

PERIODONTAL ASPECTS IN CHILDREN AND ADOLESCENTS WITH DOWN SYNDROME

Vasilica Toma¹, A. Maxim¹, Adriana Balan¹, Diana Gheban¹,
Dana Cristiana Rotaru¹, Florina Filip², Liliana Foia³

Faculty of Dental Medicine

¹Pedodontics Dept., ²Family Medicine Dept, ³Chemistry and
Biochemistry of Oral Cavity Dept.

University of Medicine and Pharmacy „Gr. T. Popa” Iași

Abstract:

The Down syndrome is the most frequent genetic anomaly, presenting an incidence of 1/650 births (Winston, 2004). The individuals affected by DS frequently develop a form of aggressive periodontitis which affect both temporary and permanent teeth (Saxen, 1977, Svantum and Gjermo, 1978 a.o.) and can lead to the precocious expulsion of the teeth. Starting with these data, we tried to clinically evaluate the periodontal status in a group of 12 patients with DS, compared to the control group (which comprised 24 children without general diseases) and establish on the basis of clinical indicators (QHI, PBI, CAL) the forms of periodontal disease. Our results showed the preponderance of superficial chronic periodontitis (66.67%) in the children with DS, followed by gingivitis (33.3%), the aggressive forms (aggressive periodontitis) not being probably encountered due to the fact that the group was too young for the juvenile stage and however the children were hospitalized in the dental office of Sfânta Maria Policlinics, Iasi.

Key words: Down Syndrome, children, teenagers, clinical evaluation, periodontal disorders

INTRODUCTION

The Down Syndrome – 21 Trisomy is the first genetic disease described in 1866 by John Langdon Down. DS is the most frequent genetic anomaly, presenting an incidence of 1/650 births, being characterized by an autosomal transmission of the 21 chromosome trisomy. The individuals affected by DS frequently develop an aggressive form of disease which affects both temporary and permanent teeth (Cohen, 1961, Johnson și Young, 1973, Saxen, 1977, Svantum and Gjermo, 1978 a.o.) and can lead to the precocious expulsion of the teeth.

Starting with these data, we tried to clinically evaluate the periodontal status and to establish the forms of periodontal diseases in a group of children with DS.

MATERIAL AND METHOD

For realizing that purpose we constituted two groups of study:

I. Group I (control) – comprised 24 children with ages between 6-18 years old, without general diseases, being under

dental treatment in the assistance requested by the Infantile Dentistry Clinics Iasi, Dept. of Pedodontics.

II. Group II (active) – comprised 12 patients with age comprised between 5-16 years old being in the evidence of Neuropsychiatry – Genetic Diseases Office and the Dental Office of Sfânta Maria Policlinics.

The evaluation of the periodontal status was performed in the groups of children and adolescents through the calculation of the following indexes of diagnostic for periodontal disease:

The Quiqley and Hein coloured bacterial plaque index

Papillary bleeding Index (Saxen and Muhlemann)

The level of attachment loss (CAL) evaluated through periodontal probing and radiological exam.

All the evaluations of clinical indexes were performed at the level of Ramfjörd teeth, mesial sites. The evaluation of plaque deposits were made with the help

of coloured plaque indexes Quiqley – Hein, as it follows:

$$QH = \frac{\text{The sum of values for every tooth of B and L surfaces}}{\text{The total number of examined surfaces}}$$

This index is evaluated on the basis of scores:

- 0 – absence of plaque
- 1 – separated isles of plaque at the level of cervical area
- 2 – fine band of plaque of up to 1 mm height at cervical area
- 3 – band of plaque comprised between 1 mm and 1/3 from the crown of the tooth
- 4 – band of plaque comprised between 1/3 and 2/3 from the tooth crown
- 5 – bacterial plaque which covers more than 2/3 of the tooth crown

The intensity of inflammation was appreciated through the intensity of papillary bleeding (PBI index). Indicator of gingival bleeding severity, it notes from 0 – 4 different progressive phases of bleeding

- 0 = without bleeding
- 1 = punctiform bleeding
- 2 = line/points, fine line of bleeding or several points at gingival margin
- 4 = drop that flows – massive bleeding

The clinical attachment loss was evaluated through periodontal probing appreciating the depth of the pockets and also the degree of recession, at the level of every test tooth in at least 6 sites (B, L, MB, ML, DL) the reference element for evaluating the attachment loss was amelocementar junction (AML) expressing the distance between the bottom of the sulcus/pocket in millimetres.

The Rx exam, useful in appreciating the importance of attachment loss was

performed on retro-dento-alveolar cliches in isometric and orthoradial incidence or through ortopantomography (OPT). Comparisons were made between the two groups for every calculated index, the values for every patient were calculated together for creating the average of the group, using a common ground estimated as standard of correction for the error of unequal variances. Under this circumstance the patients were in the centre of the study and not the number of sites.

The statistical differences between the averages of the values of interest according to the groups were tested using the One-Way ANOVA test completed of KRUSKAL-Wallis test (or Mann-Whitney/Wilcoxon test) for the average age and clinical values. The statistically significant differences were considered for the values of the level of significance (p) lower than 0.05, according to a level of confidence of 95%.

RESULTS

The bacterial plaque index

For evaluating the patients with Down syndrome from the point of view of the clinical indexes, their values were compared to the values recorded in the control group. In the analysis of every clinical index the statistical indicators which describe correctly the characteristics of the group were calculated, comparing them at the same time with the values of the respective indicators of the control group.

Table I. The statistical indicators of the Quikley-Hein index of bacterial plaque (QHI) for the group of patients with Down Syndrome

Group \ QHI	Average	-95%	+95%	Std. Dev	Variance	Std. Error	Min	Max	Q25	Median Value	Q75
Control	2.995	2.859	3.130	0.576	0.332	0.068	2.166	4.133	2.500	3.000	3.500
Down Syndrome	3.622	3.408	3.836	0.632	0.400	0.105	3.000	5.000	3.083	3.495	3.830
Total	3.204	3.077	3.330	0.663	0.439	0.064	2.166	5.000	2.660	3.000	3.660

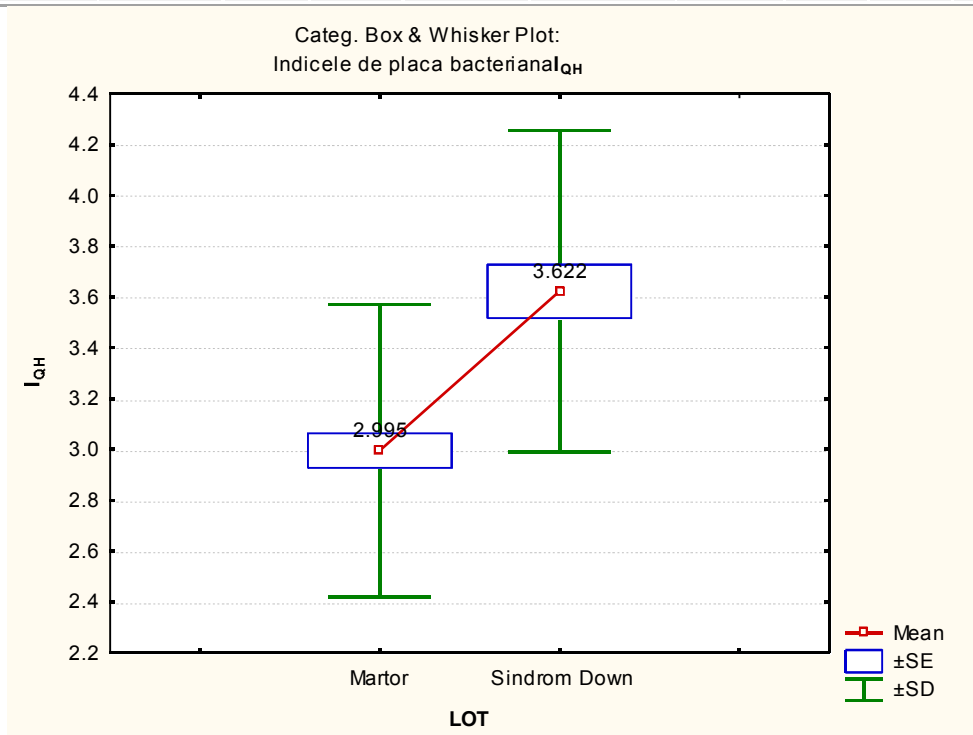


Fig.1. Average values of the bacterial plaque index in the control group (QHI) and the group of patients with Down Syndrome

Clinical Attachment loss (CAL)

Table II. Statistical indicators of the attachment loss for the group of patients with Down Syndrome

Group \ CAL	Average	-95%	+95%	Std Dev.	Variance	Sst Er.	Min	Max	Q25	Median Value	Q75
Control	0.388	-0.067	0.842	1.077	1.160	0.220	0.000	4.000	0.000	0.000	0.000
Down Syndrome	1.417	0.562	2.272	1.346	1.811	0.388	0.000	3.500	0.000	1.750	2.500
Total	0.731	0.306	1.155	1.254	1.574	0.209	0.000	4.000	0.000	0.000	1.750

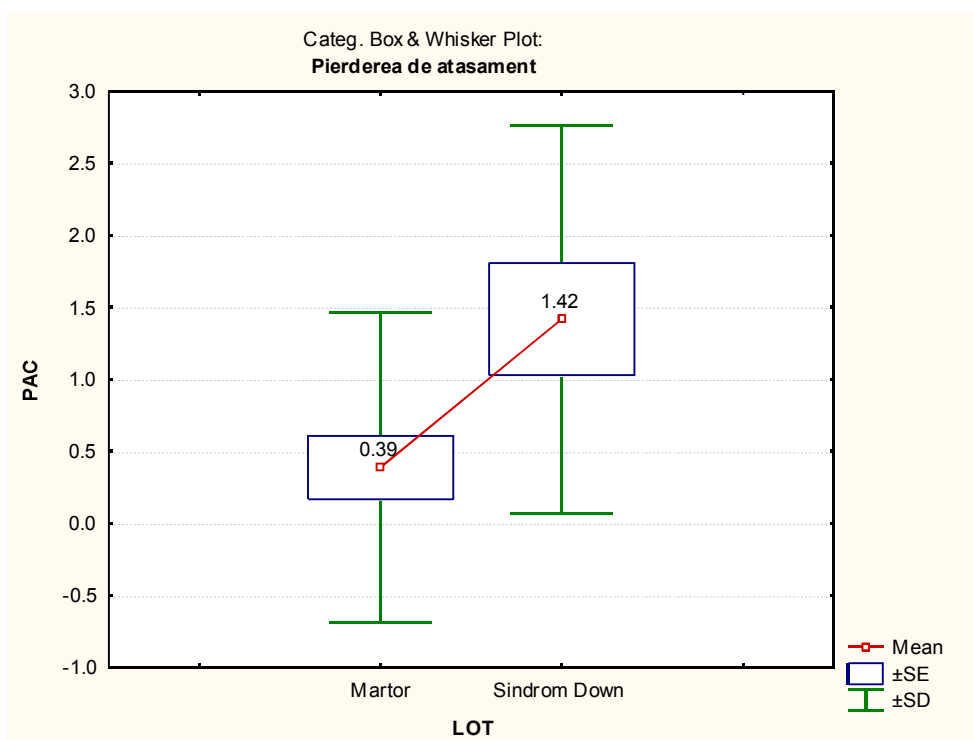


Fig.2. The average values of attachment loss (CAL) in the control group and the group of patients with Down Syndrome

From the above image an increase of 3.6 times higher of the average value for the CAL index in the Down group

compared to the control group can be noticed.

Papillary bleeding index

Table. III. The statistical indexes pf the papillary bleding index for the group of patients with Down Syndrome

Group \ ISP	Average	-95%	+95%	Std. Dev.	Variance	Std.Er.	Min	Max	Q25	Median Value	Q75
Control	1.560	1.271	1.850	0.686	0.470	0.140	0.500	2.660	0.833	1.500	2.165
Down Syndrome	2.678	2.291	3.064	0.609	0.370	0.176	2.000	4.000	2.248	2.580	2.830
Total	1.933	1.647	2.218	0.843	0.711	0.141	0.500	4.000			

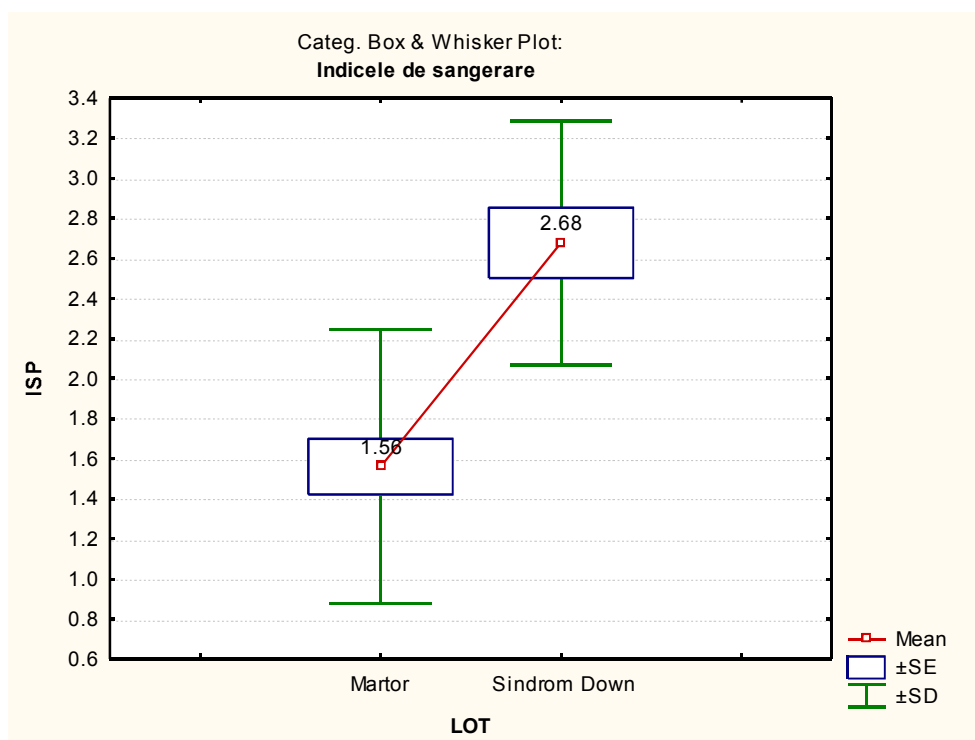


Fig.3. The average values of PBI in the control group and the group of patients with Down syndrome

Corroborating the clinical exam with the values of the calculated clinical indexes (QHI, PBI, CAL) and with the radiological exam, we established the periodontal diagnosis for every patient.

The analysis of the parameters connected with the repartition depending on diagnostic and systemic condition showed the following:

Control group

The highest weight belongs to the microbial inflammatory diseases induced by bacterial plaque, gingivitis respectively (87.50%), followed by the superficial alteration of the sustaining periodontium (chronic superficial periodontitis) (8.33%), and aggressive periodontitis (4,17%) (Table 4).

Table IV. The incidence of the cases depending on the diagnosis in the control group

Diagnostic (control group)	No. of cases	%
Generalized bacterial gingivitis	21	87.50%
Chronic superficial periodontitis	2	8.33%
Aggressive (juvenile) periodontitis	1	4.17%
Total	24	

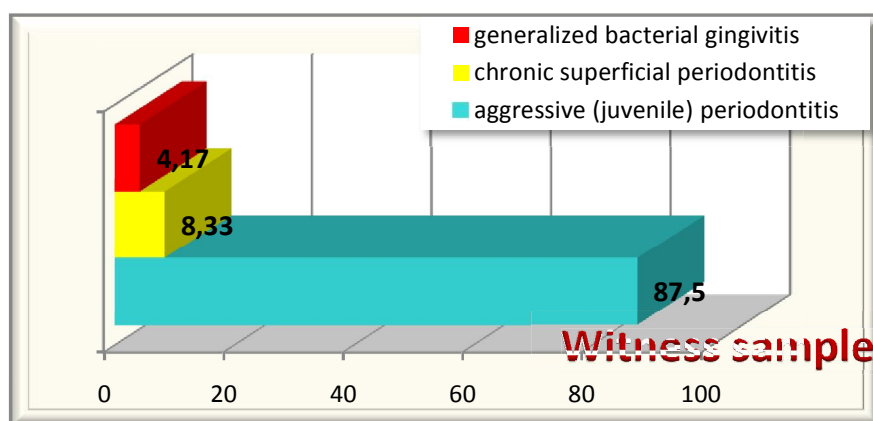


Fig.4. The incidence of the diseases in the control group

The group of patients with Down Syndrome

Although the data from literature show that in the patients with Down Syndrome aggressive forms of periodontitis are encountered (Cohen et al. 1961, Johnson and Young 1963, Saxen et al. 1967, Snajder et al., 1968, Svatum and Gjermo,1978) in a proportion of 90% (Dow 1951) and between

90-100% (Kisling and Krebs, 1963, Cohen and Goldmann, 1960, Johnson and Young 1963), in our study for the patients with Down Syndrome, the highest weight is represented by chronic superficial periodontitis(66.67%), followed by gingivitis (33.33%), the aggressive forms of disease being absent.

Table V. The repartition of cased depending on diagnosis

Diagnosis	No of. cases	%
Generalized bacterial gingivitis	4	33.33%
Chronic marginal periodontitis	8	66.67%
Aggressive periodontitis	0	0.00%
Total	12	

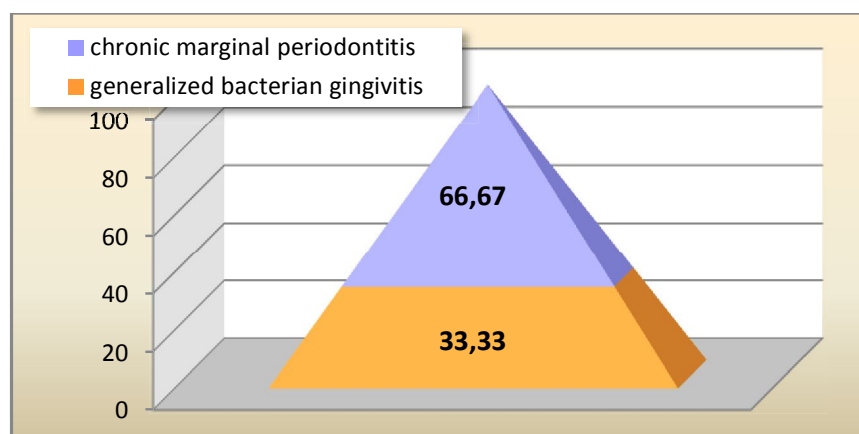


Fig. 5. The repartition of the cases depending on diagnosis

DISCUSSIONS

The Down Syndrome – 21 Trisomy - the first genetic disease described in 1866 by John Langdon Down. DS is the most frequent genetic anomaly, (1/650 new bourns). The affected individuals frequently develop a form of aggressive periodontal disease which affects both temporary and permanent teeth (Cohen 1961, Johnson and Young 1973, Saxen 1977, Snajder 1968, Svatum and Gjermo 1978) and can lead to the precocious expulsion of the teeth, loss of alveolar bone measured on orthopatomography, being found in 69% from the patients with 21 trisomy (Saxen et al. 1977).

The periodontal destructions are characterized by the formation of deep periodontal pockets, associated to increased quantities of bacterial plaque and intense gingival inflammation (maximum values QHI=5, PBI=4, CAL=4 mm), values which cannot be explained only on local factors (bacterial plaque, calculus) (Ulseth, 1991).

Dow in 1951 reported that more than 90% from the children with Down syndrome, having ages between 8-12 years old, develop some forms of periodontal disease. Subsequent studies also reported the prevalence of periodontal disease in percentages of 90-100% in the patients with 21 trisomy (Kisling and Krebs, 1963; Johnson and Young, 1963; Cohen and Goldman 1960) and 76.5% of the cases

(Rusu et al. 1972); Orner (1976) reported that the periodontal index in the children with Down Syndrome was 4.5 times higher than in the healthy patients.

Our results enrol as patient data in those from literature, the prevalence of periodontal disease being 33.33% generalized bacterial gingivitis and 66.67% chronic marginal periodontitis.

Numerpus other studies have shown an increased prevalence of periodontal disease in patients with Down syndrome compared to other deficiencies (Johnson and Young 1963; Snajder 1968, Cutress 1971, Brown 1973; Reuland – Bosma and Van Diyk 1986, Cichon 1998 and others) and Shallow (1964) found an increased prevalence of periodontal disease in hospitalized children with Down Syndrome compared to those not hospitalized.

CONCLUSIONS

Although the data from literature shows that in the patients with Down Syndrome aggressive forms of periodontitis are encountered Cohen 1961, Johnson and Young 1973, Saxen 1977, Snajder 1968, Svatum and Gjermo 1978) in a proportion of 90% (Dow 1951) and between 90-100% (Kisling and Krebs, 1963, Cohen and Goldmann, 1960, Johnson and Young 1963), in our study of patients with Down Syndrome, the weight is represented by chronic marginal

periodontitis (66.67%), followed by gingivitis (33.33%), the aggressive forms of periodontal disease being absent; probably due to the fact that our group was

too small for the juvenile stage and however our patients were already hospitalized in the dental office of Sfânta Maria Policlinics, Iasi.

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