initiate and exacerbate atherogenesis and thromboembolic events.\textsuperscript{8}

According to the results of this study, the periodontal indices were quite similar in both groups. The dental and periodontal status of patients with cirrhosis did not differ from the control group. Authors observed that the severity and duration of liver disease had no influence on dental and periodontal disease. These results are partially in agreement with a previous study, which showed the measures of oral hygiene ($P < 0.01$), dental care ($P <
and periodontal parameters were worse and the number of teeth requiring treatment (P < 0.001) was higher in alcoholics with or without cirrhosis than in healthy subjects and nonalcoholic patients with cirrhosis. In the latter study, alcoholics had a lower total number of teeth than patients without alcohol abuse and healthy controls (P < 0.05). In another study, the amount of plaque was equal in test and control groups, whereas the cirrhotics had a higher degree of severity of gingival inflammation as well as a greater amount of subgingival calculus than the controls. Test and control groups exhibited no significant difference with regards to loss of attachment and tooth loss, and similar correlations between loss of attachment and age were demonstrated in the two groups. Patients suffering from cirrhosis for more than 3 years showed significantly greater loss of attachment, as well as more plaque and calculus compared with those with disease duration of less than 3 years.

Other studies have concluded the liver cirrhosis patients demonstrate greater pocketing and attachment loss compared to healthy matched controls. These same differences were observed in post-transplantation patients. Gingival overgrowth occurred as a result of the immunosuppressive therapy with Cyclosporin A, while to a lesser degree with tacrolimus. Replacement of Cyclosporin A by tacrolimus in patients manifesting gingival overgrowth might be recommended whenever possible to overcome this problem. It was shown that the liver cirrhosis patients demonstrated greater bone loss compared to healthy controls and restoration of liver functions following transplantation seems to have the potential to reverse some of these radiographic changes.

Although the evidence is not yet definitive, recent studies have shown that chronic infection by bacterial organisms such as Chlamydia pneumoniae, Helicobacter pneumoniae, Helicobacter pylori and a variety of other dental pathogens may play a causative role in atherosclerosis. The presence of cirrhosis itself, therefore, is not a predisposing factor for dental and periodontal diseases. These diseases may appear to be caused primarily by bad oral hygiene and poor dental care. Further longitudinal studies will be necessary to substantiate these findings.

Conclusion

According to the results of the present study, there is not a significant relationship between periodontal disease and liver cirrhosis. More research is needed to draw any conclusion about the relationship between periodontitis and cirrhosis.

References