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Original Article

Prevalence and Distribution of Different Types of Bone Defects in Chronic Periodontitis In Bagalkot Subjects - A Clinical Study

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Abstract

Background: Chronic destructive periodontitis involves resorption of the bone supporting the teeth. Various treatment aspects of this disease depend on the amount of alveolar bone remaining around the teeth. Hence, it is important to know the exact topography of the bone loss which is best possible by direct viewing during surgery. The aim of the study was to determine the prevalence and distribution of the various types of bone defects; it's relation with adverse habits and the segment wise distribution in the mandible and maxilla in Bagalkot population.

Materials and Methods: This study was carried out on 200 chronic periodontitis patients of age group 30-70 years using direct observation during periodontal surgery. The association of various osseous defects with age, sex, socioeconomic status (SES), adverse habits, geographical location and trauma from occlusion, arch, and segment-wise were assessed.

Results: There was increased horizontal bone loss with the advancing age and males were more affected. More number of defects was seen in the posterior segment. No statistically significant relation was found between the SES, geographic location, and between the two arches. Among bony defects, craters were the most common angular bony defects and were found to be related with the trauma from occlusion and adverse habits.

Conclusion: The prevalence and distribution of bone defects are multifactorial and was not related to one cause. It is related to the age, sex, SES, adverse habits, and the anatomy of the bone.

Key words: Bone defects, Chronic periodontitis, Periodontal surgery

INTRODUCTION

Alveolar bone loss is one of the most important features of periodontal disease. The variation in patterns of bone loss between individuals and between different sites in the same mouth, the rate of the bone loss, and the diversity of form of loss have stimulated a great deal of interest and speculation[1] alveolar bone is as vital a tissue as the gingiva, pulp, or periodontal ligament and should be viewed biologically as a living tissue.

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With the tremendous progress made in establishing the relative role of the external or the environmental causes of disease, the individual or genetic differences are assuming increasing importance. Periodontal bone loss from periodontitis may be either horizontal, resulting in a regular alveolar crest at a level more apical to the normal, or vertical, leading to the formation of defects contained within the bone. In either case, an increase in the distance between the cementoenamel junction and the alveolar bone crest is evident. An infrabony defect is any defect of the alveolar bone proper, the base of which lies apically to its bony margins. Like other bony deformities, these may be detected by radiographic means, by observing periodontal bone during surgical procedures or by visual examination of the dried skull.[2] Chronic periodontitis is most common inflammatory periodontal disease which leads to changes in normal architecture of the alveolar processes. These changes vary

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in degree, form, and distribution within the same mouth as well as between individuals.[1] The possible factors in the pathogenesis of the bone defects are tooth anatomy and position, the relationship of adjacent marginal ridges and cementoenamel junctions, and open contact points with resultant food impaction as well as traumatic lesions affecting the attachment apparatus.[2] Many authors have classified bony defects as interdental crater, hemiseptum, inconsistent margins, furcation invasions, infrabony defects with 1-, 2-, or 3-wall defect and combination of these defects. These lesions may also be complicated by the anatomic aberrations of the alveolar process, i.e., thick margins, exostoses, tori, fenestrations, and dehiscence.[3] The existence of bone defects present the specific problems in the clinical practice and direct observation during periodontal surgery is the only means of accurately recording the bone morphology, taking into account the limitations of radiographic examination. To date, very few studies have been done to know the prevalence and distribution of different types of bone defects encountered during periodontal surgery. This study could provide not only the prevalence and distribution but also a better approach in deciding the treatment procedures.

MATERIALS AND METHODS

The study was carried out on 200 chronic periodontitis patients in the age group of 30-70 years referred to the Department of Periodontics, P. M. N. M. Dental College and Hospital, Karnataka, India. Patients were diagnosed of chronic periodontitis after clinical and radiological examination. Patients who had received surgical periodontal treatment previously and patients with self-reported systemic diseases that influence pathogenesis of periodontal diseases was excluded from the study.

Procedure

First, pocket depth was measured and recorded using William's graduated periodontal probe along the long axis of the tooth at four sites around the tooth-mesial, facial, distal, and lingual/palatal.[4] Fremitus was measured using index finger along the buccal surfaces of the maxillary anterior teeth, and the patient was asked to tap the teeth together in the maximum intercuspal position and felt for vibrations.[5] After the initial diagnosis, nonsurgical treatment consisting of plaque control instruction, reinforcement, and scaling and root planning was carried out on all patients. Reevaluation of the therapy was performed 3-4 weeks later. Residual pocket depths >4 mm with bleeding on probing, requiring periodontal surgery were selected in individuals involving one or more segments of all patients. Periodontal surgery was performed in sextants, and the osseous defects were explored in both maxilla and mandible under good illumination using periodontal probes and explorers. The distal surface of the cuspid was designated as being in the posterior segment. When combination of more than one type of defect existed, the dominant type was recorded. Craters were recorded to the tooth distal to the defect. The morphology of the bone defects was classified according to Vrotsos *et al.* in 1999.

Interdental Craters

A concavity (saucer-shaped) in the interdental bone confined within facial and lingual walls.

Hemisepta

The remaining part of any interdental septum.

Infrabony defects

With 1-, 2- or 3-wall bony defects: Infrabony defect surrounded by 1-, 2- or 3-bony walls remaining.

The statistical analysis was performed using Chi-square test. $P < 0.05$ was considered as statistically significant difference.

$$\chi^2 = \sum \frac{O - E^2}{E}$$

Where, O=Observed number,
E=Expected number.

RESULTS

Out of 200 subjects, 116 (58%) patients had horizontal bone defect (Figure 1). In the age group of 30-40 years, 37 (36.6%) patients had craters, 4 (4%) had one-wall defects (Figure 2), 7 (6.9%) had two-wall defects (Figure 3), and 19 (18.8%) with three-wall defects (Figure 4). In the age group of 41-50 years, 5 (9.3%) patients had craters, 1 (1.9%) had two-wall defects, and 8 (14.8%) had three-wall defects. In the age group of 51-70 years, 3 (6.7%) patients with crater defects (Figure 5) were seen. In 125 males, 66 patients had horizontal bone defects, 29 (23.2%) patients had craters, 1 (0.8%) had one-wall defects, 4 (3.2%) had two-wall defects, and 25 (20%) had three-wall defects. Out of 75 females, 50 patients had horizontal bone defects. A total of 16 (21.3%) patients had craters, 3 (4%) had one-wall defects, 4 (5.3%) had two-wall defects, and 2 (2.7%) had three-wall defects. In 74 urban patients, 28 (21.9%) patients had craters, 3 (2.3%) had one-wall defects, 7 (5.5%) had two-wall defects, and 16 (12.5%) had three-wall defects. In 20 periurban patients, 7 (23.3%) patients had craters, 1 (3.3) had one-wall defects, 2 (6.7%) had three-wall defects. Out of 22

Figure 1: Horizontal bone defect in relation to 34-36

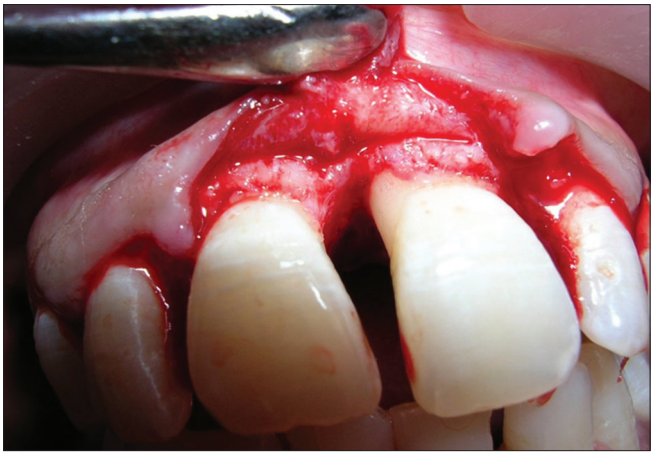


Figure 2: One-wall defect in relation to 21

Figure 3: Two-wall defect in relation to 16

rural patients, 10 (23.8%) patients had craters, 1 (2.4%) had two-wall defects, and 9 (21.4%) had three-wall defects. Out of 72 low socioeconomic status (SES) patients, 42 had horizontal bone defects, 16 (22.2%) patients had craters, 3 (2.1%) had one-wall defects, 1 (1.4%) were with two-wall defects, and 10 (13.9%) had three wall-defects.

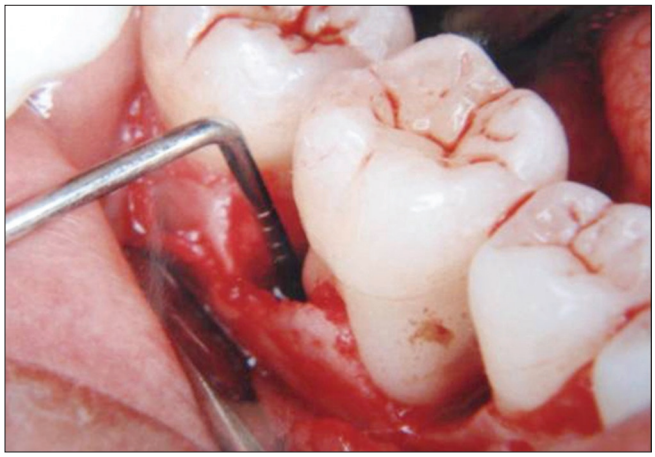


Figure 4: Three-wall defect in relation to 46

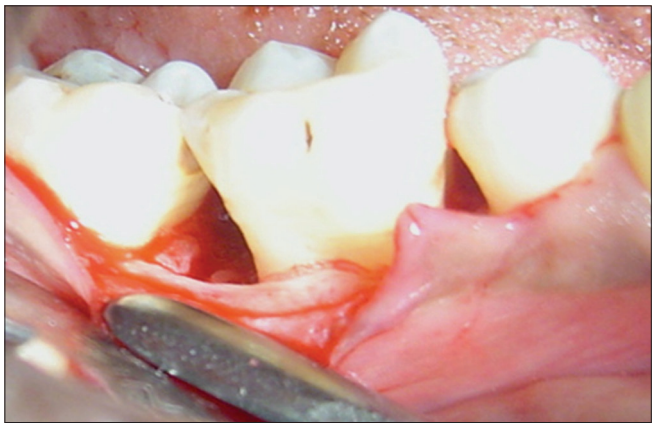


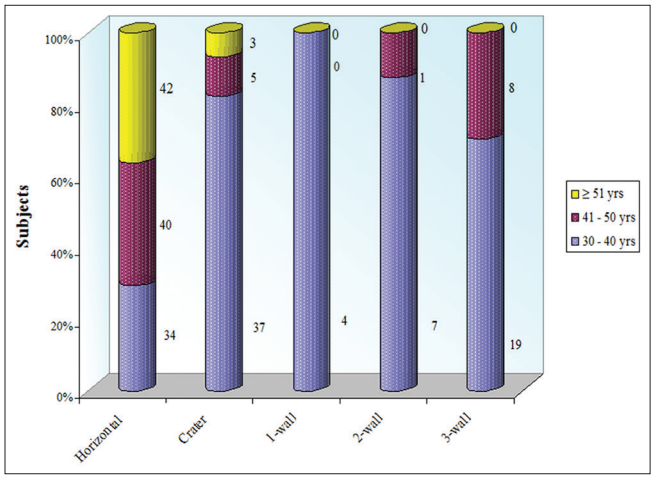
Figure 5: Crater in relation to 46 and 47

Out of 121 middle SES patients, 69 had horizontal bone defects, 28 (23.1%) patients had craters, 1 (0.8%) had one-wall defects, 7 (5.8%) had two-wall defects, and 16 (13.2%) had three-wall defects. Out of seven upper SES patients, 5 had horizontal bone defects, 1 (14.3%) patients had craters, and 1 (14.3%) had three-wall defects. The association between various defects and geographical location and SES was not statistically significant. Out of 200 patients, only 8 patients were recorded positive with trauma from occlusion (TFO). Among those 3, patients had horizontal bone defects (37.5%), 2 patients had craters (25%), 1 had two-wall defect (12.5%) and 2 patients had three-wall defects (25%). Out of 192 patients without TFO, 113 (58.9%) had horizontal bone defects, 43 (22.4%) patients had craters, 4 (2.1%) had one-walled defects, 7 (3.6%) had two-wall defects, and 25 (13%) with three-wall defects. The association between various defects and trauma from occlusion was also not statistically significant. In the study, 84 patients had angular defects, of craters were present in almost half the number of defects. In the mandible, craters were present in 23 of the posterior and two in the anterior sextant of the mandible. Two one-wall defects were seen

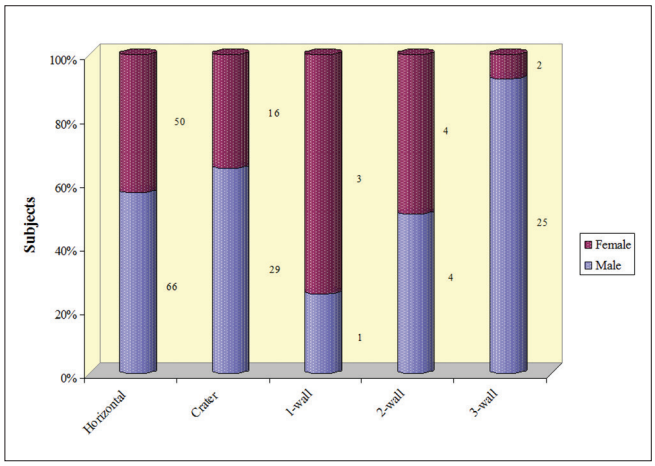
in the posterior sextant. Two-wall defect was present in one of the anterior and three in the posterior sextant. Three-wall defect was present in one of the anterior and 12 in the posterior sextant. In the maxilla, 20 craters were found in the posterior region. One-wall defect was recorded in one patient in the anterior region and one in the posterior region. Two-wall defect was present in four cases in the posterior segment. Out of 14 three-wall defects, 13 were present in the posterior region and one in the anterior region.

DISCUSSION

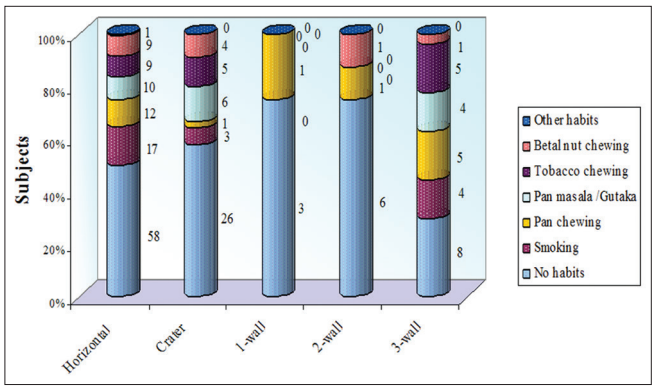
In the present study, almost half of the various defects were present in the age group of 30-40 years (Graph 1). It was found in accordance with the review that shows vertical defects which does not appear to increase after the age of 50 years.[6] In another study, it was recorded that only 18% of the participants had one or more periodontal intrabony defects, but the prevalence was higher in older than in younger age.[7] Relative higher distribution of intrabony defects might be due to steep increase in bone height loss from the age of 15 through 44 years. After the age of 50 years, there was little or no further alveolar bone loss.[6] In the present study, periodontal bone defects were more in males (Graph 2) when compared to the females (62.5% vs. 37.5%, respectively). Higher distribution of periodontal bony defects in males may be due to the adverse habits such as smoking, tobacco, and betel nut chewing act as risk factors for the periodontal disease. In the present study, association of various bone defects with the variables (smoking, pan chewing, pan masala, tobacco chewing, and betel nut chewing) was studied. A more number of defects were seen with the smokers, followed by pan chewing, pan masala, tobacco chewing, and betel nut chewing (Graph 3). This result was in relevance with the study conducted by Sheiham[8] where he found that severity of periodontal disease was more in smokers than non-smokers. Summers and Oberman[9] also concluded that one of the risk factors of periodontal disease is smoking, independent of age. Mehta *et al.*[10] in his study relating betel nut chewing with periodontal disease also found that betel nut chewing was one of the detrimental factors in the periodontal disease. This habit causes more destruction on the posterior region, which is in accordance with the present study. In this study, angular defects were found to be more in the mandibular region than in the maxilla (52.38% vs. 47.62%, respectively) depicting no statistical significance (Graph 4). A possible explanation may be that the greater thickness of supporting alveolar bone in the mandible and the



Graph 1: Distribution of various bony defects by age

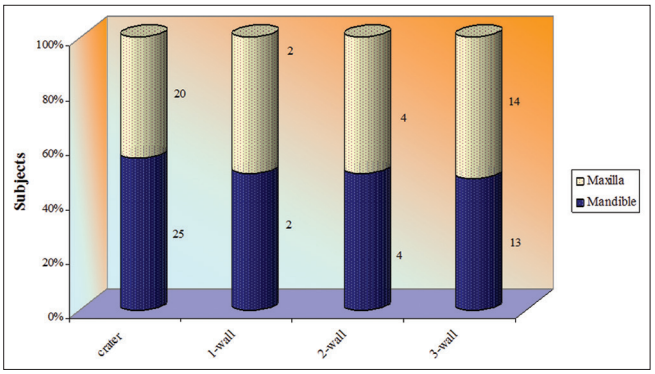


Graph 2: Distribution of various bony defects by sex

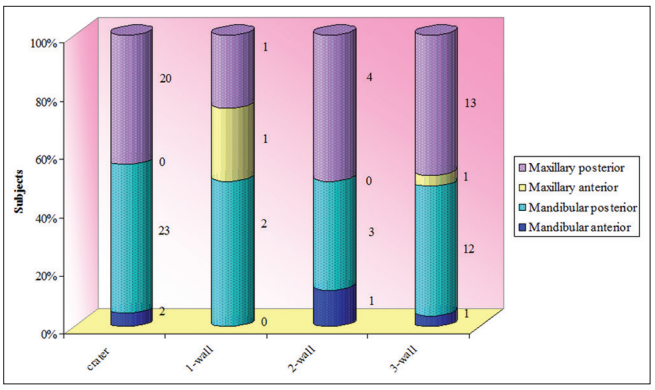


Graph 3: Distribution of various bony defects by adverse habits

radius of the effect of bacterial plaque is suggested to be approximately 1.5-2 mm, bony plates thinner than 1.5-2 mm might be completely destroyed by bacterial plaque (horizontal bone resorption); in contrast, thicker bony plates will develop infrabony defects. It was also suggested an association between thickness of bone surrounding the roots and morphology of



Graph 4: Distribution of various angular bony defects by arch



Graph 5: Prevalence of various angular bony defects by segment

bone resorption associated with periodontitis. This allows the formation of a greater number of intrabony defects.[11] The angular defects most commonly recorded in posterior region were craters, which accounted for almost half of the defects followed by three-wall, two-wall, and one-wall defects, which were similar to the study conducted by Vrotsos *et al.*[3] The more number of craters seen posteriorly may be that the interdental area is difficult to clean and the cancellous bone is more reactive and has a rapid turnover than cortical bone. Furthermore, it is well known that the interproximal bone between the anterior teeth is pyramidal, whereas between molars it is flat buccolingually. In addition, because of specific arrangement of capillaries in the interproximal gingival region and the col, inflammation results in a midplane bone resorption, with buccal and lingual plates affected much less, because the buccolingual dimension is far greater than the mesiodistal dimension.[3] The distribution of defects in the posterior sextants of the left and right side of maxilla and mandible showed no statistical significant result. While, there was a difference of distribution in the anterior and posterior sextants. Comparisons of angular defects in posterior and anterior segments showed that posterior mandibular segment had the slightly higher percentage of teeth with defects (20%) followed by the posterior maxillary segment (19%). The proportions of teeth with defect in the anterior segment of both the arches were similar. The higher percentage of bone defects in the posterior segments compared with anterior portions may be explained by the anatomical factors (Graph 5). The thin alveolar process, both in maxillary and mandibular anterior segments, leads to horizontal bone resorption and does not allow the formation of a large number of infrabony defects.

CONCLUSION

Chronic destructive periodontitis is the most common disease resulting in resorption of the bone supporting the teeth and leads to changes in the normal architecture of the alveolar processes. Periodontal bone loss from periodontitis may be either horizontal or vertical, leading to the formation of defects contained within the bone. An intrabony defect may be detected by radiographic means, bone sounding, or by visual examination of the bone defects during periodontal surgery or of dried skull. The disadvantage of radiographical examination is that it always shows 20-30% less destruction than the original and lingual or buccal cortical plates cannot be seen due to high percentage of root to bone mineralization. In bone sounding, we can only judge the bone level by tactile sensation. Direct observation of bone during surgery is the only means to accurately record the bone morphology.[4] Within the scope and limitations of the present study, it was concluded that prevalence and distribution of bone defect are multifactorial and were not related to one cause. It is related to the age, sex, SES, adverse habits, and the anatomy of the bone. Further studies may be required to know the standardized anatomy of the defect so the disease can be early identified and the proper treatment plan can be implemented.

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