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ORIGINAL ARTICLE

Prevalence of intrabony defects in a Swedish adult population. A radiographic epidemiological study

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ABSTRACT

Aim: The purpose of this radiographic study was to evaluate the prevalence of intrabony defects and to study the correlation between these defects and clinical variables in a Swedish adult population. Another aim was to study the combined relationship of intrabony defects and furcation involvement with clinical variables.

Materials and methods: The present study was performed using bitewing and apical radiographs from 329 subjects. The clinical and radiographic data were collected from the study that was carried out in Jönköping 2003. Intrabony defects were measured from the bottom of the pocket to the highest point of alveolar bone crest. Furcations were considered healthy if the furcation was filled with bone up to the fornix. SPSS was used to analyze the results.

Results: 2014 molars and 5898 non-molars were included in the study. The prevalence of intrabony defects was 2.2% in the whole population. Multivariate analysis showed that periodontal pockets ($p < 0.0001$), plaque ($p < 0.001$), age ($p < 0.02$) and gender ($p < 0.03$) had a significant relationship with the occurrence of intrabony defects. On the other hand, gingivitis, smoking habits and education level were not associated with intrabony defects. Multivariate analysis showed that the only variable associated with presence of intrabony defects and furcation involvement was presence of periodontal pockets ($p < 0.0001$). Whilst, gingivitis, education level, plaque, gender, age and smoking habits were not associated with the outcome.

Conclusions: Mandibular second molars were most likely to display intrabony defects, whilst mandibular incisors were the least likely to do so.

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Prevalence; intrabony defects; tooth furcation; periodontitis

Introduction

The pattern of periodontal bone loss may be either horizontal or vertical (intrabony). Intrabony defects are defects that occur when the base of the defect is located apically to its bony margin. Many studies have shown that the radius of the effect of microbial plaque (dental biofilm) is about 1.5–2 mm.[1,2] Several factors can be involved in the formation of intrabony defects such as the thickness of the buccal-lingual width of the alveolar process, a lesser degree of convexity of the cementoenamel junction of the molars, and the width of the interproximal bone.[3,4]

A study on 48 skulls showed that molars were the teeth most frequently affected by interproximal intrabony defects. Moreover the mandibular molars had more intrabony defects than the maxillary molars.[3] Another study on 100 mandibles showed that second molars were overrepresented in comparison to first molars.[5] A Swedish study that included 194 patients showed that the prevalence of intrabony defects with 2.5 mm or deeper was 3%.[6] Intrabony defects were also shown to increase with age.[7] Gender, smoking, level of education, plaque index, gingival index and periodontal pockets were associated with an increased risk of progression and severity of periodontitis.[8–17] A study on 41 skulls showed that the validity of radiographs to diagnose intrabony defects was 98.1%.[18]

The clinical importance of this study is that intrabony defects are associated with further loss of marginal bone.[6] Another clinical aspect is that periodontal pockets associated with intrabony defects do not respond to non-surgical periodontal treatment as well as pockets with horizontal bone loss do.[19] Furcation-involved molars are considered to be at a higher risk for further progression of periodontitis and tooth loss.[20,21] The aim of this cohort study was to evaluate the prevalence of intrabony defects in a Swedish adult population (the subjects were 40, 50, 60 and 70 years old). A second aim was to study the correlation between intrabony defects and different variables (gender, age, level of education, smoking, plaque, gingivitis and presence of periodontal pockets). A third aim was to study the combined relationship of intrabony defects and furcation involvement and the variables mentioned above. This will be carried out using the clinical registration and radiographs from the 2003 Jönköping study.

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Materials and methods

This study was performed using the radiographs and clinical data from the 2003 Jönköping study and included individuals who lived in Jönköping City, Sweden. All individuals ($n = 329$) aged 40, 50, 60 and 70 years in the original study were included in this study.[22]

The subjects of the original study were selected randomly from the register of the County Government Board. The total number of participants was 987 individuals in the age groups 3, 5, 10, 15, 15, 20, 30, 40, 50, 60, 70 and 80 years. The Jönköping epidemiological studies on oral health and disease started in 1973 and have been repeated every 10 years since then. The radiographs assessed in the current study were panorals, periapicals and bitewings. These were examined by the author U.N. The radiographs were placed on an illuminated screen (Örebro, Malmö, Sweden) with diffuse white light and then analyzed in a dark room with the aid of observation binoculars.[23]

In the present study, the inclusion and exclusion criteria, which were based on tooth level, were assessed on radiographs. The inclusion criteria were:

1. All teeth except third molars.
2. Approximal intrabony defects ≥3mm around all teeth.
3. Furcation involvement grade II and III in molars.[24]

Exclusion criteria were:

1. Untreatable teeth and root remnants.
2. Dental implants.

A graduated transparent ruler was used to measure intrabony defect depth on radiographs. Panorals were used to identify the missing teeth. Bitewing radiographs were used to identify the furcation involvement and to measure the depth of the intrabony defects from the distal surface of canines to the distal surface of the second molar. Periapical radiographs were used to measure the depth of the intrabony pocket between the mesial surfaces of the canines. These X-ray films were taken using parallel technique.

Intrabony defects were measured from the bottom of the pocket to the highest point of alveolar bone crest on radiographs. The base of the pocket was considered as the most coronal point where the periodontal space still retained its normal width.[25] The depth of the pocket was measured by drawing a line parallel to the long axis of the tooth and was rounded off to the nearest millimeter. Regarding the diagnosis of furcation involvement, the reader is referred to the previous study by the same authors.[26]

Clinical variables, which were registered in 2003, were collected and analyzed regarding: (1) intrabony defects and (2) intrabony defects and furcation involvement. These variables were:

Gingival index: the occurrence of gingival inflammation corresponding to Gingival Indices 2 and 3 was recorded for all tooth surfaces. Gingival inflammation was recorded if the gingiva bled on gentle probing.[27] In the regression analysis, subjects with score 0 or 1 were classified as 0 and subjects with score 2 or 3 were classified as 1.

Plaque index: the presence of visible plaque was recorded for all tooth surfaces after drying with air according to the criteria for Plaque Indices 2 and 3.[28] In the regression analysis, subjects with score 0 or 1 were classified as 0 and subjects with score 2 or 3 were classified as 1.

Smoking status: either never/former smoker or current smoker. Never/former smokers were classified as no and current smokers were classified as yes. The data regarding smoking habits were collected through survey questionnaire which the subjects received at the time of the clinical examination at 2003.

Presence of clinical periodontal pockets: depth ≥4 mm (no or yes). The depth of the pockets was registered from marginal gingival to the bottom of the periodontal pocket.

Level of education: primary school/high school or college/university.

Gender: male or female.

Age: 40, 50, 60 and 70 years old.

Ethical considerations

The original study protocol was approved in 2003 by the Ethical Committee at the University of Linköping, Linköping, Sweden.[22]

Statistical analysis

The analysis was carried out at both univariate and multivariate level with SPSS. The questionnaires and clinical records were developed as a web survey in the software program EsMaker NX2 (Entergate AB, Halmstad, Sweden). Frequencies, mean values and distributions were calculated. Data processing was performed using SPSS version 22 (IBM Corporation, Armonk, NY).

Results

A total number of 329 subjects, 165 male and 164 female, were included in this study. In total, 2014 molars (1020 and 994 were found in the maxilla and mandible, respectively) and 5898 non-molar teeth (2,863 and 3,035 were found in the maxilla and mandible, respectively) were evaluated. Thirteen non-molars in four patients could not be examined because of blurred radiographs. These teeth were excluded from the study.

In the whole population, the prevalence of intrabony defects was 2.2%. Figure 1 shows that intrabony defects occurred in 4.1% and 1.5% molars and non-molars, respectively. The prevalence of occurrences of intrabony defects was significantly higher in molars than non-molars, ($p<0.0001$). Table 1 shows the prevalence of intrabony defects at each group. The prevalence of intrabony defects was higher in mandibular molars than in the maxillary molars, but the difference was not significant (Table 2). Also, the second molars had a higher frequency of defects, about the double. Amongst the molars, the mandibular second molar with 5.9% was most likely to display intrabony defects, whilst the

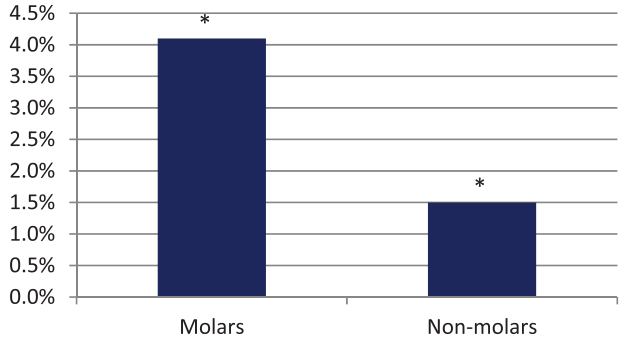

Figure 1. The prevalence of intrabony defects at molars and non-molars. *$p < 0.0001$

Table 1. The prevalence of intrabony defects in each age group.

40 years old	1.6%
50 years old	2.3%
60 years old	2.5%
70 years old	2.3%

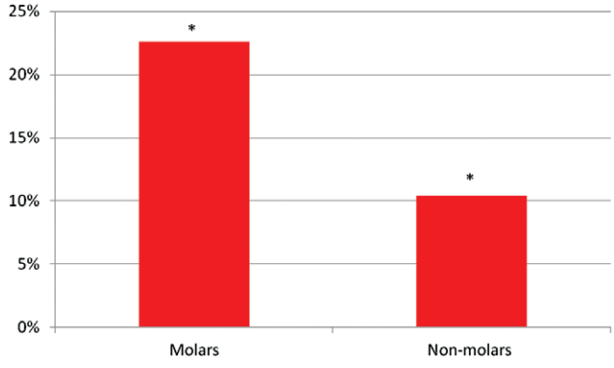

Figure 2. The prevalence of missing molars and non-molars. *$p < 0.0001$

Table 2. The prevalence of intrabony defects at maxillary and mandibular molars.

Maxillary molars		Mandibular molars	
3.7%		4.6%	
First molar	Second molar	First molar	Second molar
2.7%	4.8%	3.3%	5.9%

maxillary first molar with 2.7% was the least likely to display such defects.

Figure 2 shows that the prevalence of missing teeth was significantly higher in molars than non-molars, ($p < 0.0001$). There was no significant difference regarding the frequency of missing teeth between the molars, see Table 3.

Figure 3 shows the prevalence of intrabony defects at non-molars. The frequency of occurrence of intrabony defects around non-molars was significantly higher in the maxilla ($p \leq 0.0001$). On the other hand, there was no difference regarding intrabony defects between maxillary and mandibular premolars, canines and incisors (Table 4). Figure 4 shows

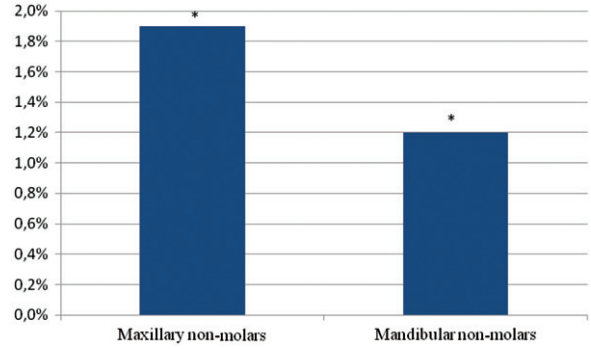

Figure 3. The prevalence of intrabony defects at maxillary and mandibular non-molars. *$p < 0.0001$

Table 3. The prevalence of missing maxillary and mandibular molars.

Maxillary molars		Mandibular molars	
21.7%		23.6%	
First molar	Second molar	First molar	Second molar
19.8%	23.6%	24.8%	22.5%

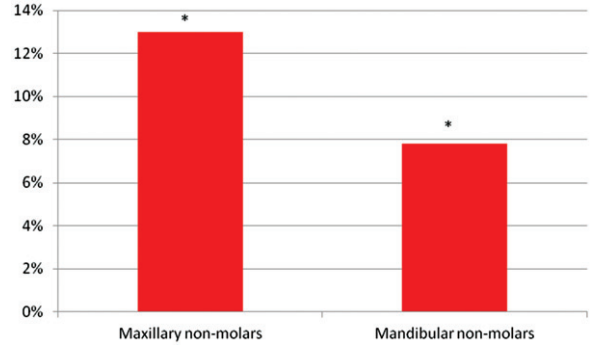

Figure 4. The prevalence of missing maxillary and mandibular non-molars. *$p < 0.0001$

that the prevalence of missing non-molars was significantly higher in the maxilla ($p \leq 0.0001$), while there was no significant difference regarding missing teeth between maxillary and mandibular premolars, canines and incisors (Table 5).

Regarding the association between clinical variables and intrabony defects at univariate level, the results showed that gingivitis, plaque, periodontal pockets and age were significantly correlated to the outcome ($p < 0.0001$). Also, smoking was significantly associated with the presence of intrabony defects ($p < 0.001$). Moreover, females had a significantly higher prevalence of intrabony defects, ($p < 0.02$). On the other hand, level of education was not correlated to the outcome, see Table 6. Multivariate analysis showed that periodontal pockets ($p < 0.0001$), plaque ($p < 0.001$), age ($p < 0.02$) and gender ($p < 0.03$) were significantly associated with the presence of intrabony defects, see Table 7. Smoking had a non-significant tendency to affect the presence of intrabony defects ($p < 0.07$).

Table 8 shows the association between the clinical variables and the presence of both intrabony defects and furcation involvement. Univariate analysis showed that plaque, smoking and periodontal pockets were significantly associated with the outcome ($p < 0.0001$). Also, age ($p < 0.004$) as well as gingivitis ($p < 0.002$) was significantly correlated to the outcome. Education level and gender were not associated with these outcomes.

Multivariate analysis showed that the only clinical variable that was associated with both intrabony defects and furcation involvement was periodontal pockets ($p < 0.001$), see Table 9. Age and plaque had a non-significant tendency to affect the outcome ($p < 0.07$). Smoking, gender, gingivitis and level of education were not associated with furcation involvement and intrabony defects.

Table 4. The prevalence of intrabony defects at non-molars.

Tooth group	Intrabony defects
Premolars (total)	1.9%
Maxillary premolars	2.5%
Mandibular premolars	1.3%
Canines (total)	1.7%
Maxillary canines	1.8%
Mandibular canines	1.6%
Incisors (total)	1.1%
Maxillary incisors	1.4%
Mandibular incisors	0.9%

Table 5. The prevalence of missing maxillary and mandibular non-molars.

Tooth group	Missing
Premolars (total)	17.1%
Maxillary premolars	20.1%
Mandibular premolars	14.0%
Canines (total)	4.6%
Maxillary canines	6.8%
Mandibular canines	2.4%
Incisors (total)	6.6%
Maxillary incisors	8.9%
Mandibular incisors	4.2%

Discussion

To our knowledge, this study is the first epidemiological study that evaluates intrabony defects in a randomized population with different level of periodontal disease experience. It was a part of a radiological evaluation of furcation involvement in molars and intrabony defects in the 2003 Jönköping study. The first part of the study examined the prevalence of furcation involvement.[26] This study showed that the prevalence of intrabony defects in the whole population (40, 50, 60 and 70 years of age) was 2.2%. This percentage is comparable to another study that was performed in Gothenburg, Sweden.[6]

A study on 337 dry skulls was performed to identify the prevalence of intrabony defects.[29] The distribution of frequency of the defects was higher around mandibular molars than around maxillary molars. A higher frequency of intrabony defects was also seen around the second molars than

Table 6. Clinical variables and intrabony defects (univariate logistic regression analyses).

Variable	*p* value	Odds ratio	95% confidence interval
Periodontal pockets (No pockets= 0, ≥1 pockets =1)	0.0001	9.58	5.54–16.55
Age	0.0001	1.04	1.02–1.07
40 years	0.002	1.00	
50 years	0.04	1.16	0.53–2.52
60 years	0.03	2.79	1.34–5.80
70 years	0.02	2.88	1.39–5.97
Plaque index (Score 0 or 1 = 0, score 2 or 3 = 1)	0.0001	1.03	1.02–1.04
Gingival index (Score 0 or 1 = 0, score 2 or 3 = 1)	0.0001	1.02	1.01–1.04
Smoking (Former or non-smoker= 0, current= 1)	0.001	2.85	1.57–5.16
Gender (Male= 0, female= 1)	0.02	1.80	1.11–2.93
Education level (Primary or high school= 0, college/university= 1)	0.18	1.43	0.85–2.41

Table 7. Clinical variables and intrabony defects (multivariate logistic regression analyses).

Variable	*p* value	Odds ratio	95% confidence interval
Periodontal pockets (No pocket= 0, ≥1 pockets =1)	0.0001	7.73	4.27–14.00
Plaque index (Score 0 or 1 = 0, score 2 or 3 = 1)	0.001	1.02	1.01–1.03
Age	0.02	1.03	1.01–1.06
Gender (Male= 0, female= 1)	0.03	1.93	1.06–3.52

around first molars. The molar most frequently displaying intrabony defects was mandibular second molars, whilst maxillary first molars were the least likely to display intrabony defects. The frequency of distribution of intrabony defects was higher at maxillary non-molars than at the mandibular. The non-molar most frequently displaying intrabony defects was maxillary premolars, whilst mandibular incisors and premolars were the least likely to display intrabony defects. A study on 100 mandibles showed that second molars were overrepresented compared to first molars regarding intrabony defects.[5] Another study on 148 skulls showed that the second molars had a higher prevalence of intrabony defects than first molars did.[3] These studies showed that the prevalence of intrabony defects was much higher around maxillary non-molars than around mandibular non-molars.[3,29] Moreover, a study on 286 subjects with diagnosis moderate or advanced periodontitis showed that mandibular molars had the highest prevalence of intrabony defects, whilst maxillary and mandibular anterior teeth had the lowest.[30] These statements are in agreement with our results. The reasons behind the overrepresentation of molars are the difficulty of keeping the interproximal areas clean, the flat crest between molars makes self-cleaning more difficult than it is in the more convex anterior teeth and thicker buccal-lingual width of the alveolar process in the posterior teeth.[3] A clinical study was carried out to examine the relationship between the interproximal distance and the prevalence of intrabony defects.[4] The study consisted of 81 patients who were referred to the Department of Periodontology at New York University for treatment of periodontitis. The conclusion was that in locations where interdental distances were between 2.1 and 4.5 mm the risk of intrabony defects increased. It was suggested that the effect of microbial plaque was 1.5–2 mm. This will cause bony plates thinner than this dimension to be completely destroyed by microbial plaque.[1,2]

Many studies have shown that intrabony defects increased with age.[7,17] This statement was confirmed in a study which was performed on skulls.[3] These results are in agreement with the outcome of our study.

In our study, females had a significantly higher prevalence of intrabony defects. Radiographs belonging to 209 individuals, referred for dental treatment at a dental college in Aarhus, Denmark, were studied.[17] That study showed a slightly higher prevalence of intrabony defects in males but the difference was not significant. The reason for these differing results may be different definitions of intrabony defects and the fact that relatively few individuals above the age of 44 were included in the Danish study. The study also showed that loss of attachment was correlated to increasing prevalence of intrabony defects. Our study showed that periodontal pockets increased the risk for intrabony defects.

In our study, plaque and gingival index showed a significant relationship with intrabony defects. The Danish study also showed that a higher score of plaque and gingival index was correlated to a higher prevalence of intrabony defects.[17] In the Danish study, analysis was carried out on tooth level, whereas in our study the analysis was performed on patient level.

Univariate analysis showed that smoking was significantly correlated to the presence of intrabony defects. The Danish study showed that smoking was not correlated to increased risk of intrabony defects. Many studies have shown that smoking has a negative effect on the periodontium, i.e. increased bone and attachment loss.[14,31–33] The effect of smoking is dose-related, i.e. the number of cigarettes and years. As previously mentioned, the study included few individuals older than 44 years of age and the number of smokers was not mentioned. These factors may affect the statistical analysis and the result.

Level of education did not have any effect on the occurrence of intrabony defects. On the other hand, many studies have shown that level of education has a strong association with the occurrence of periodontitis.[14,34]

Multivariate analysis showed that periodontal pockets, age, plaque and gender were significantly correlated to the occurrence of intrabony defects.

Univariate analysis showed that periodontal pockets, age, smoking, plaque and gingivitis were associated with both furcation involvement and intrabony defects. On the other hand, multivariate analysis showed that the only variable that was associated with furcation involvement and intrabony defects was periodontal pockets.

Table 9. Relationship between clinical variables and both intrabony defects and furcation involvement (multivariate logistic regression analyses).

Variable	*p* value	Odds ratio	95% confidence interval
Periodontal pockets (No pocket= 0, ≥1 pockets =1)	0.0001	15.13	6.98–32.81

Table 8. Relationship between clinical variables and both intrabony defects and furcation involvement (univariate logistic regression analyses).

Variable	*p* value	Odds ratio	95% confidence interval
Periodontal pockets (No pocket= 0, ≥1 pockets =1)	0.0001	15.19	7.25–31.82
Age	0.004	1.04	1.01–1.07
40 years	0.03	1.00	
50 years	0.24	1.87	0.67–5.24
60 years	0.01	3.57	1.34–9.50
70 years	0.01	3.57	1.34–9.50
Plaque index (Score 0 or 1 = 0, score 2 or 3 = 1)	0.0001	1.02	1.01–1.03
Gingival index (Score 0 or 1 = 0, score 2 or 3 = 1)	0.002	1.02	1.01–1.03
Smoking (Former or non-smoker= 0, current= 1)	0.0001	3.22	1.68–6.19
Gender (Male= 0, female= 1)	0.25	1.40	0.79–2.51
Education level (Primary or high school= 0, college/university= 1)	0.43	1.29	0.69–2.43

The main reasons for performing this study were to assess the frequency of intrabony defects as well as the association of these defects with clinical and demographic variables in a population with different levels of periodontal disease experience, i.e. a population representative of society in general. Another aim was to study the combined relationship of intrabony defects and furcation involvement and with the same clinical variables. This will highlight the importance of identifying different risk indicators which in turn will optimize dental service by focusing the required resources on individuals with high risk to develop such defects. Another clinical reason is that periodontal pockets associated with intrabony defects are more difficult to treat with non-surgical periodontal treatment as compared with pockets with horizontal bone loss.[19] This may in turn increase the need of surgical correction. Histological studies show that healing of intrabony defects after periodontal surgery lead to the formation of a long-contact epithelium.[35,36] This is considered less resistant to recolonization by subgingival plaque.[37,38] Additionally, intrabony defects with depth of $\geq$3mm can be candidates for regenerative treatment.[39,40] As furcation involvement is considered to be a risk factor for loss of molars, it is important to study the association between clinical variables and furcation involvement on subject level.[20,21]

In conclusion, in the sample studied, molars had a significantly higher prevalence of intrabony defects than non-molars and that periodontal pockets were the only risk indicator for both intrabony defects and furcation involvement.

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Disclosure statement

The authors declare that they have no conflict of interests.

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