Bull Group Int Rech Sci Stomatol et Odontol - Vol 39 n° 3-4, 1996

Dental findings in patients with liver cirrhosis. A study of 100 cases

J.V. BAGÁN*, L. ALAPONT**, J.A. DEL OLMO***, J.M. RODRIGO****,
E. LLORIA*****, Y. JIMÉNEZ******

* *Professor of Oral Medicine and Head Department of Stomatology, Valencia University General Hospital.*
** *Consultant Department of Stomatology, Valencia University General Hospital.*
*** *Consultant Department of Hepatology, Valencia University Clinic Hospital.*
**** *Professor and Head Department of Hepatology, Valencia University Clinic Hospital.*
***** *Oral and maxillofacial surgeon. Consultant Department of Stomatology, Valencia University General Hospital.*
****** *Consultant Department of Stomatology, Valencia University General Hospital.*

SUMMARY

A study was made of the dental findings in 100 patients with liver cirrhosis (LC) by examining the number of carious, missing and filled teeth. A significantly greater number of carious and missing teeth were observed in the patients with cirrhosis than in a control group of 50 healthy individuals. In the LC group, caries were found to affect more teeth in those patients with alcohol-induced LC than in those with liver disease of other causes. Finally, no relationship was observed between the number of carious, missing or filled teeth and certain determinations including serum glutamate pyruvate transaminase (SGPT), serum glutamate oxalacetate transaminase (SGOT), alkaline phosphatase, platelet number, hepatitis B and C positivity markers, or antinuclear (ANA), antimitochondrial (AMA) or anti-smooth muscle autoantibodies (ASm).

KEY WORDS:

Dental caries - Liver cirrhosis.

RÉSUMÉ

Une étude des fiches dentaires de 100 patients présentant une cirrhose hépatique (CH) a été réalisée en examinant l'index des dents cariées, manquantes ou plombées. Une augmentation significative du nombre de dents cariées ou manquantes a été observée chez les patients avec cirrhose, par rapport au groupe de référence composé de 50 individus sains. Dans le groupe de CH, les patients avec cirrhose éthylique avaient un plus grand nombre de caries que les autres pour lesquels l'origine de la maladie hépatique était différente. Finalement aucune relation n'a été observée entre l'index des dents cariées, manquantes ou plombées et certaines déterminations analytiques comme les transaminases alanine aminotransférases (ALAT ou SGPT), les transaminases aspartate aminotransférases (ASAT ou SGOT), les phosphates alcalines, la numération plaquettaire, les marqueurs des hépatites B ou C, ou les anticorps antinucléaires (ANA), anti-mitochondries (AMA) ou antimuscle lisse (ASm).

MOTS CLÉS:

Carie dentaire - Cirrhose hépatique.

INTRODUCTION

The term liver cirrhosis (LC) was proposed by Laennec in 1819; at present, LC may be defined as a clinico-morphological condition comprising a range of liver diseases of different etiologies. In this sense, LC is currently most often caused by alcohol abuse and hepatitis C infection. Initially, only a series of non-pathognomonic stigmas and analytical alterations suggest cirrhosis, including elevated transaminases, anemia, leukopenia and diminished platelet numbers. This may progress to a second phase in which the effects of liver destructurization become apparent (Anthony *et al.* 1977, Erlinger *et al.* 1991, Saunders *et al.* 1981, Sherlock 1993). Cirrhosis often entails a series of extrahepatic manifestations (Goldin *et al.* 1973), thus in the present study we analyzed dental findings in a group of patients with LC, compared with a control group of healthy subjects.

MATERIALS AND METHODS

Dental findings were studied in 100 non-diabetic patients with histopathologically diagnosed LC, and in a series of 50 healthy controls who were receiving no medication and were free of liver disease. There were no significant age or sex differences between the two groups ($p<0.05$).

In the LC group we evaluated the Quick index, platelet number, and the concentrations of serum glutamate pyruvate transaminase (SGPT), serum glutamate oxalacetate transaminase (SGOT), alkaline phosphatase (AP), albumen and total proteins. Tests were also made for the viral hepatitis B and C serological markers (hepatitis B surface antigen, HBsAg; hepatitis B surface antibody, anti-HBs; hepatitis C antibody, anti-HC) and of the following circulating auto-antibodies: antinuclear (ANA), antimitochondrial (AMA) and anti-smooth muscle (ASm). In addition, the time course of the disease was investigated, and all cases attributable to alcohol abuse were identified. In this way, two subgroups were established, according to whether LC was caused by ethanol (30 patients with LC and chronic alcoholism associated or not to viral hepatitis C) or otherwise (70 patients with LC of causes unrelated to alcohol abuse).

In all patients (LC and control), we evaluated dental health by examining the number of carious, missing and filled teeth. We also recorded the plaque index (Silness and Loë, 1964) and the simplified oral hygiene index (Green-Vermeillon, 1964) in both groups. Measurements were all made by the same operator, in order to avoid errors.

The Student t-test was used to compare the means of quantitative variables, while simple regression was applied to determine the relationship between quantitative variables. Statistical significance was considered for $p<0.05$.

RESULTS

The sum of carious, missing and filled teeth in the LC group was significantly greater than among the controls (17.8, st.dev. 9 and 13.5 st.dev. 8.5, respectively; $t=2.69$, $p<0.05$).

The mean number of carious teeth was found to be significantly greater in the patients with liver disease than in the controls (2.4 and 1.3 respectively, $t=2.106$, $p<0.05$). The same applied to the number of missing teeth (14.6 and 10.6 respectively, $t=2.278$, $p<0.05$), though the mean number of filled teeth was lower in the LC group than among the controls (0.86 and 1.5 respectively, $t=-2.25$, $p=0.02$). (Table I).

We found statistically significant differences ($p<0.05$) in the mean number of carious teeth between the two LC subgroups, i.e., with and without alcohol etiology (3.9 and 1.7, respectively). There were no differences, $p>0.05$, in the mean number of missing teeth (14.9 and 14 in the alcoholic and non-alcoholic subgroups, respectively).

TABLE I: Differences between the liver cirrhosis patients and controls.
TABLEAU I: Différences entre les patients cirrothiques et les contrôles.

	Mean carious teeth (*)	Mean missing teeth (*)	Mean filling teeth (*)	Plaque index (*)	SOHI (*)
Liver disease	2,4	14,6	0,86	2,2	1,9
Controls	1,3	10,6	1,5	1,9	1,5

(*) Significant differences ($p<0.05$)
SOHI: Simplified oral hygiene index, Green-Vermeillon 1964.
(*) Différences significatives ($p<0.05$)

In terms of oral hygiene, the patients with LC presented a significantly higher plaque index than the controls without liver disease (2.2 and 1.9 respectively; $t=2.02$, $p=0.04$) (Table I). In addition, more plaque was observed in the patients with LC caused by alcohol than in patients with LC of non-alcoholic origin (2.4 versus 2.1, respectively). The simplified oral hygiene index was significantly greater in the patients with LC than among the controls (1.9 and 1.5, respectively; $t=2.23$, $p=0.02$).

We observed no significant values ($p>0.05$) on correlating the number of carious, missing and filled teeth with the levels of SGOT, SPGT and AP, the Quick index, platelet number, and the evolution time of LC.

Finally, no significant differences were detected in the number of carious, missing or filled teeth in terms of patient positivity or negativity to the hepatitis B and C serological markers, or to the circulating auto-antibodies (ANA, AMA, ASm).

DISCUSSION

The results obtained in the present study revealed a markedly deteriorated dental health in our patients with liver cirrhosis, as reflected by a significantly higher number of carious, and missing teeth than in the healthy subjects. The number of carious teeth was greater among the cirrhotics, though these patients did not present more filleds than the controls. This suggests that the carious teeth were inadequately treated in the LC group, as also reflected by the greater number of missing teeth in the patients with liver disease.

Richards *et al.* (1994) reported the case of a 55-year-old woman who developed a rampant carious condition caused by a high level of oral dryness. This led to confirmation of Sjögren's syndrome and primary biliary cirrhosis. Thus, the oral dryness was the cause of an important dental deterioration. In the present study the greater number of caries among patients with LC cannot be accounted for in these terms, since only 5% of our patients suffered primary biliary cirrhosis. Nevertheless, these few patients presented a higher number of carious and missing teeth than those without primary biliary cirrhosis.

Recently Novacek *et al.* (1995) studied 97 patients with cirrhosis, 68 alcoholics without cirrhosis and 71 healthy controls. They found that oral hygiene, dental care and the periodontal status were worse and the number of carious teeth was higher in alcoholics with or without cirrhosis than in healthy controls and nonalcoholics with cirrhosis; also alcoholics had more missing teeth. They concluded that the dental and periodontal condition was worse in the latter and caused by bad oral hygiene and poor dental care.

In this sense Di Lauro and Tarantino (1983) consider that, due to the severity and overriding nature of LC, the patient tends to pay little attention to his or her oral condition. This may in part explain our findings. Moreover, poor oral hygiene is further favored in alcoholic patients, particularly among those with a low socioeconomical status, as was the case in many of our patients. As reflected by our results, patients with LC present greater dental deterioration, though this may be more due to poor dental hygiene than to the systemic disease itself. Indeed, we were unable to establish significant relationships between the number of carious, missing or filled teeth and liver parameters such as SGOT, SGPT and AP concentrations, platelet number, hepatitis markers or auto-antibodies (ANA, AMA, ASm).

REFERENCES

Anthony, P., Ishak, K.G., Nayak, N.C., Poulsen, H.E., Scheuer, P.J., So-bin, L.H. — The morphology of cirrhosis: definition, nomenclature and classification. *Bull. WHO, 55:* 521-540, 1977.

Di Lauro, F., Tarantino, G. — Importanza dell'integrità anatomo- funzionale dell'apparato stomatognatico nel cirrotico. *Minerva Stom., 32:* 501-503, 1983.

Erlinger, S., Benhamou, J.P. — Cirrhosis: Clinical Aspects. In: McIntyre, Benhaou, Bircher, Rizzeto, Rodes. *Oxford Textbook of clinical Hepatology. Oxford Medical Publications,* 380-390, 1991.

Goldin, P.C., Smith, M., Williams, R. — Multisystem involvement in chronic liver disease. *Am. J. Med., 55:* 772-782, 1973.

Novacek, G., Plachetzky, U., Pötzi, R., Lentner, S., Slavicek, R., Gangl, A., Ferenci, P. — Dental and periodontal disease in patients with cirrhosis-role of etiology of liver disease. *Journal of Hepatology, 22:* 576-582, 1995.

Richards, A., Rooney, J., Prime, S., Scully, C. — Primary biliary cirrhosis. Sole presentation with rampant dental caries. *Oral Surg. Oral Med. Oral pathol., 77:* 16-18, 1994.

Saunders, J.B., Walters, J.R., Davies, P., Paton, A. — A 20 years prospective study of cirrhosis. *Brit. Med. J., 282:* 263-266, 1981.

Sherlock, S. — Hepatic Cirrhosis. En: Sherlock, S., Dooley, J. Diseases of the Liver and Biliary System. *Blackwell Osney Mead. Oxford,* 357-369, 1993.

Address for correspondence:
J.V. Bagán
Hospital General Universitario
Servicio de Estomatología
Avd/Tres Cruces s/n
Valencia (Spain)